FN Clarivate Analytics Web of Science

VR 1.0

PT J

AU Rao, VLR

Dogan, A

Bowen, KK

Dempsey, RJ

AF Rao, VLR

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Dempsey, RJ

TI Traumatic brain injury leads to increased expression of peripheral-type

benzodiazepine receptors, neuronal death, and activation of astrocytes

and microglia in rat thalamus

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE astrocytes; microglia; mRNA; peripheral-type benzodiazepine receptor;

PK11195; quantitative autoradiography; traumatic brain injury

ID TUMOR-NECROSIS-FACTOR; GLOBAL FOREBRAIN ISCHEMIA; CORTICAL IMPACT

INJURY; PROTEIN MESSENGER-RNA; LIGAND H-3 PK11195; BINDING-SITES;

PORTACAVAL ANASTOMOSIS; QUANTITATIVE AUTORADIOGRAPHY; CULTURED

ASTROCYTES; ENHANCED EXPRESSION

AB In mammalian CNS, the peripheral-type benzodiazepine receptor (PTBR) is localized on the outer mitochondrial membrane within the astrocytes and microglia. PTBR transports cholesterol to the site of neurosteroid biosynthesis. Several neurodegenerative disorders were reported to be associated with increased densities of PTBR. In the present study, we evaluated the changes in the PTBR density and gene expression in the brains of rats as a function of time (6 h to 14 days) after traumatic brain injury (TBI). Sham-operated rats served as control. Between 3 and 14 days after TBI, there was a significant increased in the binding of PTBR antagonist [H-3]PK11195 (by 106 to 185%, P < 0.01, as assessed by quantitative autoradiography and in vitro filtration binding) and PTBR mRNA expression (by 2- to 3.4-fold, P < 0.01, as assessed by RT-PCR) in the ipsilateral thalamus. At 14 days after the injury, the neuronal number decreased significantly (by 85 to 90%, P < 0.01) in the ipsilateral thalamus. At the same time point, the ipsilateral thalamus also showed increased numbers of the glial fibrillary acidic protein positive cells (astrocytes, by similar to 3.5-fold) and the ED-1 positive cells (microglia/macrophages, by similar to 36-fold), the two cell types known to be associated with PTBR, Increased PTBR expression following TBI seems to be associated with microglia/macrophages than astrocytes as PTBR density at different periods after TBI correlated better with the number of ED-1 positive cells (r(2) = 0.95) than the GFAP positive cells (r(2) = 0.56). TBI-induced increased PTBR expression is possibly an adaptive response to cellular injury and may play a role in the pathophysiology of TBI. (C) 2000 Academic Press.

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RI Dogan, Aclan/AAF-8305-2019

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WC Neurosciences

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PT J

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AF Dietrich, A

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TI The effect of Ginkgo biloba extract (EGb 761) on gliotic reactions in

the hippocampal formation after unilateral entorhinal cortex lesions

SO RESTORATIVE NEUROLOGY AND NEUROSCIENCE

LA English

DT Article

DE Ginkgo biloba; microglia; astrocyte; neuronal death; entorhinal cortex;

traumatic brain injury; stereology

ID CENTRAL-NERVOUS-SYSTEM; MESSENGER-RNA; CULTURED ASTROCYTES; GERBIL

HIPPOCAMPUS; AMEBOID MICROGLIA; COUNTING METHODS; RAT HIPPOCAMPUS;

NEURONS; RECOVERY; CELLS

AB Purpose: Ginkgo biloba extract (EGb 761) has been shown to facilitate behavioral and neuro-morphological recovery from brain injury, but less is known about its effects on glia. Since gliosis may be an important component of the recovery process, we tested the hypothesis that EGb 761 alters the time course and development of microglial activation and astrocytosis after brain injury.

Methods. Rats were treated with either saline or EGb 761 and killed at 2 hrs, 1, 3, 7, and 14 days following unilateral entorhinal cortex (EC) lesions. Microglia and their precursors were visualized with a silver impregnation method. and astrocytes with GFAP.

Results: Blood-borne monocytes/macrophages were seen as early as 2 hrs after injury in all animals. The side contralateral to the injury showed minimal microglial activation and there were no significant effects of drug treatment. On the side ipsilateral to the lesion EGb 761 enhanced microglial activation at 3, 7, and 14 days in the molecular layer and the hilus of the dentate gyrus; the areas of most profound deafferentation after EC injury. Regions of the corpus callosum also showed enhanced microglial activation over the same time course. Reactive astrocytes were stained with GFAP and were found to be more numerous than activated microglia, particularly in the ipsilateral corpus callosum. EGb 761 treatment enhanced astrocytosis at 3 days in the molecular layer, the hilus, and the corpus callosum on the ipsilateral side.

Conclusions. Taken together our results show that EGb 761 enhances, accelerates and prolongs the activation of microglia and astrocytosis at the site of injury.

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J9 RESTOR NEUROL NEUROS

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PT J

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TI Rapid and widespread microglial activation induced by traumatic brain

injury in rat brain slices

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE brain slice; C3b complement receptor (CR3); controlled impact injury;

microglial activation; OX42

ID COLONY-STIMULATING FACTORS; NITRIC-OXIDE; EXTRACELLULAR POTASSIUM;

MONONUCLEAR PHAGOCYTES; SPREADING DEPRESSION; MACROPHAGES; INVITRO;

ADULT; CELLS; EXPRESSION

AB In order to assess the role of circulating blood in early microglial activation after traumatic brain injury (TBI), controlled cortical impact injury was applied to adult rat brain slices (400 mu m in thickness) and the microglial response was examined. The complement receptor (CR3) expression and morphological transformation of the microglia were evaluated by OX42 immunohistochemistry. At 5 min following injury, activated microglia with intense CR3 expression appeared throughout the hemisphere on the injured side. In contrast, the morphology and CR3 expression of the microglia on the contralateral side were indistinguishable from those of the resident ramified microglia seen in normal brains, At 30 min following injury, microglial activation was more pronounced on the injured side, while the microglia on the contralateral side still retained a ramified morphology, These results are consistent with our previous observations made in in vivo experiments, which indicate that, as the brain slice paradigm excludes variables arising from the circulating blood, the rapid and widespread microglial activation observed following TBI can not be attributed exclusively to the infiltration of blood-borne macrophages or molecules. Rather this activation is most likely caused by intrinsic mechanisms within the brain tissue, such as traumatic depolarization.

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WC Critical Care Medicine; Clinical Neurology; Neurosciences

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AU Csuka, E

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Ammann, E

Trentz, O

Kossmann, T

Morganti-Kossmann, MC

TI Cell activation and inflammatory response following traumatic axonal

injury in the rat

SO NEUROREPORT

LA English

DT Article

DE astrocytes; experimental; immunohistochemistry; impact-acceleration;

inflammation; MHC class II; microglia; rat; traumatic brain injury

ID DIFFUSE BRAIN INJURY; CEREBROSPINAL-FLUID; MESSENGER-RNA; HEAD-INJURY;

ASTROCYTES; MODEL; EXPRESSION

AB In a rat model of traumatic brain injury cell activation was characterized immunohistochemically from 2 h up to 2 weeks. Reactive astrocytosis became apparent perivascularly and in the grey matter within 4 h after trauma. Increased OX42 immunoreactivity indicated microglial activation in cortex and hippocampus as early as 4 h, whereas up-regulation of MHC class II (OX6) was evident in white matter tracts at 24 h. Although macrophage (EDI) numbers increased in the meninges and perivascularly, brain infiltration appeared marginal. Accumulation of lymphocytes and granulocytes was not observed. Our results show that traumatic axonal injury induces a rapid and sustained glial activation in the absence of leukocyte infiltration. Thus, cell activation following diffuse trauma strongly differs from that found after focal brain damage, awaiting further functional characterization. NeuroReport 11:2587-2590 (C) 2000 Lippincott Williams & Wilkins.

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U1 0

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TI Dynamics of microglial activation after human traumatic brain injury are

revealed by delayed expression of macrophage-related proteins MRP8 and

MRP14

SO ACTA NEUROPATHOLOGICA

LA English

DT Article

DE intracellular signalling; Ki-67; microglia activation; normal brain

ischemia

ID CALCIUM-BINDING PROTEINS; LYMPHOCYTE CHEMOATTRACTANT FACTOR;

CENTRAL-NERVOUS-SYSTEM; MULTIPLE-SCLEROSIS LESIONS; CLASS-II ANTIGENS;

CD8(+) T-CELLS; HEAD-INJURY; RAT-BRAIN; CNS; ASTROCYTES

AB Human traumatic brain injury (TBI) is ideally suited for investigation of the kinetics of human microglial cell activation as the onset of lesion formation is precisely defined. The present study provides evidence of a distinct delay in macrophage/microglia response following TBI. Eighteen brains of patients who had survived TBI for 1 h to 6 months were analysed by immunohistology. Samples of contusional and non-contusional areas were studied using antibodies directed against antigens of microglia/macrophages [major histocompatibility complex class II, CD4, interleukin (IL)-16, macrophage-related protein (MRP) 8 and MRP14]. IL-16, a natural ligand to CD4, was expressed constitutively by numerous microglial cells in all cases throughout the brain. CD4 could be detected regularly on perivascular cells. MRP8 and MRP14, which are only expressed on activated macrophages and microglial cells, could be detected only within brains with a survival time of more than 72 h post TBI. In addition, proliferation of microglia detected by MIB-1 was not present until 72 h. This delayed expression of the activation markers MRP8 and MRP14 and the proliferation marker MIB-1 is comparable to experimental closed head injuries but strictly different from acute activation found in ischemic brains.

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TC 97

Z9 108

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U2 2

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J9 ACTA NEUROPATHOL

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TI Differential regulation of the monocytic calcium-binding peptides

macrophage-inhibiting factor related protein-8 (MRP8/S100A8) and

allograft inflammatory factor-1 (AIF-1) following human traumatic brain

injury

SO ACTA NEUROPATHOLOGICA

LA English

DT Article

DE allograft-inflammatory factor-1; microglia response factor-1;

macrophage-inhibiting factor related-protein-8/S 100A8; traumatic brain

injury; human

ID FACTOR-I; ACTIVATED MACROPHAGES; MICROGLIAL CELLS; EXPRESSION; LESIONS;

MRP14; ENCEPHALOMYELITIS; POLYPEPTIDE; CONTUSION; NEURITIS

AB Intracellular calcium (Ca2+) has been shown to function as second messenger and to be associated with activation of different cell types including microglia. Previously, in human focal cerebral infarctions an early expression of macrophage-related protein-8 (MRP8/ S100A8), a member of the Ca2+-binding S100-protein family, in microglia has been reported. On the other hand, a delayed activation of microglia was observed following traumatic brain injury (TBI). We therefore examined immunohistochemically microglial expression of MRP8 and allograft inflammatory factor-1 (AIF-1), identical to microglial response factor-1 (mrf-1) and ionized calcium binding adaptor molecule-1 (iba1) in human brains after TBI and in control brains. Both, MRP8 and AIF-1 are Ca2+-binding peptides which have been associated with microglial activation in experimental models and in human cerebral infarctions. Detection of AIF-1 in controls confirmed constitutive expression of this peptide in a subset of microglial cells. After TBI, the density of AIF-1(+) microglia did not increase significantly. Lesional expression of AIF-1 did not significantly differ from other brain regions. Furthermore, following TBI, we found no significant differences in the density of AIF-1+ microglia as compared to controls. Microglial MRP8 expression was not detectable in controls and within the first 3 days post TBI, but increased rapidly after 3 days post TBI, suggesting a subpopulation of microglial cells to be AIF-1(-)/MRP8(+). We conclude that the delayed expression of MRP8 and the lack of AIF-1 up-regulation in microglia after TBI is in contrast to ischemic brain lesions and might reflect different activation cascades of microglia.

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Z9 28

U1 0

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JI Acta Neuropathol.

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WC Clinical Neurology; Neurosciences; Pathology

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TI Lesion-associated accumulation of uPAR/CD87-expressing infiltrating

granulocytes, activated microglial cells/macrophages and upregulation by

endothelial cells following TBI and FCI in humans

SO NEUROPATHOLOGY AND APPLIED NEUROBIOLOGY

LA English

DT Article

DE fibrinolysis; human; ischaemia; tissue remodelling; traumatic brain

injury; urokinase-type plasminogen activator receptor

ID PLASMINOGEN-ACTIVATOR; UROKINASE RECEPTOR; MESSENGER-RNA; MATRIX

METALLOPROTEINASES; CLINICAL-TRIALS; BRAIN EDEMA; U937 CELLS;

EXPRESSION; MODULATION; SYSTEM

AB Urokinase-type plasminogen activator receptor (uPAR/CD87) together with its ligand, urokinase-type plasminogen activator (uPA), constitutes a proteolytic system associated with tissue remodelling and leucocyte infiltration. uPAR is a member of the glycosyl phosphatidyl inositol (GPI) anchored protein family. The functional role of uPAR comprises fibrinolysis by conversion of plasminogen to plasmin. In addition, uPAR promotes cell adhesion, migration, proliferation, re-organization of the actin cytoskeleton, and angiogenesis. Furthermore, uPAR is involved in prevention of scar formation and is chemoattractant to macrophages and leucocytes. In order to investigate the pathophysiological role of uPAR following human CNS injury we examined necrotic brain lesions resulting from traumatic brain injury (TBI; n = 28) and focal cerebral infarctions (FCI; n = 17) by immunohistochemistry. Numbers of uPAR(+) cells and uPAR(+) blood vessels were counted. Following brain damage, uPAR(+) cells increased significantly within 12 h, reached a maximum after 3-4 days and remained elevated until later stages. uPAR was expressed by infiltrating granulocytes, activated microglia/macrophages and endothelial cells. Numbers of uPAR(+) vessels increased in parallel subsiding earlier following FCI than post TBI. The restricted, lesion-associated accumulation of uPAR(+) cells in the brain parenchyma and upregulated expression by endothelial cells suggests a crucial role for the influx of inflammatory cells and blood-brain barrier (BBB) disturbance. Through a failure in BBB function, uPAR participates in formation of brain oedema and thus contributes to secondary brain damage. In conclusion, the study defines the localization, kinetic course and cellular source of uPAR as a potential pharmacological target following human TBI and FCI.

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NR 40

TC 35

Z9 41

U1 0

U2 0

PU WILEY

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J9 NEUROPATH APPL NEURO

JI Neuropathol. Appl. Neurobiol.

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WC Clinical Neurology; Neurosciences; Pathology

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TI Markers for cell-mediated immune response are elevated in cerebrospinal

fluid and serum after severe traumatic brain injury in humans

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE blood-brain barrier; cellular immunity; cerebrospinal fluid; human brain

injuries; immune markers; microglia activation; T-cell activation

ID SEVERE HEAD-INJURY; TUMOR-NECROSIS-FACTOR; ACUTE-PHASE RESPONSE;

SPINAL-CORD INJURY; RAT-BRAIN; BETA-2-MICROGLOBULIN LEVELS;

INTERLEUKIN-2 RECEPTOR; INFLAMMATORY RESPONSE; MESSENGER-RNA;

INTERFERON-GAMMA

AB The brain is believed to be an immunologically privileged organ, sheltered from the systemic immunological defense by the blood-brain barrier (BBB). However, there is increasing evidence for a marked inflammatory response in the brain after traumatic brain injury (TBI), Markers for cellular immune activation, neopterin, beta2-microglobulin (beta 2M), and soluble interleukin-2 receptor (sIL-2R), were measured for up to 3 weeks in cerebrospinal fluid (CSF) and serum of 41 patients with severe TBI in order to elucidate the time course and the origin of the cellular immune response following TBI. Neopterin gradually increased during the first posttraumatic week in both CSF and serum. Concentrations in CSF were generally higher than in serum, suggesting intrathecal release of this marker. beta 2M showed similar kinetics but with higher serum than CSF concentrations. Nonetheless, intrathecal release as assessed by the beta 2M index could be postulated for most of the patients. The mean levels of sIL-2R in both CSF and serum were elevated during the whole study period, serum concentrations being up to 2 x 10(4) times higher than in CSF. No significant intrathecal production of sIL-2R could be detected. The present data shows that severe TBI leads to a marked cell-mediated immune response within the brain and in the systemic circulation. In the intrathecal compartment the activated cells appear to be predominantly of the macrophage/microglia lineage, while the immune activation in the systemic circulation seems to involve mainly T-lymphocytes.

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TI The duality of the inflammatory response to traumatic brain injury

SO MOLECULAR NEUROBIOLOGY

LA English

DT Review

DE traumatic brain injury; inflammation; neurons; astrocytes; microglia;

blood brain barrier; cytokines; interleukin-6; transforming growth

factor-beta; tumor necrosis factor-alpha

ID TUMOR-NECROSIS-FACTOR; SEVERE HEAD-INJURY; AMYLOID PRECURSOR PROTEIN;

CENTRAL-NERVOUS-SYSTEM; GROWTH-FACTOR-BETA; INTERCELLULAR-ADHESION

MOLECULE-1; TRANSIENT FOREBRAIN ISCHEMIA; CORTICAL IMPACT INJURY;

ACUTE-PHASE RESPONSE; ADULT-RAT BRAIN

AB One and a half to two million people sustain a traumatic brain injury (TBI) in the US each year, of which approx 70,000-90,000 will suffer from long-term disability with dramatic impacts on their own and their families' lives and enormous socio-economic costs. Brain damage following traumatic injury is a result of direct (immediate mechanical disruption of brain tissue, or primary injury) and indirect (secondary or delayed) mechanisms. These secondary mechanisms involve the initiation of an acute inflammatory response, including breakdown of the blood-brain barrier (BBB), edema formation and swelling, infiltration of peripheral blood cells and activation of resident immunocompetent cells, as well as the intrathecal release of numerous immune mediators such as interleukins and chemotactic factors. An overview over the inflammatory response to trauma as observed in clinical and in experimental TBI is presented in this review. The possibly harmful /beneficial sequelae of post-traumatic inflammation in the central nervous system (CNS) are discussed using three model mediators of inflammation in the brain, tumor necrosis factor-alpha (TNF-alpha), interleukin-6 (IL-6), and transforming growth factor-beta (TGF-beta). While the former two may act as important mediators for the initiation and the support of post-traumatic inflammation, thus causing additional cell death and neurologic dysfunction, they may also pave the way for reparative processes. TGF-beta, on the other hand, is a potent anti-inflammatory agent, which may also have some deleterious long-term effects in the injured brain. The implications of this duality of the post-traumatic inflammatory response for the treatment of brain-injured patients using anti-inflammatory strategies are discussed.

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TI Early expression of glutamate transporter proteins in ramified microglia

after controlled cortical impact injury in the rat

SO GLIA

LA English

DT Article

DE astrocytes; controlled cortical impact injury; glutamate; glutamate

transporter; glutamate uptake; microglia; traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; AMINO-ACID TRANSPORTER; RELEASE; LOCALIZATION;

GLT-1; EXCITOTOXICITY; INHIBITION; INCREASES; NEURONS; MODEL

AB Traumatic brain injury is followed by increased extracellular glutamate concentration. Uptake of glutamate is mainly mediated by the glial glutamate transporters GLAST and GLT-1. Extent and distribution of GLAST and GLT-1 were studied in a rat model of controlled cortical impact injury (CCH). Western Blot analysis revealed lowest levels of GLAST and GLT-1 with a decrease by 40%-54% and 42%-49% between 24 and 72 h posttrauma. By 8 h after CCll, CSF glutamate levels were increased (10.5 muM vs. 2.56 VM in controls; P < 0.001), reaching maximum values by 48 h. A significant increase in de novo GLAST and GLT-1 expressing ramified microglia was observed within 4 h, reached a stable level by 48 li, and remained high up to 72 h after CCH. Furthermore, ramified microglia de novo expressed the neuronal glutamate transporter EAAC1 after CCII Following CCII, GLAST/GLT-1 and GFAP coexpressing astrocytes were immediately reduced, reaching minimum levels within 8 h. This reduction of expression could be either due to protein downregulation or loss of astrocytes. At 72 h, a marked population of GLAST- and GLT-1-positive reactive astrocytes appeared. These results support the hypothesis that reduced astrocytic GLAST and GLT-1 protein levels following MI contribute to evolving secondary injury. Microglia are capable of de novo expressing glutamate transporter proteins, indicating that the expression of glial and neuronal glutamate transporters is not restricted to a specific glial or neuronal lineage. Ramified microglia may play an important compensatory role in the early regulation of extracellular glutamate after CCII. (C) 2001 Wiley-Liss, Inc.

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TI Traumatic brain injury induces prolonged accumulation of

cyclooxygenase-1 expressing microglia/brain macrophages in rats

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE bystander damage; inflammation; prostaglandin; tissue remodeling

ID SPINAL-CORD INJURY; CEREBRAL-ISCHEMIA; GENE DISRUPTION; EFFECTOR-CELLS;

IN-VIVO; INFLAMMATION; COX-1; DIFFERENTIATION; DISEASE; GLIOMA

AB Inflammatory cellular responses to brain injury are promoted by proinflammatory messengers. Cyclooxygenases (prostaglandin endoperoxide H synthases [PGH]) are key enzymes in the conversion of arachidonic acid into prostanoids, which mediate immunomodulation, mitogenesis, apoptosis, blood flow, secondary injury (lipid peroxygenation), and inflammation. Here, we report COX-1 expression following brain injury. In control brains, COX-1 expression was localized rarely to brain microglia/macrophages. One to 5 days after injury, we observed a highly significant (P < 0.0001) increase in COX-1(+) microglia/macrophages at perilesional areas and in the developing core with a delayed culmination of cell accumulation at day 7, correlating with phagocytic activity. There, cell numbers remained persistently elevated up to 21 days following injury. Further, COX-1(+) cells were located in perivascular Virchow-Robin spaces also reaching maximal numbers at day 7. Lesion-confined COX-1(+) vessels increased in numbers from day 1, reaching the maximum at days 5-7. Double-labeling experiments confirmed coexpression of COX-1 by ED-1(+) and OX-42(+) microglia/ macrophages. Transiently after injury, most COX-1(+) microglia/macrophages coexpress the activation antigen OX-6 (MHC class II). However, the prolonged accumulation of COX-1(+), ED-1(+) microglia/macrophages in lesional areas enduring the acute postinjury inflammatory response points to a role of COX-1 in the pathophysiology of secondary injury. We have identified localized, accumulated COX-1 expression as a potential pharmacological target in the treatment of brain injury. Our results suggest that therapeutic approaches based on long-term blocking including COX-1, might be superior to selective COX-2 blocking to suppress the local synthesis of prostanoids.

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TI Enhanced glial activation and expression of specific CNS

inflammation-related molecules in aged versus young rats following

cortical stab injury

SO JOURNAL OF NEUROIMMUNOLOGY

LA English

DT Article

DE microglia; astrocytes; aging; brain trauma; neurodegenerative disease

ID BLOOD-BRAIN-BARRIER; EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS; NF-KAPPA

B; MONONUCLEAR PHAGOCYTES; NERVOUS-SYSTEM; CYTOKINES; ASTROCYTES; ADULT;

INTERLEUKIN-10; MICROGLIA

AB Aging is associated with increased glial responsiveness that may enhance the brain's susceptibility to injury and disease. To determine whether unique age-related molecular responses occur in brain injury, we assessed m-RNA levels of representative central nervous system (CNS) inflammation-related molecules in young (3 months) and aged (36 months) Fisher 344/Brown Norwegian F1 hybrid rats following cortical stab. Enhanced glial activation in older animals was accompanied by increased expression of a subset of inflammation-related mRNAs, including IL-1 beta, TNF alpha, IL-6, ICAM-1, inducible nitric oxide synthase (iNOS), metalloprotednase-9 (NEAP-9), and complement 3 alpha -chain 1 (C3 alpha1). Recognition of these age-specific differences may guide development of novel treatment regimes for older individuals. (C) 2001 Elsevier Science BY. All rights reserved.

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NR 54

TC 99

Z9 119

U1 0

U2 5

PU ELSEVIER

PI AMSTERDAM

PA RADARWEG 29, 1043 NX AMSTERDAM, NETHERLANDS

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J9 J NEUROIMMUNOL

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WC Immunology; Neurosciences

WE Science Citation Index Expanded (SCI-EXPANDED)

SC Immunology; Neurosciences & Neurology

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ER

PT J

AU Orihara, Y

Ikematsu, K

Tsuda, R

Nakasono, I

AF Orihara, Y

Ikematsu, K

Tsuda, R

Nakasono, I

TI Induction of nitric oxide synthase by traumatic brain injury

SO FORENSIC SCIENCE INTERNATIONAL

LA English

DT Article

DE inducible nitric oxide synthase; cerebrovascular smooth muscle cell;

neutrophil; microglia; traumatic brain injury

ID SMOOTH-MUSCLE CELLS; CEREBRAL BLOOD-FLOW; HEAD-INJURY;

HUMAN-NEUTROPHILS; L-ARGININE; RATS; EXPRESSION; IMMATURE

AB We investigated the dynamic induction/expression of inducible nitric oxide synthase (iNOS) using human brains made available through death by traumatic brain injury (TBI). Astrocytes. micro.-lia. and neutrophils were identified in tissue using immunohistochemical staining with antibodies against glial fibrillary acidic protein (GFAP). MHC class II antigen, and neutrophil elastase. respectively. The localization of iNOS protein in each of these cell types was evaluated using immunohistochemistry.

Within 2 days of injury, iNOS immunoreactivity was not detected. However, after 2 days, immunoreactivity was detected in the traumatized brain. The iNOS immunoreactivity was localized on neutrophils and microglia/macrophages in the areas around the tissue necrosis in the traumatized cortical hemisphere, in the deep part of the cortex and the dentate gyri of the hippocampi adjacent to the hemorrhage, and within the cytoplasm of vascular smooth muscle cell of a small artery or arteriole surrounding the injured region. This reactivity was absent after 8 days post-injury,

These observations confirmed the prolonged induction of iNOS within various cells in the injured brain. These responses suggest that iNOS plays a crucial role in cerebrovascular damage and/or secondary brain damage subsequent to traumatic brain injury. Furthermore. the dense nitric oxide (NO) generated by iNOS may play a role in neuronal cell death after injury. (C) 2001 Elsevier Science Ireland Ltd. All rights reserved.

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NR 32

TC 62

Z9 67

U1 0

U2 3

PU ELSEVIER SCI IRELAND LTD

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JI Forensic Sci.Int.

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WC Medicine, Legal

WE Science Citation Index Expanded (SCI-EXPANDED)

SC Legal Medicine

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ER

PT J

AU Hutchison, JS

Derrane, RE

Johnston, DL

Gendron, N

Barnes, D

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TI Neuronal apoptosis inhibitory protein expression after traumatic brain

injury in the mouse

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE apoptosis; caspase; microglia; neuronal apoptosis inhibitor protein

(NAIP); tumor necrosis factor-alpha (TNF alpha); immunofluorescence;

Western blot

ID NECROSIS-FACTOR-ALPHA; PROGRAMMED CELL-DEATH; SPINAL-CORD INJURY;

CLOSED-HEAD INJURY; DNA FRAGMENTATION; POLY(ADP-RIBOSE) POLYMERASE;

COGNITIVE DEFICITS; RAT-BRAIN; TNF-ALPHA; IN-VIVO

AB Apoptosis of brain cells is triggered by traumatic brain injury (TBI) and is blocked by caspase inhibitors. The neuronal apoptosis inhibitor protein (NAIP), which has been shown to inhibit apoptosis by both caspase-dependant and caspase-independent mechanisms, is neuroprotective in rat models of cerebral ischemia and axotomy. In order to gain a better appreciation of CNS apoptosis following head injury in general and the possible involvement of NAIP specifically, we have configured a mouse model of TBI. In addition to demonstrating apoptosis, the spatiotemporal expression or levels of a number of proteins with apoptosis modulating effects have been determined. Apoptosis of neurons and oligodendrocytes following TBI was observed in brain sections which were triple-stained with in situ end labeling, bisbenzimide and immunofluorescent stain for neuron specific nuclear protein and myelin-associated glycoprotein, respectively. Further evidence for apoptosis following TBI in this model was obtained in brain samples using ligation-mediated PCR amplification of DNA fragments and gel electrophoresis. The temporal profile of apoptosis was similar to the temporal profile of microglial activation determined by CD11b staining and TNF alpha expression induced by TBI. NAIP staining in sections of cerebral cortex and subcortical white matter increased at 6 h and decreased towards control levels at 24 h post-TBI. Temporal changes in the expression of NAIP were also observed using Western blot analysis of brain samples removed from injured cortex and sub-cortical white matter. At the time that NAIP expression decreased markedly (24 h post-TBI), procaspase-3 levels also decreased, PARP cleavage increased, and the highest levels of apoptosis were observed. These findings have implications in our understanding of traumatically induced programmed cell death and may be useful in the configuration of therapies for this common injury state.

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Research Council Canada; University of Ottawa

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J9 J NEUROTRAUM

JI J. Neurotrauma

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WC Critical Care Medicine; Clinical Neurology; Neurosciences

WE Science Citation Index Expanded (SCI-EXPANDED)

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ER

PT J

AU Sanz, O

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Castellano, B

AF Sanz, O

Acarin, L

González, B

Castellano, B

TI NF-κB and IκBα expression following traumatic brain injury to the

immature rat brain

SO JOURNAL OF NEUROSCIENCE RESEARCH

LA English

DT Article

DE astrocyte; microglia; glial response; developing brain; transcription

factor; inflammation

ID THALAMIC GLIAL RESPONSE; INDUCED APOPTOSIS; TRANSCRIPTION FACTOR;

HIPPOCAMPAL-NEURONS; CYTOKINE EXPRESSION; EXCITOTOXIC LESION; YOUNG

BRAIN; ACTIVATION; PROTEINS; SUPPRESSION

AB NF-kappaB is one of the most important modulators of stress and inflammatory gene expression in the nervous system. In the adult brain, NF-kappaB upregulation has been demonstrated in neurons and glial cells in response to experimental injury and neuropathological disorders, where it has been related to both neurodegenerative and neuroprotective activities. Accordingly, the aim of this study was to evaluate the cellular and temporal patterns of NF-kappaB activation and the expression of its endogenous inhibitor IkappaBalpha following traumatic brain injury (TBI) during the early postnatal weeks, when the brain presents elevated levels of plasticity and neuroprotection. Our results showed that cortical trauma to the 9-day-old rat brain induced a very fast upregulation of NF-kappaB, which was maximal within the first 24 hours after injury. NF-kappaB was mainly observed in neuronal cells of the degenerating cortex as well as in astrocytes located in the corpus callosum adjacent to the injury, where a pulse-like pattern of microglial NF-kappaB activation was also found. In addition, astrocytes of the corpus callosum, and microglial cells to a lower extent, also showed de novo expression Of IkappaBalpha within the time of NF-kappaB activation. This study suggests an important role of NF-kappaB activation in the early mechanisms of neuronal death or survival, as well as in the development of the glial and inflammatory responses following traumatic injury to the immature rat brain. (C) 2002 Wiley-Liss, Inc.

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CR Acarin L, 1999, NEUROSCIENCE, V89, P549, DOI 10.1016/S0306-4522(98)00331-5

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NR 38

TC 48

Z9 54

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J9 J NEUROSCI RES

JI J. Neurosci. Res.

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WC Neurosciences

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SC Neurosciences & Neurology

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PT J

AU Beschorner, R

Nguyen, TD

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Pedal, I

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Schluesener, HJ

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Schwab, JM

TI CD14 expression by activated parenchymal microglia/macrophages and

infiltrating monocytes following human traumatic brain injury

SO ACTA NEUROPATHOLOGICA

LA English

DT Article

DE traumatic brain injury; inflammation; immune response; CD14

ID TUMOR-NECROSIS-FACTOR; MICROGLIAL CELLS; DEPENDENT MECHANISMS;

MONONUCLEAR-CELLS; NEGATIVE BACTERIA; GENE-EXPRESSION; UP-REGULATION;

SOLUBLE CD14; EX-VIVO; LIPOPOLYSACCHARIDE

AB The immune response in the central nervous system (CNS) is under tight control of regulatory mechanisms, resulting in the establishment of immune privilege. CNS injury induces an acute inflammatory reaction, composed mainly of invading leukocytes and activated microglial cells/macrophages. The generation of this robust immune response requires binding of receptors such as CD14, a pattern recognition receptor of the immune system. CD14, a surface molecule of monocytic cells, is up-regulated after monocyte stimulation and is involved in cellular activation. To examine CD14 expression in human brain lesions we investigated sections of brains obtained at autopsy from 25 cases following closed traumatic brain injury (TBI) and 5 control brains by immunohistochemistry. Detection of CD14 in controls demonstrated constitutive expression by perivascular cells, but not in parenchymal microglial cells, equivalent to known expression pattern of ED2 in rats. Following TBI, numbers of CD14(+) cells in perivascular spaces and in the brain parenchyma increased in parallel within 1-2 days, both at the lesion and in adjacent perilesional areas. The number of CD14(+) cells in perivascular spaces and in the brain parenchyma reached maximum levels within 4-8 days and remained elevated until weeks after trauma. In contrast to activated parenchymal microglia/macrophages, resting parenchymal microglial cells lacked CD14. Thus, early CD14 expression constitutes an essential part of the acute inflammatory CNS response following trauma.

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PT J

AU Basu, A

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Styren, SD

DeKosky, ST

Levison, SW

AF Basu, A

Krady, JK

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TI The type 1 interleukin-1 receptor is essential for the efficient

activation of microglia and the induction of multiple proinflammatory

mediators in response to brain injury

SO JOURNAL OF NEUROSCIENCE

LA English

DT Article

DE cytokines; IL-1; IL-6; TNF-alpha; traumatic brain injury;

prostaglandins; astrocytes; null mutant mice

ID NECROSIS-FACTOR-ALPHA; FACTOR MESSENGER-RNA; EXCITOTOXIC NEURONAL

DAMAGE; NF-KAPPA-B; GROWTH-FACTOR; TRANSGENIC MICE; RAT-BRAIN;

ALZHEIMERS-DISEASE; INFLAMMATORY RESPONSE; CONVERTING-ENZYME

AB Interleukin-1 (IL-1) is induced immediately after insults to the brain, and elevated levels of IL-1 have been strongly implicated in the neurodegeneration that accompanies stroke, Alzheimer's disease, and multiple sclerosis. In animal models, antagonizing IL-1 has been shown to reduce cell death; however, the basis for this protection has not been elucidated. Here we analyzed the response to penetrating brain injury in mice lacking the type 1 IL-1 receptor (IL-1R1) to determine which cellular and molecular mediators of tissue damage require IL-1 signaling. At the cellular level, fewer amoeboid microglia/macrophages appeared adjacent to the injured brain tissue in IL-1R1 null mice, and those microglia present at early postinjury intervals retained their resting morphology. Astrogliosis also was mildly abrogated. At the molecular level, cyclooxygenase-2 (Cox-2) and IL-6 expression were depressed and delayed. Interestingly, basal levels of Cox-2, IL-1, and IL-6 were significantly lower in the IL-1R1 null mice. In addition, stimulation of vascular cell adhesion molecule-1 mRNA was depressed in the IL-1R1 null mice, and correspondingly, there was reduced diapedesis of peripheral macrophages in the IL-1R1 null brain after injury. This observation correlated with a reduced number of Cox-2(+) amoeboid phagocytes adjacent to the injury. In contrast, several molecular aspects of the injury response were normal, including expression of tumor necrosis factor-alpha and the production of nerve growth factor. Because antagonizing IL-1 protects neural cells in experimental models of stroke and multiple sclerosis, our data suggest that cell preservation is achieved by abrogating microglial/macrophage activation and the subsequent self-propagating cycle of inflammation.

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Z9 158

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AU Liu, PH

Wang, YJ

Tseng, GF

AF Liu, PH

Wang, YJ

Tseng, GF

TI Close axonal injury of rubrospinal neurons induced transient

perineuronal astrocytic and microglial reaction that coincided with

their massive degeneration

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE axotomy; trauma; spinal cord injury; brain stem; red nucleus; CNS

ID RAT CORTICOSPINAL NEURONS; NEUROTROPHIC FACTORS; SPINAL MOTONEURONS;

DISTAL AXOTOMY; RED NUCLEUS; ADULT-RAT; CORD; REGENERATION; TERMINALS;

NERVE

AB To learn more about the pathophysiology of axonal injury and the significance of axon collaterals on the survival of axotomized cord-projection central neurons, we studied the survival rate, surrounding astrocytic and microglial reactions, and bouton coverage on rat rubrospinal cell bodies following their axonal lesion at the brain stem and upper cervical level. T e brain stem lesion disconnected most rubrospinal neurons from all their targets, while the upper cervical lesion spared their supraspinal collaterals. Much higher cell loss accompanied by robust astrocytic and microglial reaction was found following brain stem than upper cervical lesion starting 4 days postaxotomy. The reaction of astrocytes had subsided while microglial reaction remained relatively robust by 10 weeks postaxotomy when the cell loss had slowed down. Ultrastructural observation revealed that reactive astrocytes covered 40%, an increase from the 20% of control, of brain stem-axotomized rubrospinal cell body surface at 4 days and 2 weeks and returned to normal levels by 10 weeks postlesion. An increase of apposition by axons and dendrites and a moderate decrease of round and flattened vesicle-containing bouton contacts at 4 days and 2 weeks and returning to normal levels at 10 weeks postaxotomy accompanied this. It appears that although axotomy induced robust astrocytic reaction around cord-projection central neurons, this, unlike their periphery-projection counterparts, failed to effectively strip their somatic synapses. In effect, this might in part determine neuronal fate following axonal injury. (C) 2002 Elsevier Science (USA).

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WANG YJ, IN PRESS J NEUROTRAU

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AF Mueller, CA

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Meyermann, R

Schwab, JM

TI Lesional expression of a proinflammatory and antiangiogenic cytokine

EMAP II confined to endothelium and microglia/macrophages during

secondary damage following experimental traumatic brain injury

SO JOURNAL OF NEUROIMMUNOLOGY

LA English

DT Article

DE EMAP II; secondary brain damage; traumatic brain injury; inflammation;

antiangiogenic

ID ACTIVATING POLYPEPTIDE-II; TUMOR-NECROSIS-FACTOR; SPINAL-CORD;

INFLAMMATORY RESPONSE; VASCULAR ARCHITECTURE; MICROGLIAL CELLS; CORTICAL

IMPACT; MACROPHAGES; RAT; ENCEPHALOMYELITIS

AB We analyzed expression of Endothelial Monocyte-Activating Polypeptide II (EMAP II), a proinflammatory, antiangiogenic cytokine in rat brains after stab wound injury and observed a highly significant (p<0.0001) lesional accumulation confined to microglia/macrophages. Maximum numbers were seen at day 5 declining until 21 days after injury. Further, EMAP II+ microglia/macrophages formed clusters in perivascular Virchow-Robin spaces. Prolonged accumulation of EMAP II+, EDI+ microglia/macrophages and increased lesional numbers of EMAP II+ endothelial/smooth muscle cells during the acute postinjury period might indicate that EMAP II enrich the proinflammatory and antiangiogenic repertoire of effector molecules expressed by activated microglia/macrophages during secondary damage. (C) 2002 Elsevier Science B.V. All rights reserved.

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AU Delgado, M

Ganea, D

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TI Vasoactive intestinal peptide prevents activated microglia-induced

neurodegeneration under inflammatory conditions: potential therapeutic

role in brain trauma

SO FASEB JOURNAL

LA English

DT Article

DE VIP; inflammation; endotoxin; cytokines; neuropeptides

ID TUMOR-NECROSIS-FACTOR; SPINAL-CORD-INJURY; MESSENGER-RNA; FACTOR-ALPHA;

RAT-BRAIN; CHEMOKINE PRODUCTION; TNF-ALPHA; IN-VITRO; KAPPA-B; PITUITARY

AB In most neurodegenerative disorders, including multiple sclerosis, Parkinson's disease, and Alzheimer's disease, a massive neuronal cell death occurs as a consequence of an uncontrolled inflammatory response, where activated microglia and its cytotoxic agents play a crucial pathologic role. Because current treatments for these diseases are not effective, several regulatory molecules termed "microglia-deactivating factors" recently have been the focus of considerable research. Vasoactive intestinal peptide ( VIP) is a neuropeptide with a potent antiinflammatory effect, which has been found to protect from other inflammatory disorders, such as endotoxic shock and rheumatoid arthritis. In the present study, we investigate the effect of VIP on inflammation-mediated neurodegeneration in vitro and in vivo as well as on the putative neuroprotective effect of VIP on experimental pathological conditions in which central nervous system (CNS) inflammation is involved, such as brain trauma. The involvement of activated microglia and their derived cytotoxic products is also studied. VIP has a clear neuroprotective effect on inflammatory conditions by inhibiting the production of microglia-derived proinflammatory factors (tumor necrosis factor alpha, interleukin-1beta, nitric oxide). In this sense, VIP prevents neuronal cell death following brain trauma by reducing the inflammatory response of neighboring microglia. Therefore, VIP emerges as a valuable neuroprotective agent for the treatment of pathologic conditions of the CNS where inflammation-induced neurodegeneration occurs.

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WC Biochemistry & Molecular Biology; Biology; Cell Biology

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AU Wilson, S

Raghupathi, R

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AF Wilson, S

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Graham, DI

TI Continued <i>in situ</i> DNA fragmentation of microglia/macrophages in

white matter weeks and months after traumatic brain injury

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE human traumatic brain injury; TUNEL staining; Wallerian degeneration

ID SPINAL-CORD-INJURY; DIFFUSE AXONAL INJURY; MISSILE HEAD-INJURY;

CONTROLLED CORTICAL IMPACT; VEGETATIVE STATE; CELL-DEATH; APOPTOSIS;

RATS; NEUROPATHOLOGY; DAMAGE

AB Paraffin-embedded material from the pons of head-injured patients whose disability could be attributed to diffuse traumatic axonal injury, and controls, was identified from the department's archive. The cases were divided into three groups based on survival, viz Group I (n = 5) who survived for between 4 and 8 weeks, Group 2 (n = 5) for between 3 and 9 months, and Group 3 (n = 5) who survived for more that 12 months. Sections were stained by the TUNEL (TdT-mediated UTP nick end labelling) technique, and by H&E, LFB/CV and immunohistochemically for astrocytes (GFAP) and microglia/macrophages (CD68). Microscopic abnormalities were mapped onto line diagrams of two levels of the pons and quantitation of the response determined by an eye-piece graticule placed over the medial lemmisci, cortico-spinal and transverse fiber tracts. Data were pooled by region of interest. In the H&E and LFB/CV stained sections, there was variable pallor of staining in ascending and descending fiber tracts due to loss of myelin: within these same tracts there was an astrocytosis and increased numbers of microglia/macrophages compared with controls. In the white matter tracts of the controls, there was on average 1-2 TUNEL+ cells per unit area. In contrast, there were on average 2-16 TUNEL+ cells in the cortico-spinal tracts and in the medial lemnisci of all groups of head-injured patients. CD68(+) cells co-located with the TUNEL+, and their number mirrored the TUNEL I staining with on average 16-30 cells per unit area in Group 1, 14-27 cells per unit area in Group 2, and 12-14 cells per unit area in Group 3. There was a statistical association between the TUNEL+ and CD68(+) cells. Few changes were seen in the transverse fiber tracts of the pons. These findings indicate that most of the in situ DNA fragmentation occurred in microglia/macrophages in ascending and descending fiber tracts of the brain stem in which by conventional light microscopy there is Wallerian degeneration. However, in addition, a few TUNEL+ oligodendrocyte-like cells were also seen.

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Z9 35

U1 0

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J9 J NEUROTRAUM

JI J. Neurotrauma

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AF Bellander, BM

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Von Euler, G

Ohlsson, M

Svensson, M

TI Activation of microglial cells and complement following traumatic injury

in rat entorhinal-hippocampal slice cultures

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE complement; slice cultures; traumatic brain injury

ID SULFATED GLYCOPROTEIN-2; BRAIN MACROPHAGES; NERVOUS-SYSTEM; MYELIN;

ATTACK; PROTEIN; DEGENERATION; RECRUITMENT; INCREASE; NUCLEUS

AB The complement cascade has been suggested to be involved in development of secondary brain damage following traumatic brain injury (TBI). Previous studies have shown that reactive microglia are involved in activation of the complement cascade following various injuries to the nervous system. Macrophages seem to have a significant role in this process, but it is still unclear whether these cells, as well as the complement components, are derived from reactive microglia or if these biological events only can occur as a result from the influx of plasma and monocytes via a disrupted blood-brain barrier (BBB). The aim of this study was to investigate the response of microglial cells and the complement system in the absence of plasma/blood components following a standardized crush injury in an entorhinal-hippocampal slice culture. There was a clear increase in complement component C1q and C5b-9-IR (Membrane Attack Complex, MAC) in the area near the crush injury. MAC-IR appeared as numerous dots in clusters which co-localized with anti-NeuN labelled neurons in the injury border zone. Complement C1q-IR co-localized with reactive microglia, co-labelled with OX42 antisera. These findings show activation of the complement cascade near the injury zone and in particular, formation of MAC at the surface of neurons in this area. There was a distinct activation of microglial cells (OX42-IR) near the site of injury, as well as an increase in ED-1 expressing macrophages. In the absence of blood and plasma components it is likely that ED-1-labelled cells represent reactive microglia transformed into macrophages. In addition, Neurons (Neun-IR) near the injury were found to co-localize with clusterin-IR indicating upregulation of a defense system to the endogenous complement attack. The present study provides evidence that microglia and complement is activated in the injury border zone of the tissue slice in a similar fashion as in vivo following TBI, despite the absence of plasma/blood products and cells. These findings support the hypothesis that reactive microglia have a key role in complement activation following TBI by local synthesis of complement with a potential impact on development of secondary neuronal insults.

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JI J. Neurotrauma

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SC General & Internal Medicine; Neurosciences & Neurology

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PT J

AU Grossman, KJ

Goss, CW

Stein, DG

AF Grossman, KJ

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TI Effects of progesterone on the inflammatory response to brain injury in

the rat

SO BRAIN RESEARCH

LA English

DT Article

DE progesterone; traumatic brain injury; microglia; astrocyte; edema;

neuronal survival

ID CEREBRAL-ARTERY OCCLUSION; FIBRILLARY ACIDIC PROTEIN; NITRIC-OXIDE

SYNTHASE; CONTUSION INJURY; SPINAL-CORD; CORTICAL CONTUSION; REACTIVE

GLIOSIS; CORTEX ABLATION; MICROGLIA; CNS

AB The effects of progesterone on the cellular inflammatory response to frontal cortex in jury were examined on Postsurgical days 1, 3 5, 7 and 9 in male rats treated with progesterone (4 mg/kg) and/or vehicle. Rats with bilateral contusions showed increased levels of edema on days I, 3 and 5, more reactive astrocytes on days 3, 5, 7 and 9, and more macrophages/activated microglia on days 1, 3, 5 and 9 compared to shams. The number of neurons in the medial dorsal nucleus (MDN) of the thalamus reduced on days 5 and 9 after injury compared to shams. Progesterone reduced edema levels and increased the accumulation of macrophages/activated microglia compared to vehicle controls (p<0.025); however, these changes in the inflammatory response were not related to MDN neuronal Survival. Our results Confirm the possibility that one way progesterone mediates its neuroprotective effects following injury is through its actions on the inflammatory response. (C) 2004 Elsevier B.V. All rights reserved.

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AU Rodriguez-Paez, AC

Brunschwig, JP

Bramlett, HM

AF Rodriguez-Paez, AC

Brunschwig, JP

Bramlett, HM

TI Light and electron microscopic assessment of progressive atrophy

following moderate traumatic brain injury in the rat

SO ACTA NEUROPATHOLOGICA

LA English

DT Article

DE progressive atrophy; inflammation; macrophages/microglia; traumatic

brain injury; white matter

ID AMYLOID PRECURSOR PROTEIN; ENDOTHELIAL GROWTH-FACTOR; NONDISRUPTIVE

AXONAL INJURY; NITRIC-OXIDE SYNTHASE; CLOSED-HEAD-INJURY; OPTIC-NERVE

FIBERS; INFLAMMATORY RESPONSE; STRETCH-INJURY; WHITE-MATTER;

POSTTRAUMATIC HYPOTHERMIA

AB The presence of progressive white matter atrophy following traumatic brain injury (TBI) has been reported in humans as well as in animal models. However, a quantitative analysis of progressive alterations in myelinated axons and other cellular responses to trauma has not been conducted. This study examined quantitative differences in myelinated axons from several white and gray matter structures between non-traumatized and traumatized areas at several time points up to 1 year. We hypothesize that axonal numbers decrease over time within the structures analyzed, based on our previous work demonstrating shrinkage of tissue in these vulnerable areas. Intubated, anesthetized male Sprague-Dawley rats were subjected to moderate (1.8-2.2 atm) parasagittal fluid-percussion brain injury, and perfused at various intervals after surgery. Sections from the fimbria, external capsule, thalamus and cerebral cortex from the ipsilateral hemisphere of traumatized and sham-operated animals were prepared and. estimated total numbers of myelinated axons were determined by systematic random sampling. Electron micrographs were obtained for ultrastructural analysis. A significant (P < 0.05) reduction in the number of myelinated axons in the traumatized hemisphere compared to control in all structures was observed. In addition, thalamic and cortical axonal counts decreased significantly (P < 0.05) over time. Swollen axons and macrophage/microglia infiltration were present as late as 6 months post-TBI in various structures. This study is the first to describe quantitatively chronic axonal changes in vulnerable brains regions after injury. Based on these data, a time-dependent decrease in the number of myelinated axons is seen to occur in vulnerable gray matter regions including the cerebral cortex and thalamus along with distinct morphological changes within white matter tracts after TBI. Although this progressive axonal response to TBI may include Wallerian degeneration, other potential mechanisms underlying this progressive pathological response within the white matter are discussed.

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WC Clinical Neurology; Neurosciences; Pathology

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SC Neurosciences & Neurology; Pathology

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AU Cernak, I

Stoica, B

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Faden, AI

AF Cernak, I

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TI Role of the cell cycle in the pathobiology of central nervous system

trauma

SO CELL CYCLE

LA English

DT Article

DE cell cycle; apoptosis; neuronal injury; traumatic brain injury;

astrocyte proliferation; microglial proliferation; neuroprotection;

flavopiridol

ID BLOOD-BRAIN-BARRIER; SPINAL-CORD-INJURY; STIMULATED HOMOLOGOUS

MACROPHAGES; KINASE INHIBITOR FLAVOPIRIDOL; IN-VITRO;

ALZHEIMERS-DISEASE; DEPENDENT KINASES; MICROGLIAL ACTIVATION; NEURONAL

APOPTOSIS; PARKINSONS-DISEASE

AB Upregulation of cell cycle proteins occurs in both mitotic and post-mitotic neural cells after central nervous system (CNS) injury in adult animals. In mitotic cells, such as astroglia and microglia, they induce proliferation, whereas in post-mitotic cells such as neurons they initiate caspase-related apoptosis. We recently reported that early central administration of the cell cycle inhibitor flavopiridol after experimental traumatic brain injury (TBI) significantly reduced lesion volume, scar formation and neuronal cell death, while promoting near complete behavioral recovery. Here we show that in primary neuronal or astrocyte cultures structurally different cell cycle inhibitors ( flavopiridol, roscovitine, and olomoucine) significantly reduce upregulation of cell cycle proteins, attenuate neuronal cell death induced by etoposide, and decrease astrocyte proliferation. Flavopiridol, in a concentration dependent manner, also attenuates proliferation/ activation of microglia. In addition, we demonstrate that central administration of flavopiridol improves functional outcome in dose-dependent manner after fluid percussion induced brain injury in rats. Moreover, delayed systemic administration of flavopiridol significantly reduces brain lesion volume and edema development after TBI. These data provide further support for the therapeutic potential of cell cycle inhibitors for the treatment of clinical CNS injury and that protective mechanisms likely include reduction of neuronal cell death, inhibition of glial proliferation and attenuation of microglial activation.

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NR 84

TC 98

Z9 108

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PU TAYLOR & FRANCIS INC

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J9 CELL CYCLE

JI Cell Cycle

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WC Cell Biology

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SC Cell Biology

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AU Zhang, ZR

Artelt, M

Bernet, M

Trautmann, K

Schluesener, HJ

AF Zhang, ZR

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Trautmann, K

Schluesener, HJ

TI Lesional accumulation of P2X<sub>4</sub> receptor<SUP>+</SUP> monocytes

following experimental traumatic brain injury

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE P2X(4) receptor; traumatic brain injury; microglia/macrophages

ID SPINAL MICROGLIA; UP-REGULATION; CELL-DEATH; ATP; ACTIVATION;

MACROPHAGES; MECHANISMS; EXPRESSION; STRIATUM; RELEASE

AB P2X(4) receptor (P2X(4)R) is an ATP-gated ion channel. ATP is an important messenger in traumatic brain injury. Here, we report expression of P2X(4)R in rat traumatic brain injury with focus on the early phase, most amenable to therapy. Accumulation of P2X(4)R(+) cells was observed as early as 6 h after injury and continued to increase 4 days post-injury at the lesion and remote areas. Double staining revealed that most P2X(4)R(+) cells coexpressed ED-1, a marker for reactive microglia/macrophages, but not nestin or W3/13. Our data suggest that P2X(4)R expression defines a Subtype of activated microglia/macrophages involved in the early processes following traumatic brain injury. (c) 2005 Elsevier Inc. All rights reserved.

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AF Zhang, Z.

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TI Early infiltration of CD8<SUP>+</SUP> macrophages/microglia to lesions

of rat traumatic brain injury

SO NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury; microglia; macrophages; weight-drop model;

endothelial monocyte-activating polypeptide II

ID EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS; HEMATOGENOUS MACROPHAGES;

P2X(4) RECEPTOR; EMAP-II; EXPRESSION; MICROGLIA; MONOCYTES; ACTIVATION;

ACCUMULATION; RECRUITMENT

AB Local inflammatory responses play an important role in mediating secondary tissue damage in traumatic brain injury. Characterization of leukocytic subpopulations contributing to the early infiltration of the damaged tissue might aid in further understanding of lesion development and contribute to definition of cellular targets for selective immunotherapy. In a rat traumatic brain injury model, significant CD8(+) cell accumulation was observed 3 days post-injury. The CD8(+) cells were strictly distributed to the pannecrotic areas and around the pannecrotic perimeter. The morphology, time course of accumulation and distribution of CD8(+) cells were similar to that of reactive ED1(+) and enclothelial monocyteactivating polypepticle II+ microglia/macrophages, but different from W3/13(+) T cells. Further double-labeling experiments confirmed that the major cellular sources of CD8 were reactive macrophages/microglia. Both the location of these CD8(+) macrophages/microglia to the border of the pannecrosis and their co-expression of endothelial monocyte-activating polypeptide II and P2X(4) receptor suggest they might have a central role in lesion development and might thus be candidates fordevelopment of immunotherapeutic, anti-inflammatory strategies. (c) 2006 IBRO. Published by Elsevier Ltd. All rights reserved.

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Ninnemann, O

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Nitsch, R

Hendrix, S

TI Macrophage/microglia activation factor expression is restricted to

lesion-associated microglial cells after brain trauma

SO GLIA

LA English

DT Article

DE entorhinal cortex lesion; deafferentiation; CD11b; 1B4

ID ENTORHINAL CORTEX LESION; DENTATE GYRUS; MULTIPLE-SCLEROSIS; ATHYMIC

MICE; INJURY; CNS; IDENTIFICATION; MACROPHAGES; ASTROCYTES; CLONING

AB After traumatic brain lesion, microglial cells are rapidly activated, migrate toward the sites of injury, and cause secondary damage that accounts for most of the loss of brain function. In the present study, we have characterized a new macrophage/microglia activation factor (MA-F). Using the monocytic cell line U937, we were able to demonstrate that MAF is upregulated after TPA-induced differentiation into macrophages. We have generated a specific antibody against MAY In BV-2 microglial cells, MAF is partially co-localized with 1134, a classical microglial marker. In addition, we have analyzed the in vivo expression patterns of MAF after entorhinal cortex lesion. We were able to show a substantial upregulation of MAF on selected CD11b(+) and IB4(+) macrophages/microglial cells in the deafferented hippocampus and in the perilesional region, while no MAF expression was detectable on the contralateral side. Confocal microscopy revealed a lysosome-like expression pattern in BV-2 cells, as well as in ECL-associated macrophages/microglial cells in vivo. Furthermore, we were able to demonstrate that U937 cells with downregulated MAF converted slower and to a significantly reduced extent to the macrophageal phenotype after TPA treatment. In addition, MAF downregulation in BV-2 microglial cells substantially reduced the phagocytotic uptake of dextran beads. Our data indicate that MAF is expressed in selected macrophages/microglial cells around the lesion and in the degenerating hippocampus after ECL. Furthermore, MAF expression in monocytic cells seems to play a functional role in the differentiation to a phagocytosing phenotype and may be, at least partially, required for phagocytotic activity, specifically in lesioned tissue after brain trauma. (c) 2005 Wiley-Liss, Inc.

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TI Differential regulation of the CXCR2 chemokine network in rat brain

trauma:: Implications for neuroimmune interactions and neuronal survival

SO NEUROBIOLOGY OF DISEASE

LA English

DT Article

DE brain injury; CXCL1; CINC-1; KC; CXCL2; MIP-2; CXCR2; leukocytes;

microglia; neuroprotection

ID MACROPHAGE-INFLAMMATORY PROTEIN-2; CONTROLLED CORTICAL IMPACT;

CENTRAL-NERVOUS-SYSTEM; MARROW-DERIVED CELLS; BONE-MARROW; MICROGLIAL

CELLS; CONSTITUTIVE EXPRESSION; NONPEPTIDE ANTAGONIST; INTERNATIONAL

UNION; IDENTIFICATION

AB Chemokine receptors represent promising targets to attenuate inflammatory responses and subsequent secondary damage after brain injury. We studied the response of the chemokines CXCL1/CINC-1 and CXCL2/MIP-2 and their receptors CXCR1 and CXCR2 after controlled cortical impact injury in adult rats. Rapid upregulation of CXCL1/CINC-1 and CXCL2/MIP-2, followed by CXCR2 (but not CXCR1), was observed after injury. Constitutive neuronal CXCR2 immunoreactivity was detected in several brain areas, which rapidly but transiently downregulated upon trauma. A second CXCR2-positive compartment, mainly colocalized with the activated microglia/macrophage marker ED1, was detected rapidly after injury in the ipsilateral cortex, progressively emerging into deeper areas of the brain later in time. It is proposed that CXCR2 has a dual role after brain injury: (i) homologous neuronal CXCR2 downregulation would render neurons more vulnerable to injury, whereas (ii) chemotaxis and subsequent differentiation of blood-borne cells into a microglial-like phenotype would be promoted by the same receptor. (c) 2005 Elsevier Inc. All rights reserved.

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TI Injury severity determines Purkinje cell loss and microglial activation

in the cerebellum after cortical contusion injury

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE traumatic brain injury; controlled cortical impact; neuronal loss;

cerebellum; activated microglia; Purkinje cells

ID TRAUMATIC BRAIN-INJURY; FLUID PERCUSSION INJURY; MORRIS WATER MAZE;

IMPACT INJURY; COGNITIVE DEFICITS; HEAD-INJURY; NEURONAL INJURY;

TIME-COURSE; RAT; MODEL

AB Clinical evidence suggests that the cerebellum is damaged after traumatic brain injury (TBI) and experimental studies have validated these observations. We have previously shown cerebellar vulnerability, as demonstrated by Purkinje cell loss and microglial activation, after fluid percussion brain injury. In this study, we examine the effect of graded controlled cortical impact (CCI) injury on the cerebellum in the context of physiologic and anatomical parameters that have been shown by others to be sensitive to injury severity. Adult male rats received mild, moderate, or severe CCI and were euthanized 7 days later. We first validated the severity of the initial injury using physiologic criteria, including apnea and blood pressure, during the immediate postinjury period. Increasing injury severity was associated with an increased incidence of apnea and higher mortality. Severe injury also induced transient hypertension followed by hypotension, while lower grade injuries produced an immediate and sustained hypotension. We next evaluated the pattern of subcortical neuronal loss in response to graded injuries. There was significant neuronal loss in the ipsilateral cortex, hippocampal CA2/CA3, and laterodorsal thalamus that was injury severity-dependent and that paralleled microglial activation. Similarly, there was a distinctive pattern of Purkinje cell loss and microglial activation in the cerebellar vermis that varied with injury severity. Together, these findings emphasize the vulnerability of the cerebellum to TBI. That a selective pattern of Purkinje cell loss occurs regardless of the type of injury suggests a generalized response that is a likely determinant of recovery and a target for therapeutic intervention. (c) 2006 Elsevier Inc. All rights reserved.

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TI Tacrolimus depresses local immune cell infiltration but fails to reduce

cortical contusion volume in brain-injured rats

SO IMMUNOBIOLOGY

LA English

DT Article

DE traumatic brain injury; ICAM-1; microglia; macrophages; granulocytes

ID NECROSIS-FACTOR-ALPHA; CLOSED-HEAD INJURY; FLUID PERCUSSION INJURY;

NITRIC-OXIDE SYNTHASE; SPINAL-CORD-INJURY; CEREBRAL-ISCHEMIA; NEUTROPHIL

INFILTRATION; NEUROPROTECTIVE ACTION; INFLAMMATORY RESPONSE;

MOLECULAR-MECHANISMS

AB The immunosuppressant drug tacrolimus (FK-506) failed to show an anti-edematous effect despite suppressing pro-inflammatory cytokines in cerebrospinal fluid following focal traumatic brain injury. By questioning the role of the inflammatory response as a pharmacological target, we investigated the effects of FK-506 on immune cell infiltration in brain-injured rats.

Following induction of a cortical contusion, male Sprague-Dawley rats received FK-506 or physiological saline intraperitoneally. Brains were removed at 24 h, 72 h or 7 days, respectively. Frozen brain sections (7 mu m) were stained immunohistologically for markers of endothelial activation (intercellular adhesion molecule-1-ICAM-1), neutrophil infiltration (His-48), and microglial and macrophage activation (Ox-6; ED-1), respectively. Immunopositive cells were counted microscopically. Contusion volume (CV) was quantified morphometrically 7 days after trauma.

Inflammatory response was confined to the ipsilateral cortex and hippocampal formation, predominating in the contusion and pericontusional cortex. Strongest ICAM-1 expression coincided with sustained granulocyte accumulation at 72 h which was suppressed by FK-506. Ox-6 + cells prevailing at 72 h were also significantly reduced by FK-506. ED-1 + cells reaching highest intensity at 7 days were significantly attenuated at 72 h. Cortical CV was not influenced.

FK-506 significantly decreased post-traumatic local inflammation which, however, was not associated with a reduction in cortical CV. These results question the importance of post-traumatic local immune cell infiltration in the secondary growth of a cortical contusion. (c) 2007 Elsevier GmbH. All rights reserved.

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TI Metallothionein I and II attenuate the thalamic microglial response

following traumatic axotomy in the immature brain

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE inflicted childhood neurotrauma; mice; microglial activation; negative

binomial regression; secondary injury

ID FOCAL CEREBRAL-ISCHEMIA; CENTRAL-NERVOUS-SYSTEM; INFLICTED HEAD-INJURY;

CELL-DEATH; RAT-BRAIN; DOPAMINERGIC-NEURONS; CEREBROSPINAL-FLUID;

MOLECULAR-MECHANISMS; SENSORIMOTOR CORTEX; PARKINSONS-DISEASE

AB The clinical manifestations of inflicted traumatic brain injury in infancy most commonly result from intracranial hemorrhage, axonal stretch and disruption, and cerebral edema. Often hypoxia ischemia is superimposed, leading to early forebrain and later thalamic neurodegeneration. Such acute and delayed cellular injury activates microglia in the CNS. Although activated microglia provide important benefits in response to injury, microglial release of reactive oxygen species can be harmful to axotomized neurons. We have previously shown that the antioxidants metallothionein I and II (MT I & II) promote geniculocortical neuronal survival after visual cortex lesioning. The purpose of this investigation was to determine the influence of MT I & II on the density and rate of thalamic microglial activation and accumulation following in vivo axotomy. We ablated the visual cortex of 10-day-old and adult MT I & II knock out (MT-/-) and wild-type mice and then determined the density of microglia in the dorsal lateral geniculate nucleus (dLGN) over time. Compared to the wild-type strain, microglial activation occurred earlier in both young and adult MT-/- mice. Similarly, microglial density was significantly greater in young MT-/- mice 30, 36, and 48 hours after injury, and 3, 4, and 5 days after injury in MT-/- adults. In both younger and older mice, time and MT I & II deficiency each contributed significantly to greater microglial density. Only in younger mice did MT I & II expression significantly slow the rate (density X time) of microglial accumulation. These results suggest that augmentation of NIT I & II expression may provide therapeutic benefits to infants with inflicted brain injury.

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TC 15

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U2 0

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J9 J NEUROTRAUM

JI J. Neurotrauma

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WC Critical Care Medicine; Clinical Neurology; Neurosciences

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ER

PT J

AU Koshinaga, M

Suma, T

Fukushima, M

Tsuboi, I

Aizawa, S

Katayama, Y

AF Koshinaga, M.

Suma, T.

Fukushima, M.

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Aizawa, S.

Katayama, Y.

TI Rapid microglial activation induced by traumatic brain injury is

independent of blood brain barrier disruption

SO HISTOLOGY AND HISTOPATHOLOGY

LA English

DT Article

DE microglia; blood brain barrier; brain injury; CR3

ID SPREADING DEPRESSION; RAT; CELLS; DAMAGE; ASTROCYTES; EXPRESSION;

ISCHEMIA; RELEASE

AB Following CNS injury, microglia respond and transform into reactive species exhibiting characteristic morphological changes that have been termed "activated" or "ameboid" microglia. In an attempt to establish that microglial reactions induced immediately after injury are caused by intrinsic mechanisms rather than infiltration of blood and its constituents, oxygenized Ringer's solution was perfused into the cerebral circulation of rats so that the circulating blood could be eliminated prior to injury induction. Under artificial respiration, a catheter was inserted from the cardiac apex into the ascending aorta, and oxygenized Ringer's solution was immediately perfused with a pulsatile blood pump, resulting in wash out of the circulating blood from the brain within 1 min. Subsequently, a cortical contusion was induced in the unilateral parietal cortex using a controlled cortical impact (CCI) device. At 5 min following the injury, the brain was fixed by perfusion of fixative through the catheter and removed. Coronal vibratome sections were then processed for CR3 immunohistochemistry to examine the microglial activation. It appeared that microglial activation with both morphological transformation and an increase in CR3 immunoreactivity was induced throughout the hemisphere ipsilateral to the injury side exclusively, even in rats with elimination of circulating blood. The microglial reactions did not differ substantially from those observed in the control rats with extensive BBB disruption. The present results thus provide direct evidence that the microglial activation induced immediately after injury is independent of infiltration of circulating blood induced by concurrent BBB disruption.

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NR 27

TC 12

Z9 17

U1 0

U2 0

PU F HERNANDEZ

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J9 HISTOL HISTOPATHOL

JI Histol. Histopath.

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WC Cell Biology; Pathology

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SC Cell Biology; Pathology

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ER

PT J

AU Redell, JB

Dash, PK

AF Redell, John B.

Dash, Pramod K.

TI Traumatic brain injury stimulates hippocampal catechol-<i>O</i>-methyl

transferase expression in microglia

SO NEUROSCIENCE LETTERS

LA English

DT Article

DE protein array; microarray; proteomics; dopamine; COMT; TBI

ID RAT FRONTAL-CORTEX; HYPOXIA-INDUCIBLE FACTOR-1-ALPHA; CONTROLLED

CORTICAL IMPACT; DOPAMINE TRANSPORTER; PROTEIN EXPRESSION; PREFRONTAL

CORTEX; WORKING-MEMORY; METHYLTRANSFERASE; TECHNOLOGY; GENOTYPE

AB Outcome following traumatic brain injury (TBI) is in large part determined by the combined action of multiple processes. In order to better understand the response of the central nervous system to injury, we utilized an antibody array to simultaneously screen 507 proteins for altered expression in the injured hippocampus, a structure critical for memory formation. Array analysis indicated 41 candidate proteins have altered expression levels 24h after TBI. Of particular interest was catechol-O-methyl transferase (COMT), an enzyme involved in metabolizing catecholamines released following neuronal activity. Altered catecholamine signaling has been observed after brain injury, and may contribute to the cognitive dysfunctions and behavioral deficits often experienced after TBI. Our data shows that COMT expression in the injured ipsilateral hippocampus was elevated for at least 14 d after controlled cortical impact injury. We found strong co-localization of COMT immunoreactivity with the microglia marker lbal near the injury site. Since dopamine transporter expression has been reported to be down-regulated after brain injury, COMT-mediated catecholamine metabolism may play a more prominent role in terminating catecholamine signaling in injured areas. (c) 2006 Elsevier Ireland Ltd. All rights reserved.

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Z9 47

U1 0

U2 1

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PT J

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TI Transient neuroprotection by minocycline following traumatic brain

injury is associated with attenuated microglial activation but no

changes in cell apoptosis or neutrophil infiltration

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE apoptosis; cytokines; inflammation; microglia; minocycline; traumatic

brain injury

ID CLOSED-HEAD INJURY; INTERLEUKIN-1 RECEPTOR ANTAGONIST; FOCAL

CEREBRAL-ISCHEMIA; PROINFLAMMATORY CYTOKINE EXPRESSION; IMPROVES

FUNCTIONAL RECOVERY; CYTOCHROME-C RELEASE; SPINAL-CORD-INJURY; MOUSE

MODEL; TNF-ALPHA; DEATH

AB Cerebral inflammation and apoptotic cell death are two processes implicated in the progressive tissue damage that occurs following traumatic brain injury (TBI), and strategies to inhibit one or both of these pathways are being investigated as potential therapies for TBI patients. The tetracycline derivative minocycline was therapeutically effective in various models of central nervous system injury and disease, via mechanisms involving suppression of inflammation and apoptosis. We therefore investigated the effect of minocycline in TBI using a closed head injury model. Following TBI, mice were treated with minocycline or vehicle, and the effect on neurological outcome, lesion volume, inflammation and apoptosis was evaluated for up to 7 days. Our results show that while minocycline decreases lesion volume and improves neurological outcome at I day post-trauma, this response is not maintained at 4 days. The early beneficial effect is likely not due to anti-apoptotic mechanisms, as the density of apoptotic cells is not affected at either time-point. However, protection by minocycline is associated with a selective anti-inflammatory response, in that microglial activation and interleukin-1 beta expression are reduced, while neutrophil infiltration and expression of multiple cytokines are not affected. These findings demonstrate that further studies on minocycline in TBI are necessary in order to consider it as a novel therapy for brain-injured patients. Crown Copyright (c) 2006 Published by Elsevier Inc. All rights reserved.

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NR 54

TC 197

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U2 23

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ER

PT J

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TI Antibodies to CD11b, CD68, and lectin label neutrophils rather than

microglia in traumatic and ischemic brain lesions

SO JOURNAL OF NEUROSCIENCE RESEARCH

LA English

DT Article

DE MCAO; stab wound; microglia; macrophage; Iba1; NO; proinflammatory

cytokines

ID CEREBRAL-ARTERY OCCLUSION; NITRIC-OXIDE SYNTHASE; SPINAL-CORD-INJURY;

NG2 PROTEOGLYCAN; FOCAL ISCHEMIA; CELLS; EXPRESSION; REPERFUSION;

CORTEX; OLIGODENDROCYTES

AB Resident quiescent microglia have been thought to respond rapidly to various pathologic events in the brain by proliferating and producing many bioactive substances, including proinflammatory cytokines and nitric oxide (NO). In this study, we investigated the reaction of microglia in traumatic and ischemic lesions caused by stab wounds and the transient 90-min occlusion of middle cerebral artery in a mature rat brain. Although many lba1(+) resident microglia underwent apoptotic degeneration in the lesion core within 24 hr after the onset of the brain insult as revealed by TUNEL staining, numerous small, round, isolectin B4(+)/CD11b(+)/CD68(+) cells were localized in the lesion core. These small, round cells with diameters of 7-9 mu m and polymorph nuclei expressed neutrophil-specific elastase, alkaline phosphatase, and platelet-activating factor receptor. Accordingly, they were not activated microglia but neutrophils. Immunohistochemical staining with antibodies to inducible NO synthase (iNOS) showed that most iNOS(+) cells were neutrophils. The results from spatial and kinetic analyses using RT-PCR and immunoblotting were consistent with the immunohistochemical observations. These results suggest the necessity of reevaluating the traditional view on the roles of activated microglia in severe neuropathologic events. Note that the traditional microglial markers isolectin B4, CD11b, and CD68 are not specific for microglia, particularly in a pathologic brain. (c) 2007 Wiley-Liss, Inc.

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TI Expression of EAAT1 reflects a possible neuroprotective function of

reactive astrocytes and activated microglia following human traumatic

brain injury

SO HISTOLOGY AND HISTOPATHOLOGY

LA English

DT Article

DE microglial activation; neuroprotection; traumatic brain injury;

excitatory amino acid transporters

ID GLIAL GLUTAMATE TRANSPORTER; AMINO-ACID TRANSPORTER-1; FOCAL

CEREBRAL-ISCHEMIA; GLT-1 EXPRESSION; CELL-DEATH; CLINICAL-TRIALS;

CORTICAL IMPACT; MICE LACKING; SPINAL-CORD; RAT MODEL

AB Glutamate-mediated excitotoxicity is known to cause secondary brain damage following stroke and traumatic brain injury (TBI). However, clinical trials using NMDA antagonists failed. Thus, glial excitatory amino acid transporters (EAATs) might be a promising target for therapeutic intervention. Methods and Results. We examined expression of EAAT1 (GLAST) and EAAT2 (Glt-1) in 36 TBI cases by immunohistochemistry. Cortical expression of both EAATs decreased rapidly and widespread throughout the brain (in lesional, adjacent and remote areas) following TBI. In the white matter numbers of EAAT1+ parenchymal cells increased 39-fold within 24h (p < 0.001) and remained markedly elevated till later stages in the lesion (90-fold, p < 0.01) and in peri-lesional regions (86-fold, p < 0.01). In contrast, EAAT2+ parenchymal cells and EAAT1+ or EAAT2+ perivascular cells did not increase significantly. Within the first days following TBI mainly activated microglia and thereafter mainly reactive astrocytes expressed EAAT1. Perivascular monocytes and foamy macrophages lacked EAAT1 immunoreactivity. We conclude that following TBI i) loss of cortical EAATs contributes to secondary brain damage, ii) glial EAAT1 expression reflects a potential neuroprotective function of microglia and astrocytes, iii) microglial EAAT1 expression is restricted to an early stage of activation, iv) blood-derived monocytes do not express EAAT1 and v) pharmacological modification of glial EAAT expression might further limit neuronal damage.

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TI Dexamethasone attenuates early expression of three molecules associated

with microglia/macrophages activation following rat traumatic brain

injury

SO ACTA NEUROPATHOLOGICA

LA English

DT Article

DE dexamethasone; traumatic brain injury; EMAP-II; P2X4R; AIF-1;

microglia/macrophages

ID INFLAMMATORY FACTOR-I; NECROSIS-FACTOR-ALPHA; SPINAL-CORD-INJURY;

EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS; NITRIC-OXIDE SYNTHASE;

MICROGLIAL CELLS; POLYPEPTIDE-II; MACROPHAGES/MICROGLIAL CELLS; RECEPTOR

ACTIVATION; LESIONAL EXPRESSION

AB Corticosteroids have been used in the treatment of human traumatic brain injury ( TBI), which is a leading cause of death and disability, but their efficiency is still a matter of debate. Dexamethasone was considered to delay post-traumatic inflammation and retard neuronal degeneration, resulting in attenuation of secondary injury following experimental TBI. In a rat TBI model, we have investigated the effects of dexamethasone on expression patterns of markers of inflammatory activation of microglia/macrophages by immunohistochemistry. Endothelial-monocyte activating polypeptide II (EMAP-II), P2X4 receptor (P2X4R) and allograft-inflammatory factor-1 (AIF-1) were reported to be associated with the activation of microglia/macrophages post central nervous system (CNS) injury and may play roles in inflammatory cascades of secondary brain damage. Dexamethasone significantly suppressed the accumulation of EMAP-II+, P2X4R(+) or AIF(+) cells at Day-1 and 2 post-brain-trauma but not on Days 4 and 6, which is in accordance with the reported short- but not long-term protective effects of dexamethasone in TBI. These findings indicate a rather rapid but transient anti-inflammatory effect of dexamethasone in TBI.

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PT J

AU Kurpius, D

Nolley, EP

Dailey, ME

AF Kurpius, Dana

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Dailey, Michael E.

TI Purines induce directed migration and rapid homing of microglia to

injured pyramidal neurons in developing hippocampus

SO GLIA

LA English

DT Article

DE brain slice; ATP; time-lapse; motility; migration; CNS trauma

ID RAT-BRAIN; IN-VITRO; EXTRACELLULAR NUCLEOTIDES; P2Y(12) RECEPTOR;

NERVOUS-SYSTEM; CELL-MIGRATION; SLICE CULTURES; GLIAL-CELLS; ACTIVATION;

EXPRESSION

AB Traumatic CNS injury activates and mobilizes resident parenchymal microglia (MG), which rapidly accumulate near injured neurons where they transform into phagocytes. The mechanisms underlying this rapid 'homing' in situ are unknown. Using time-lapse confocal imaging in acutely excised neonatal hippocampal slices, we show that rapid accumulation of MG near somata of injured pyramidal neurons in the stratum pyramidale (SP) results from directed migration from tissue regions immediately adjacent to (< 200 mu m from) the SP. Time-lapse sequences also reveal a 'spreading activation wave' wherein MG situated progressively farther from the SP begin to migrate later and exhibit less directional migration toward the SP Because purines have been implicated in MG activation and chemotaxis, we tested whether ATP/ADP released from injured pyramidal neurons might account for these patterns of MG behavior. Indeed, application of apyrase, which degrades extracellular ATP/ADP, inhibits MG motility and homing to injured neurons in the SP Moreover, bath application of exogenous ATP/ADP disrupts MG homing by inducing directional migration toward the slice exterior and away from injured neurons. These results indicate that extracellular ATP/ADP is both necessary and sufficient to induce directional migration and rapid homing of neonatal MG to injured neurons in situ. Rapid, ATP/ADP-dependent MG homing may promote clearance of dead and dying cells and help limit secondary damage during the critical first few hours after neuronal injury. (c) 2007 Wiley-Liss, Inc.

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U2 3

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PT J

AU Ellis, EF

Willoughby, KA

Sparks, SA

Chen, T

AF Ellis, Earl F.

Willoughby, Karen A.

Sparks, Sallie A.

Chen, Tao

TI S100B protein is released from rat neonatal neurons, astrocytes, and

microglia by <i>in vitro</i> trauma and anti-S100 increases

trauma-induced delayed neuronal injury and negates the protective effect

of exogenous S100B on neurons

SO JOURNAL OF NEUROCHEMISTRY

LA English

DT Article

DE S100B; neuroprotection; traumatic brain injury

ID STRETCH-INDUCED INJURY; CULTURED ASTROCYTES; COGNITIVE RECOVERY;

EXPRESSION; INFUSION; DAMAGE; CELLS; ATP

AB S100B protein is found in brain, has been used as a marker for brain injury and is neurotrophic. Using a well-characterized in vitro model of brain cell trauma, we have previously shown that strain injury causes S100B release from neonatal rat neuronal plus glial cultures and that exogenous S100B reduces delayed post-traumatic neuronal damage even when given at 6 or 24 h post-trauma. The purpose of the current studies was to measure post-traumatic S100B release by specific brain cell types and to examine the effect of an antibody to S100 on post-traumatic delayed (48 h) neuronal injury and the protective effect of exogenous S100B. Neonatal rat cortical cells grown on a deformable elastic membrane were subjected to a strain (stretch) injury produced by a 50 ms displacement of the membrane. S100B was measured with an ELISA kit. Trauma released S100B from pure cultures of astrocytes, microglia, and neurons. Anti-S100 reduced released S100B to below detectable levels, increased delayed neuronal injury in traumatized cells and negated the protective effect of exogenous S100B on injured cells. Heat denatured anti-S100 did not exacerbate injury. These studies provide further evidence for a protective role for S100B following neuronal trauma.

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Robbins, M

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TI Multi-modal magnetic resonance imaging alterations in two rat models of

mild neurotrauma

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE immunohistochemistry; apparent diffusion coefficient; T2 weighted

imaging; fluid percussion; controlled cortical impact; traumatic brain

injury; microglia; astrocytes

ID TRAUMATIC BRAIN-INJURY; LATERAL FLUID PERCUSSION; CORTICAL IMPACT

INJURY; CLOSED-HEAD INJURY; TIME-COURSE; DIFFUSION; EDEMA; HISTOLOGY;

HIPPOCAMPUS; PERFUSION

AB Magnetic resonance imaging (MRI) is increasingly used in the assessment of the severity and progression of neurotrauma. We evaluated temporal and regional changes after mild fluid percussion (FPI) and controlled cortical impact (CCI) injury using T2-weighted-imaging (T2WI) and diffusion-weighted imaging (DWI) MRI over 7 days. Region of interest analysis of brain areas distant to the injury site (such as the hippocampus, retrosplenial and piriform cortices, and the thalamus) was undertaken. In the hippocampus of CCI animals, we found a slow increase (51%) in apparent diffusion coefficients (ADC) over 72 h, which returned to control values. The hippocampal T2 values in the CCI animals were elevated by 18% over the 7-day time course compared to control, indicative of edema formation. Histological analysis supported the lack of overt cellular loss in most brain regions after mild CCI injury. FPI animals showed a generalized decrease in hippocampal ADC values over the first 72 h, which then returned to sham levels, with decreased T2 values over the same period, which remained depressed at 7 days. Histological assessment of FPI animals revealed numerous shrunken cells in the hippocampus and thalamus, but other regions showed little damage. Increased immunohistochemical staining for microglia and astroglia at 7 days post-injury was greater in FPI animals within the affected brain regions. In summary, traumatic brain injury is less severe in mild CCI than FPI, based on the temporal events assessed with MRI.

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J9 J NEUROTRAUM

JI J. Neurotrauma

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ER

PT J

AU Zhang, ZY

Zhang, Z

Fauser, U

Artelt, M

Burnet, M

Schluesener, HJ

AF Zhang, Z.-Y.

Zhang, Z.

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Artelt, M.

Burnet, M.

Schluesener, H. J.

TI Dexamethasone transiently attenuates up-regulation of

endostatin/collagen XVIII following traumatic brain injury

SO NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury; endostatin/collagen XVIII;

microglia/macrophages; dexamethasone

ID NECROSIS-FACTOR-ALPHA; CLOSED-HEAD INJURY; ANGIOGENESIS INHIBITOR;

TUMOR-GROWTH; EXPRESSION; VEGF; RAT; DEGENERATION; CONTUSIONS; PROTEINS

AB Endostatin/collagen XVIII is a specific inhibitor of endothelial proliferation and migration in vitro. It has also been shown to have anti-angiogenic activity and tumor growth inhibitory activity in vivo and in vitro. Here we studied expression of endostatin/collagen XVIII in a rat traumatic brain injury (TBI) model, focusing on the early phase. A significant up-regulation of endostatin/collagen XVIII in TBI began as early as 24 h post-TBI. Double-staining experiment revealed that the major resource of endostatin/collagen XVIII+ cells in our TBI rat model was a subpopulation of reactivated microglia/macrophages. Our data further showed that dexamethasone attenuated up-regulation of endostatin/collagen XVIII expression at days 1 and 2, but not at day 4, post-TBI, indicating that dexamethasone might possess an early and transient influence to the angiogenesis following TBI. (c) 2007 IBRO. Published by Elsevier Ltd. All rights reserved.

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U2 2

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PT J

AU Tlaskowitz, D

Vitek, MP

AF Tlaskowitz, Daniel

Vitek, Michael P.

TI Apolipoprotein E and neurological disease: therapeutic potential and

pharmacogenomic interactions

SO PHARMACOGENOMICS

LA English

DT Review

DE apolipoprotein E; astrocytes; microglia; multiple; sclerosis;

neuroinflammation; stroke; subarachnoid; hemorrhage; traumatic; brain

injury

ID E-DEFICIENT MICE; PERMANENT FOCAL ISCHEMIA; GLOBAL CEREBRAL-ISCHEMIA;

CLOSED-HEAD INJURY; E EPSILON-4 ALLELE; E-BASED PEPTIDE; E GENOTYPE;

APOE GENOTYPE; ALZHEIMERS-DISEASE; E POLYMORPHISM

AB The apolipoprotein E (apoE) polymorphism is emerging as a uniquely important genetic modifier that affects functional outcome from both acute and chronic neurological injuries. Recent attention has focused on common denominator mechanisms by which apoE might affect brain injury and/or brain repair responses in clinically diverse diseases. Although endogenous apoE likely serves several adaptive functions in the injured CNS, there is growing evidence that its effect on modifying brain inflammatory responses and providing protection from excitotoxic injury may be central to its protective properties. A more complete understanding of the role that apoE plays in the injured brain has led to novel therapeutic strategies for both acute and chronic neurological disease.

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Zerbinatti CV, 2005, REV NEUROSCIENCE, V16, P123

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J9 PHARMACOGENOMICS

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AU Venneti, S

Wagner, AK

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TI The high affinity peripheral benzodiazepine receptor ligand DAA1106

binds specifically to microglia in a rat model of traumatic brain

injury: Implications for PET imaging

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE peripheral benzodiazepine receptor; microglia; PET imaging; traumatic

brain injury; DAA1106; PK11195

ID POSITRON-EMISSION-TOMOGRAPHY; CONTROLLED CORTICAL IMPACT; MILD COGNITIVE

IMPAIRMENT; IN-VIVO; ISCHEMIC-STROKE; ACTIVATED MICROGLIA;

MULTIPLE-SCLEROSIS; PARKINSONS-DISEASE; PK11195 BINDING; MOUSE-BRAIN

AB Traumatic brain injury (TBI) is a significant cause of mortality, morbidity, and disability. Microglial activation is commonly observed in response to neuronal injury which, when prolonged, is thought to be detrimental to neuronal survival. Activated microglia can be labeled using PK11195, a ligand that binds the peripheral benzodiazepine receptor (PBR), receptors which are increased in activated microglia and sparse in the resting brain. We compared the binding properties of two PBR ligands PK11195 and DAA1106 in rats using the controlled cortical impact (CCI) model of experimental TBI. While both ligands showed relative increases with specific binding in the cortex ipsitateral to injury compared to the contralateral side, [H-3]DAA1106 showed higher binding affinity compared with [H-3](R)-PK11195. Combined immunohistochemistry and autoradiography in brain tissues near the injury site showed that [H-3]DAA1106 binding co-registered with activated microglia more than astrocytes. Further, increased [H-3]DAA1106-specific binding positively correlated with the degree of microglial activation, and to a lesser degree with reactive astrocytosis. Finally, in vivo administration of each ligand in rats with TBI showed greater retention of [C-11]DAA1106 compared to [C-11](R)-PK11195 at the site of the contusion as assessed by ex vivo autoradiography. These results in a rat model of TBI indicate that [C-11] DAA1106 binds with higher affinity to microglia when compared with PK11195, suggesting that [C-11]DAA1106 may represent a better ligand than [C-11](R)-PK11195 for in vivo PET imaging of activated microglia in TBI. (c) 2007 Elsevier Inc. All rights reserved.

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TI Neuroinflammatory responses after experimental diffuse traumatic brain

injury

SO JOURNAL OF NEUROPATHOLOGY AND EXPERIMENTAL NEUROLOGY

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DT Article

DE fluid percussion; macrophage; microglia; rat; traumatic axonal injury

ID AXONAL INJURY; INFLAMMATORY RESPONSE; MICROGLIAL ACTIVATION;

NERVOUS-SYSTEM; CELL-DYNAMICS; BARRIER; NUCLEUS; AXOTOMY; MODEL; SITES

AB Little is known about microglial activation and macrophage localization after diffuse brain injury (DBI). DBI-mediated perisomatic traumatic axonal injury (TAI) was recently identified within the neocortex, hippocampus, and thalamus, providing an opportunity to characterize immune cell responses within diffusely injured brain loci uncomplicated by contusion. By using moderate midline/central fluid percussion injury, microglial/macrophage responses were examined with antibodies targeting immune cell phenotypes and amyloid precursor protein, a marker of TAI. Parallel assessments of blood-brain barrier alterations were also performed. Within 6 to 48 hours postinjury, microglial activation within injured loci was observed, whereas microglia within non-TAI-containing regions maintained a resting phenotype. Microglial activation shared a spatiotemporal relationship with TAI though no clear interactions were observed. By 7 to 28 days postinjury, activated microglia contained myelin debris, yet revealed limited aggregation. Immunophenotypic macrophages were also localized to injured loci. Select macrophages approximated somatic membranes of perisomatically axotomized neurons with evidence of bouton disruption. No causality was established between blood-brain barrier alterations and these inflammatory responses. These findings indicate rapid, yet initially nonspecific, and persistent microglial/macrophage responses to DBI. DBI-mediated inflammatory responses suggest further expansion of traumatic brain injury histopathologic evaluations to identify neuroinflammation indicative of diffuse pathology.

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TI Prolonged microgliosis in the rhesus monkey central nervous system after

traumatic brain injury

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE microglia; motor cortex; motor recovery; spinal cord; stroke

ID TRKB NEUROTROPHIN RECEPTORS; OUTGROWTH IN-VITRO; RAT SPINAL-CORD;

TRUNCATED TRKB; KAINIC ACID; ARM REPRESENTATION; MULTIPLE-SCLEROSIS;

NONHUMAN-PRIMATES; MOTOR CORTEX; RECOVERY

AB Impaired fine motor functions after traumatic brain injury (TBI) in humans and non-human primates often continue to improve months after injury. To initiate a series of studies in the primate model designed to investigate possible involvement of microglia/macrophage in the long-term recovery processes, changes in these cells were studied in the rhesus monkey central nervous system at 1, 6, and 12 months after a combined unilateral lesion of the arm area of the primary motor cortex and arm area of the lateral premotor cortex. Immunohistological studies showed profound CD68 immunoreactivity in the lesion area and the contralateral lateral corticospinal tract in the spinal cord at all time points, demonstrating that microglia/macrophage remain reactive at the sites of injury and axonal degeneration/survival for at least 12 months. We also observed marked increases in brain-derived neurotrophic factor (BDNF) and its receptor subtypes, TrkB[gp145] and TrkB[TK-], around the cortical lesion site after 6-month survival. Similar increases were also observed in the spinal cord, although it was less apparent for TrkB[gp145]. Double-labeling revealed that a sub-population of CD68-immunoreacitve microglia/macrophage co-expressed BDNF in the cortex and spinal cord, and also TrkB[gp145] or TrkB[TK-] in the spinal cord. In contrast, cytokine expression of tumor necrosis factor-alpha (TNF-alpha), interleukin-1 beta (IL-1 beta), and interleukin-6 (IL-6) at these time intervals was less prominent, suggesting that immediate inflammatory responses had subsided. These results demonstrate that microglia/macrophage undergo prolonged activation after TBI in the non-human primate brain and express BDNF and its receptors, suggesting their tropic/trophic roles in the long-term recovery processes.

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J9 J NEUROTRAUM

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WC Critical Care Medicine; Clinical Neurology; Neurosciences

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PT J

AU Zhang, ZY

Zhang, ZR

Fauser, U

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AF Zhang, Zhi-Yuan

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Fauser, Uwe

Schluesener, Hermann J.

TI Global hypomethylation defines a sub-population of reactive

microglia/macrophages in experimental traumatic brain injury

SO NEUROSCIENCE LETTERS

LA English

DT Article

DE traumatic brain injury; hypomethylation; 5-methylcytosine;

microglia/macrophages; dexamethasone

ID DNA METHYLATION; HEAD-INJURY; SPINAL-CORD; MICROGLIA; RATS; ACTIVATION;

EXPRESSION; CHROMATIN; IMPACT; CELLS

AB Global alterations in gene expression have been observed indifferent traumatic brain injury (TBI) models and are considered of crucial importance to the development of subsequent tissue injury and repair. Cytosine methylation is a well-known process of endogenous DNA modification in mammals and the primary mechanism responsible for changes in epigenetic gene expression. Here we have investigated the early global spatio-temporal changes of the status of cellular DNA methylation in a rat TBI model by immunohistochemistry and analyzed the effects of dexamethasone on these changes. Global cellular hypomethylation was seen as early as day 1 in pannecrosis and day 2 in peripannecrosis following TBI. A subpopulation of reactive microglia/macrophages was identified as the major source of hypomethylated cells by double-staining experiments. Further, peripheral administration of dexamethasone suppressed this lesional hypomethylation at day 2 post-injury. In sum, our data suggest that lesional hypomethylation defines a sub-population of activated microglia/macrophages involved in the early processes following traumatic brain injury. (c) 2007 Elsevier Ireland Ltd. All rights reserved.

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ER

PT J

AU Zhang, ZR

Fauser, U

Schluesener, HJ

AF Zhang, Zhiren

Fauser, Uwe

Schluesener, Hermann J.

TI Dexamethasone suppresses infiltration of RhoA<SUP>+</SUP> cells into

early lesions of rat traumatic brain injury

SO ACTA NEUROPATHOLOGICA

LA English

DT Article

DE RhoA; dexamethasone; traumatic brain injury; microglia; macrophages;

weight-drop model

ID CENTRAL-NERVOUS-SYSTEM; SEVERE HEAD-INJURY; EXPERIMENTAL AUTOIMMUNE

ENCEPHALOMYELITIS; SPINAL-CORD-INJURY; RHO-GTPASES; LEUKOCYTE

RECRUITMENT; CLOSTRIDIUM-BOTULINUM; ACTIN CYTOSKELETON; AXON

REGENERATION; ADP-RIBOSYLATION

AB Inflammatory cell infiltration is a major part of secondary tissue damage in traumatic brain injury (TBI). RhoA is an important member of Rho GTPases and is involved in leukocyte migration. Inhibition of RhoA and its downstream target, Rho-associated coiled kinase (ROCK), has been proven to promote axon regeneration and function recovery following injury in the central nervous system (CNS). Previously, we showed that dexamethasone, an immunosuppressive corticosteroid, attenuated early expression of three molecules associated with microglia/macrophages activation following TBI in rats. Here, the effects of dexamethasone on the early expression of RhoA have been investigated in brains of TBI rats by immunohistochemistry. In brains of rats treated with TBI alone, significant RhoA(+) cell accumulation was observed at 18 h post-injury and continuously increased during our observed time period. The accumulated RhoA(+) cells were distributed to the areas of pannecrosis and selective neuronal loss. Most accumulated RhoA(+) cells were identified as active microglia/macrophages by double-labelling. Dexamethasone (1 mg/kg body weight) was intraperitoneally injected on day 0 and 2 immediately following brain injury. Numbers of RhoA(+) cells were significantly reduced on day 1 and 2 following administration of dexamethasone but returned to vehicle control level on day 4. However, dexamethasone treatment did not change the proportion of RhoA(+) cells. These observations suggest that dexamethasone has only a transient effect on early leukocyte recruitment.

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Z9 15

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U2 6

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JI Acta Neuropathol.

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ER

PT J

AU Hailer, NP

AF Hailer, Nils P.

TI Immunosuppression after traumatic or ischemic CNS damage: It is

neuroprotective and illuminates the role of microglial cells

SO PROGRESS IN NEUROBIOLOGY

LA English

DT Review

DE spinal cord injury; cerebral ischemia; traumatic brain injury;

microglial cell; microglia; astrocyte; neuron; immunology;

immunosuppression; immunosuppressive drugs; neuroprotection;

cytosine-arabinoside; steroids; methylprednisolone; dexamethasone;

cyclosporin A; CsA; tacrolimus; FK506; rapamycin; sirolimus;

mycophenolate mofetil; mycophenolic acid; minocycline; clodronate; IL-1

receptor antagonist (IL-1ra)

ID SPINAL-CORD-INJURY; HIPPOCAMPAL SLICE CULTURES; FOCAL CEREBRAL-ISCHEMIA;

CENTRAL-NERVOUS-SYSTEM; INTERLEUKIN-1 RECEPTOR ANTAGONIST; ADHESION

MOLECULE EXPRESSION; COLONY-STIMULATING FACTOR; TUMOR-NECROSIS-FACTOR;

PERIPHERAL BENZODIAZEPINE RECEPTOR; IMPROVES FUNCTIONAL RECOVERY

AB Acute traumatic and ischemic events in the central nervous system (CNS) invariably result in activation of microglial cells as local representatives of the immune system. It is still under debate whether activated microglia promote neuronal survival, or whether they exacerbate the original extent of neuronal damage. Protagonists of the view that microglial cells cause secondary damage have proposed that inhibition of microglial activation by immunosuppression is beneficial after acute CNS damage. It is the aim of this review to analyse the effects of immunosuppressants on isolated microglial cells and neurons, and to scrutinize the effects of immunosuppression in different in vivo models of acute CNS trauma or ischemia. It is found that the immunosuppressants cytosine-arabinoside, different steroids, cyclosporin A, FK506, rapamycin, mycophenolate mofetil, and minocycline all have direct inhibitory effects on microglial cells. These effects are mainly exerted by inhibiting microglial proliferation or microglial secretion of neurotoxic substances such as proinflammatory cytokines and nitric oxide. Furthermore, immunosuppression after acute CNS trauma or ischemia results in improved structure preservation and, mostly, in enhanced function. However, all investigated immunosuppressants also have direct effects on neurons, and some immunosuppressants affect other glial cells such as astrocytes. In summary, it is safe to conclude that immunosuppression after acute CNS trauma or ischemia is neuroprotective. Furthermore, circumferential evidence indicates that microglial activation after traumatic or ischemic CNS damage is not beneficial to adjacent neurons in the immediate aftermath of such acute lesions. Further experiments with more specific agents or genetic approaches that specifically inhibit microglial cells are needed in order to fully answer the question of whether microglial activation is "good or bad". (c) 2007 Elsevier Ltd. All rights reserved.

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TI Increased expression of the γ-secretase components presenilin-1 and

nicastrin in activated astrocytes and microglia following traumatic

brain injury

SO GLIA

LA English

DT Article

DE closed head injury; brain-stabbing; presenilin; nicastrin; astrocytes;

microglia

ID CLOSED-HEAD-INJURY; AMYLOID PRECURSOR PROTEIN; FAMILIAL

ALZHEIMERS-DISEASE; BETA-SECRETASE; MOUSE-BRAIN; REACTIVE ASTROCYTES;

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RECEPTOR

AB gamma-Secretase is an aspartyl protease composed of four proteins: presenilin (PS), nicastrin (Net), APH1, and PEN2. These proteins assemble into a membrane complex that cleaves a variety of substrates within the transmembrane domain. The gamma-secretase cleavage products play an important role in various biological processes such as embryonic development and Alzheimer's disease (AD). The major role of gamma-secretase in brain pathology has been linked to AD and to the production of the amyloid P-peptide. However, little is known about the possible role of gamma-secretase following acute brain insult. Here we examined by immunostaining the expression patterns of two gamma-secretase components, PSI and Net, in three paradigms of brain insult in mice: closed head injury, intracerebroventricular injection of LPS, and brain stabbing. Our results show that in naive and sham-injured brains expression of PS1 and Net is restricted mainly to neurons. However, following insult, the expression of both proteins is also observed in nonneuronal cells, consisting of activated astrocytes and microglia. Furthermore, the proteins are coexpressed within the same astrocytes and microglia, implying that these cells exhibit an enhanced gamma-secretase activity following brain damage. In view of the important role played by astrocytes and microglia in brain disorders, our findings suggest that gamma-secretase may participate in brain damage and repair processes by regulating astrocyte and microglia activation and/or function. (C) 2008 Wiley-Liss, Inc.

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TI Transient blockage of the CD11d/CD18 integrin reduces contusion volume

and macrophage infiltration after traumatic brain injury in rats

SO BRAIN RESEARCH

LA English

DT Article

DE traumatic brain injury; microglia/macrophages; CD11d/CD18 integrin;

adhesion molecules; inflammation; histopathology

ID INTERCELLULAR-ADHESION MOLECULE-1; CLOSED-HEAD INJURY;

SPINAL-CORD-INJURY; FLUID-PERCUSSION INJURY; TNF-ALPHA; INFLAMMATORY

RESPONSE; BARRIER DYSFUNCTION; IMPROVED RECOVERY; MICE DEFICIENT;

CELL-DEATH

AB The early inflammatory response to traumatic brain injury (TBI) may result in secondary damage. The purpose of this study was to evaluate the effects of a transient treatment employing a blocking monoclonal antibody (mAb) to the CD11d/CD18 integrin on histopathological outcome and macrophage infiltration following TBI. A parasagittal fluid percussion (FP) brain injury (1.8-2.1 atm) was induced in male Sprague-Dawley rats. Rats were randomized into two trauma groups, treated (N = 7) and nontreated (N = 8) animals. In the treated group, a mAb to the CD11d subunit of the CD11d/CD18 integrin was administered 30 min, 24 and 48 h after brain injury. Control animals received an isotype-matched irrelevant mAb using the same dose and treatment regimen. At 3 days after TBI, animals were perfusion-fixed for histopathological and immunocytochemical analysis. The anti-CD11d mAb treatment reduced contusion areas as well as overall contusion volume compared to vehicle treated animals. For example, overall contusion volume was reduced from 2.7 +/- 0.5 mm(3) (mean +/- SEM) to 1.4 +/- 0.4 with treatment (p < 0.05). Immunocytochemical studies identifying CD68 immunoreactive macrophages showed that treatment caused significant attenuation of leukocyte infiltration into the contused cortical areas. These data emphasize the beneficial effects of blocking inflammatory cell recruitment into the injured brain on histopathological outcome following traumatic brain injury. (C) 2008 Elsevier B.V. All rights reserved.

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TI Effects of <i>in situ</i> administration of excitatory amino acid

antagonists on rapid microglial and astroglial reactions in rat

hippocampus following traumatic brain injury

SO NEUROLOGICAL RESEARCH

LA English

DT Article; Proceedings Paper

CT American-Heart-Association International Stroke Conference 2008

CY FEB 20-22, 2008

CL New Orleans, LA

DE astroglial swelling; excitatory amino acid; microglial reaction;

traumatic brain injury; traumatic depolarization

ID SPREADING DEPRESSION; EXTRACELLULAR POTASSIUM; GLUCOSE-UTILIZATION;

CORTICAL CONTUSION; CEREBRAL-CORTEX; SPINAL-CORD; ASTROCYTES; GLUTAMATE;

RELEASE; CELLS

AB Objective: Both microglia and astrocytes respond immediately to traumatic brain injury (TBI). The present study was undertaken to examine whether or not excitatory amino acid (EAA) antagonists could attenuate such glial responses.

Methods: EAA antagonists, including the broad spectrum EAA antagonist, kynurenic acid (KYN), specific N-methyl-D-aspartate (NMDA) receptor blocker, 2-amino-5-phosphonovalerate (AP-5), and AMPA-KA receptor blocker, 6,7-dinitroquinoxaline-2,3-dione (DNQX), as well as the voltage-dependent ion channel blocker, tetrodotoxin (TTX), were administered into the unilateral hippocampus of rats through a dialysis probe for 30 minutes before the induction of unilateral controlled cortical impact injury. The rats were killed 10 minutes after injury and their brains were processed immunohistochemically for OX42 (marker for microglia) and glial fibrillary acidic protein (GFAP; marker for astrocytes).

Objective: Ten minutes after injury, microglial activation with increased OX42 immunoreactivity was evident in the entire hemisphere including the hippocampus ipsilateral to the injury side. Similarly, swollen astrocytes with increased GFAP expression could be detected exclusively on the injury side. When KYN was administered in situ before injury, both the rapid microglial and astroglial responses in the hippocampus were significantly attenuated. However, AP-5, DNQX and TTX, the voltage-dependent ion channel blocker, at doses which can inhibit each channel activation, failed to attenuate these glial reactions.

Discussion: These findings indicate that massive ionic fluxes and/or concomitantly occurring EAA release may be closely related to the initiation of microglial and astroglial responses following TBI.

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TI Early attenuation of lesional interleukin-16 up-regulation by

dexamethasone and FTY720 in experimental traumatic brain injury

SO NEUROPATHOLOGY AND APPLIED NEUROBIOLOGY

LA English

DT Article

DE dexamethasone; FTY720; interleukin-16; microglia; macrophages; traumatic

brain injury

ID SPINAL-CORD-INJURY; EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS;

HEAD-INJURY; ACTIVATED MICROGLIA/MACROPHAGES; INFLAMMATORY CYTOKINES;

TIRILAZAD MESYLATE; CEREBRAL-ISCHEMIA; MICROGLIAL CELLS; HUMAN

MONOCYTES; NERVOUS-SYSTEM

AB Aims: Interleukin-16 (IL16) is an immunomodulatory cytokine, which induces lymphocyte migration, expression of proinflammatory IL1 beta, IL6 and tumour necrosis factor-alpha, and modulates apoptosis. IL16 expression has been observed in several central nervous system diseases and may play a role in promoting inflammatory responses. Inflammation contributes considerably to secondary injury following traumatic brain injury (TBI). The aim of this study was to investigate early IL16 expression following experimental TBI and the effects of dexamethasone and FTY720 on early expression of IL16 in TBI rats. Methods: Rat TBI was induced using an open-skull weight-drop model. IL16 expression was studied by immunohistochemistry. TBI rats received an intraperitoneal injection of dexamethasone (1 mg/kg in 1 ml saline), FTY720 (1 mg/kg in 1 ml saline) or saline (1 ml) on Day 0 and Day 2 immediately after surgery. Results: Significant up-regulation of IL16 was seen as early as 24 h post TBI. Double-staining experiments, together with morphological classification, revealed a multicellular origin of IL16, including activated microglia/macrophages (about 85%), astrocytes (about 8%), neurones (about 5%) and granulocytes. Following peripheral administration of dexamethasone and FTY720, attenuated numbers of IL16(+) cells were observed on Days 1 and 2 but not on Day 4 post TBI for dexamethasone and on Day 4 but not earlier for FTY720 respectively. Conclusions: Our observations reveal that dexamethasone and FTY720 have different but complementary effects on reduction of early IL16 expression following TBI.

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TI HDAC inhibitor increases histone H3 acetylation and reduces microglia

inflammatory response following traumatic brain injury in rats

SO BRAIN RESEARCH

LA English

DT Article

DE traumatic brain injury; microglia; inflammation; histone deacetylase;

fluid percussion

ID LATERAL FLUID-PERCUSSION; ACUTE NEURONAL DEGENERATION; TRANSGENIC MOUSE

MODEL; DEACETYLASE INHIBITORS; VALPROIC ACID; NEURODEGENERATIVE

DISEASES; BEHAVIORAL DEFICITS; HUNTINGTONS-DISEASE; ALZHEIMERS-DISEASE;

SODIUM-BUTYRATE

AB Traumatic brain injury (TBI) produces a rapid and robust inflammatory response in the brain characterized in part by activation of microglia. A novel histone deacetylase (HDAC) inhibitor, 4-dimethylamino-N-[5-(2-mercaptoacetylamino)pentyl]benzamide (DMA-PB), was administered (0, 0.25, 2.5, 25 mg/kg) systemically immediately after lateral fluid percussion TBI in rats. Hippocampal CA2/3 tissue was processed for acetyl-histone H3 immunolocalization, OX-42 immunolocalization (for microglia), and Fluoro-Jade B histofluorescence (for degenerating neurons) at 24 h after injury. Vehicle-treated TBI rats exhibited a significant reduction in acetyl-histone H3 immunostaining in the ipsilateral CA2/3 hippocampus compared to the sham TBI group (p<0.05). The reduction in acetyl-histone H3 immunostaining was attenuated by each of the DMA-PB dosage treatment groups. Vehicle-treated TBI rats exhibited a high density of phagocytic microglia in the ipsilateral CA2/3 hippocampus compared to sham TBI in which none were observed. All doses of DMA-PB significantly reduced the density of phagocytic microglia (P<0.05). There was a trend for DMA-PB to reduce the number of degenerating neurons in the ipsilateral CA2/3 hippocampus (p = 0.076). We conclude that the HDAC inhibitor DMA-PB is a potential novel therapeutic for inhibiting neuroinflammation associated with TBI. (C) 2008 Elsevier B.V. All rights reserved.

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TI Exacerbated glial response in the aged mouse hippocampus following

controlled cortical impact injury

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE aging; astrocyte; CD11b; GFAP; Iba1; hippocampus; inflammation;

microglia; traumatic brain injury; S100B

ID TRAUMATIC BRAIN-INJURY; FIBRILLARY ACIDIC PROTEIN; GENE-EXPRESSION;

MICROGLIAL ACTIVATION; MESSENGER-RNA; AGING BRAIN; INFLAMMATION;

ASTROCYTES; YOUNG; MICE

AB Old age is associated with enhanced Susceptibility to and poor recovery from brain injury. An exacerbated microglial and astrocyte response to brain injury might be involved in poor outcomes observed in the elderly. The present study was therefore designed to quantitate the expression of markers of microglia and astrocyte activation using real-time RT-PCR, immunoblot and immunohistochemical analysis in aging brain in response to brain injury. We examined the hippocampus, a region that undergoes secondary neuron death, in aged (21-24 months) and adult (5-6 months) mice following controlled cortical impact (CCI) injury to the sensorimotor cortex. Basal mRNA expression of CD11b and Iba1, markers of activated microglia, was higher in aged hippocampus as compared to the adult. The mRNA expression of microglial markers increased and reached maximum 3 days post-injury in both adult and aged mice. but was higher in the aged mice at all time points studied, and in the aged mice the return to baseline levels was delayed. Basal mRNA expression of GFAP and S100B. markers of activated astrocytes, was higher in aged mice. Both markers increased and reached maximum 7 days post-injury. The mRNA expression of astrocyte markers returned to near basal levels rapidly after injury in the adult mice, whereas again in the aged mice return to baseline was delayed. Immunochemical analysis using Iba1 and GFAP antibodies indicated accentuated glial responses in the aged hippocampus after injury. The pronounced and prolonged activation of microglia and astrocytes in hippocampus may contribute to worse cognitive outcomes in the elderly following TBI. (c) 2008 Elsevier Inc. All rights reserved.

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AF Hilton, Genell D.

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TI Roscovitine reduces neuronal loss, glial activation, and neurologic

deficits after brain trauma

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE cell cycle activation; microglia; neurons; roscovitine; TBI

ID PROTEIN-KINASE INHIBITORS; CELL-CYCLE REGULATORS; IN-VITRO; DEATH;

INJURY; EXPRESSION; APOPTOSIS; ISCHEMIA; NEUROPROTECTION; PROLIFERATION

AB Traumatic brain injury (TBI) causes both direct and delayed tissue damage. The latter is associated with secondary biochemical changes such as cell cycle activation, which leads to neuronal death, inflammation, and glial scarring. Flavopiridol-a cyclin-dependent kinase (CDK) inhibitor that is neither specific nor selective-is neuroprotective. To examine the role of more specific CDK inhibitors as potential neuroprotective agents, we studied the effects of roscovitine in TBI. Central administration of roscovitine 30 mins after injury resulted in significantly decreased lesion volume, as well as improved motor and cognitive recovery. Roscovitine attenuated neuronal death and inhibited activation of cell cycle pathways in neurons after TBI, as indicated by attenuated cyclin G1 accumulation and phosphorylation of retinoblastoma protein. Treatment also decreased microglial activation after TBI, as reflected by reductions in ED1, galectin-3, p22(PHOX), and Iba-1 levels, and attenuated astrogliosis, as shown by decreased accumulation of glial fibrillary acidic protein. In primary cortical microglia and neuronal cultures, roscovitine and other selective CDK inhibitors attenuated neuronal cell death, as well as decreasing microglial activation and microglial-dependent neurotoxicity. These data support a multifactorial neuroprotective effect of cell cycle inhibition after TBI-likely related to inhibition of neuronal apoptosis, microglial-induced inflammation, and gliosis-and suggest that multiple CDKs are potentially involved in this process.

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AU Wasserman, JK

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TI White matter injury in young and aged rats after intracerebral

hemorrhage

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Stroke; Traumatic brain injury; Axonal injury; Demyelination; Microglial

activation; Amyloid precursor protein

ID AMYLOID PRECURSOR PROTEIN; MIDDLE CEREBRAL-ARTERY; DELAYED MINOCYCLINE

TREATMENT; TRAUMATIC BRAIN-INJURY; AXONAL-TRANSPORT; DAMAGED AXONS;

NEURON DEATH; BETA-APP; STROKE; EDEMA

AB Experimental studies of intracerebral hemorrhage (ICH) have focused on neuron death, with little or no information on axonal and myelin damage outside the hematoma. Because development of effective therapies will require an understanding of white matter injury, we examined white matter injury and its spatial and temporal relationship with microglial/macrophage activation in a collagenase model of rat striatal ICH. The hematoma and parenchyma surrounding the hematoma were assessed in young and aged animals at 6 h, 1, 3 and 28 days after ICH onset. Demyelination occurred inside and at the edge of the hematoma; regions where we have shown substantial neuron death. In contrast, there was axonal damage without demyelination at the edge of the hematoma, and by 3 days this damage had spread to the surrounding parenchyma, a region where we have shown there is no neuron death. Because the axonal damage preceded infiltration of activated microglia into the white matter tracts (seen at 3 days), our results support the hypothesis that these cells respond to, rather than perpetrate the damage. Importantly, axonal damage was worse in aged animals, which provides a plausible explanation for the poorer functional recovery of older animals after ICH, despite a similar loss of grey matter. Our findings support strategies that target white matter injury to reduce neurological impairment after ICH. (C) 2008 Elsevier Inc. All rights reserved.

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TI Microglial involvement in neuroprotection following experimental

traumatic brain injury in heat-acclimated mice

SO BRAIN RESEARCH

LA English

DT Article

DE Cytokines; Glia; Neuroprotection; Neurotrophin; Preconditioning

ID EXPRESSION; ERYTHROPOIETIN; DISEASE; MODEL; RAT

AB Brain-derived neurotrophic factor (BDNF) conveys neuroprotection in various settings of experimental central nervous system injury. Using a model of endogenous neuroprotection, induced in mice by chronic exposure to moderate ambient heat (heat acclimation, HA), we have previously shown that neuroprotection following traumatic brain injury involves reduced post-injury tumor necrosis factor alpha (TNF alpha) expression. As glial cells play a pivotal role in post-injury inflammation on one hand, and are also capable of inducing neuroprotection by harboring trophic factors and BDNF in particular, the effects of injury and HA on overall BDNF content at the trauma area, gliosis and glial BDNF expression were investigated. Western blotting indicated higher overall BDNF levels in HA sham-operated mice. Following injury, a decrease was observed in the HA group only, reaching levels similar to normothermic mice. Immunohistochemical studies demonstrated BDNF-positive resting microglia in non-injured HA but not normothermic animals. Post-injury astrocytosis and microglial immunoreactivity were enhanced in the HA group. Particularly, an increase in the amount of ramified microglia was observed within the penumbra, accompanied by a concomitant decrease in globular microglia, a major source of pro-inflammatory mediators. BDNF labeling on and around microglia and their processes was intensified in HA mice. Furthermore, BDNF immunoreactivity in HA mice was evident in the degenerated edges of axons. These findings, taken together with the growing body of evidence indicating the neuroprotective potential of both BDNF and microglia, suggest a possible role of these cells in HA-induced neuroprotection. (C) 2008 Elsevier B.V. All rights reserved.

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PT J

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TI Traumatic Brain Injury May Increase the Risk for Frontotemporal Dementia

through Reduced Progranulin

SO NEURODEGENERATIVE DISEASES

LA English

DT Review

DE Frontotemporal dementia; Progranulin; Traumatic brain injury; Microglia;

Elastase

ID LOBAR DEGENERATION; RAT

AB Frontotemporal lobar degeneration with TAR-DNA-binding protein inclusions (FTLD-TDP) is the most common pathological subtype of frontotemporal dementia (FTD). Mutations leading to a loss of function in the progranulin gene (PGRN) are the most common known cause of FTLD-TDP. In agreement with the proposed loss of function disease mechanism, several groups have reported decreased plasma levels of PGRN in patients carrying PGRN mutations compared to individuals without PGRN mutations. We propose that traumatic brain injury (TBI), an environmental factor, may also increase the risk of FTD by altering PGRN metabolism. TBI may lead to an increase in the central nervous system levels of microglial elastases, which proteolyze PGRN into proinflammatory products called granulins causing a reduction in PGRN levels. Hence, inhibiting microglial activation may have an important implication for the prevention of FTD in patients with TBI. Copyright (C) 2010 S. Karger AG, Basel

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TI Deleterious Effects of Minocycline After In Vivo Target Deprivation of

Thalamocortical Neurons in the Immature, Metallothionein-deficient Mouse

Brain

SO JOURNAL OF NEUROSCIENCE RESEARCH

LA English

DT Article

DE microglia; minocycline; metallothionein; traumatic brain injury

ID FOCAL CEREBRAL-ISCHEMIA; SPINAL-CORD-INJURY; CEREBELLAR GRANULE NEURONS;

CYTOCHROME-C RELEASE; TISSUE GROWTH-FACTOR; TRAUMATIC BRAIN; CELL-DEATH;

HUNTINGTONS-DISEASE; NEUROLOGICAL DYSFUNCTION; MICROGLIAL ACTIVATION

AB Compared with adults, immature metallothionein I and II knockout (MT-/-) mice incur greater neuronal loss and a more rapid rate of microglia accumulation after target deprivation-induced injury. Because minocycline has been proposed to inhibit microglial activation and associated production of neuroinflammatory factors, we investigated its ability to promote neuronal survival in the immature, metallothionein-deficient brain. After ablation of the visual cortex, 10-day-old MT-/- mice were treated with minocycline or saline and killed 24 or 48 hr after injury. By means of stereological methods, the number of microglia and neurons were estimated in the ipsilateral dorsal lateral geniculate nucleus (dLGN) by an investigator blinded to the treatment. No effect on neuronal survival was observed at 24 hr, but 48 hr after injury, an unanticipated but significant minocycline-mediated increase in neuronal loss was detected. Further, while failing to inhibit microglial accumulation, minocycline treatment increased the proportion of amoeboid microglia in the ipsilateral dLGN. To understand the molecular mechanisms underlying this neurotoxic response, we identified minocycline-mediated changes in the expression of three potentially proapoptotic/inflammatory genes: growth arrest- and DNA damage-inducible gene 45 gamma (GADD45 gamma); interferon-inducible protein 1 (IFI1), and cytokine-induced growth factor. We also observed increased mitogen-activated protein kinase p38 phosphorylation with minocycline treatment. Although minocycline inhibited calpain activity at 12 hr after injury, this effect was not sustained at 24 hr. Together, these results help to explain how minocycline has a deleterious effect on neuronal survival in this injury model. (C) 2008 Wiley-Liss, Inc.

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TI Metallothionein Treatment Attenuates Microglial Activation and

Expression of Neurotoxic Quinolinic Acid Following Traumatic Brain

Injury

SO NEUROTOXICITY RESEARCH

LA English

DT Article

DE Traumatic brain injury; Neuroinflammation; Neuron-glia interactions

ID KYNURENINE PATHWAY; DISEASE; CNS; METABOLISM; CELLS; BETA; PROTECT;

FAMILY; ZINC

AB The kynurenine pathway has been implicated as a major component of the neuroinflammatory response to brain injury and neurodegeneration. We found that the neurotoxic kynurenine pathway intermediate quinolinic acid (QUIN) is rapidly expressed, within 24 h, by reactive microglia following traumatic injury to the rodent neocortex. Furthermore, administration of the astrocytic protein metallothionein attenuated this neuroinflammatory response by reducing microglial activation (by approximately 30%) and QUIN expression. The suppressive effect of MT was confirmed upon cultured cortical microglia, with 1 mu g/ml MT almost completely blocking interferon-gamma induced activation of microglia and QUIN expression. These results demonstrate the neuroimmunomodulatory properties of MT, which may have therapeutic applications for the treatment of traumatic brain injury.

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TI SIMVASTATIN ATTENUATES MICROGLIAL CELLS AND ASTROCYTE ACTIVATION AND

DECREASES INTERLEUKIN-1B LEVEL AFTER TRAUMATIC BRAIN INJURY

SO NEUROSURGERY

LA English

DT Article

DE Astrocyte; Interleukin-1 beta; Microglia; Simvastatin; Traumatic brain

injury

ID NITRIC-OXIDE SYNTHASE; CLOSED-HEAD INJURY; INFLAMMATORY RESPONSE; CNS

INJURY; KAPPA-B; STATINS; INDUCTION; EXPRESSION; RECEPTOR;

SYNAPTOGENESIS

AB OBJECTIVE: Our previous studies demonstrated that simvastatin promotes neurological functional recovery after traumatic brain injury (TBI) in rat; however, the underlying mechanisms remain poorly understood. The purpose of this study was to investigate the anti-inflammatory effect of simvastatin by measuring the level of cytokines and activation of glial cells.

METHODS: Controlled cortical impact injury was performed in adult male Wistar rats. The rats were randomly divided into 3 groups: sham, saline control group, and simvastatin treatment group. Simvastatin was administered orally starting at day 1 after TBI until animals were killed at days 1, 3, 7, 14, and 35 after treatment. Functional outcome was measured using modified neurological severity scores. Enzyme-linked immunosorbent assay and immunohistochemical staining were used to measure the expression of interleukin (IL)-1 beta, IL-6, and tumor necrosis factor-a and to identify activated microglial cells and astrocytes.

RESULTS: At days 1 and 3 after simvastatin or saline treatment, cytokine levels in the lesion boundary zone were significantly higher in the simvastatin- and saline-treated rats compared with the sham group, peaking at day 3. Simvastatin only reduced the level of IL-1 beta but not IL-6 and tumor necrosis factor-alpha, compared with the saline group. Also, simvastatin significantly reduced the number of activated microglial cells and astrocytes compared with the saline control animals. There was also a trend toward improvement of modified neurological severity score, reaching statistical significance (P = 0.003) toward the end of the trial.

CONCLUSION: Our data demonstrate that TBI causes inflammatory reaction, including increased levels of IL-1 beta, IL-6, and tumor necrosis factor-alpha, as well as activated microglial cells. Simvastatin selectively reduces IL-1 beta expression and inhibits the activation of microglial cells and astrocytes after TBI, which might be one of the mechanisms underlying the therapeutic benefits of simvastatin treatment of TBI.

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TI Neutralization of interleukin-1β modifies the inflammatory response and

improves histological and cognitive outcome following traumatic brain

injury in mice

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DE behaviour; cognition; microglia; neutrophils; T-cells

ID CLOSED-HEAD INJURY; CONTROLLED CORTICAL IMPACT; FLUID PERCUSSION INJURY;

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UP-REGULATION; PROINFLAMMATORY CYTOKINE; MYELOPEROXIDASE ACTIVITY;

MONONUCLEAR PHAGOCYTES

AB Interleukin-1 beta (IL-1 beta) may play a central role in the inflammatory response following traumatic brain injury (TBI). We subjected 91 mice to controlled cortical impact (CCI) brain injury or sham injury. Beginning 5 min post-injury, the IL-1 beta neutralizing antibody IgG2a/k (1.5 mu g/mL) or control antibody was infused at a rate of 0.25 mu L/h into the contralateral ventricle for up to 14 days using osmotic minipumps. Neutrophil and T-cell infiltration and microglial activation was evaluated at days 1-7 post-injury. Cognition was assessed using Morris water maze, and motor function using rotarod and cylinder tests. Lesion volume and hemispheric tissue loss were evaluated at 18 days post-injury. Using this treatment strategy, cortical and hippocampal tissue levels of IgG2a/k reached 50 ng/mL, sufficient to effectively inhibit IL-1 beta in vitro. IL-1 beta neutralization attenuated the CCI-induced cortical and hippocampal microglial activation (P < 0.05 at post-injury days 3 and 7), and cortical infiltration of neutrophils (P < 0.05 at post-injury day 7). There was only a minimal cortical infiltration of activated T-cells, attenuated by IL-1 beta neutralization (P < 0.05 at post-injury day 7). CCI induced a significant deficit in neurological motor and cognitive function, and caused a loss of hemispheric tissue (P < 0.05). In brain-injured animals, IL-1 beta neutralizing treatment resulted in reduced lesion volume, hemispheric tissue loss and attenuated cognitive deficits (P < 0.05) without influencing neurological motor function. Our results indicate that IL-1 beta is a central component in the post-injury inflammatory response that, in view of the observed positive neuroprotective and cognitive effects, may be a suitable pharmacological target for the treatment of TBI.

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TI CD38 Facilitates Recovery from Traumatic Brain Injury

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DE CD38; head trauma; microglia

ID CLOSED-HEAD-INJURY; CYCLIC ADP-RIBOSE; NEURONAL CELL-DEATH; OBJECT

RECOGNITION; IN-VIVO; T-CELL; REACTIVE ASTROCYTES; SIGNAL-TRANSDUCTION;

COGNITIVE DEFICITS; RESIDENT MICROGLIA

AB Traumatic brain injury (TBI) is a major cause of death and disability worldwide. It causes progressive tissue atrophy and consequent neurological dysfunctions. TBI is accompanied by neuroinflammation, a process mediated largely by microglia. CD38 is an ectoenzyme that promotes transmembrane signaling via the synthesis of potent calcium mobilizing agents or via its receptor activity. CD38 is expressed in the brain in various cell types including microglia. In previous studies, we showed that CD38 regulates microglial activation and response to chemokines. In view of the important role of neuroinflammation in TBI and the effects of CD38 on microglial responses, the present study examines the role of CD38 in the recovery of mice from closed head injury (CHI), a model of focal TBI. For this purpose, CD38-deficient and wild-type (WT) mice were subjected to a similar severity of CHI and the effect of the injury on neurobehavioral and cognitive functions was assessed by the Neurological Severity Score (NSS) and the Object Recognition Test, at various time points post-injury. The results show that recovery after CHI (as indicated by the NSS) was significantly lower in CD38-deficient mice than in WT mice and that the object recognition performance after injury was significantly impaired in injured CD38-deficient mice than in WT mice. In addition, we also observed that the amount of activated microglia/macrophages at the injury site was significantly lower in CD38-deficient mice compared with WT mice. Taken together, our findings indicate that CD38 plays a beneficial role in the recovery of mice from CHI and that this effect is mediated, at least in part, via the effect of CD38 on microglia responses.

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TI Brain trauma induces expression of diacylglycerol kinase ζ in microglia

SO NEUROSCIENCE LETTERS

LA English

DT Article

DE Diacylglycerol kinase; Brain cryoinjury; Microglia; Iba1; GLUT5

ID CENTRAL-NERVOUS-SYSTEM; RAT-BRAIN; GLIAL SCAR; INJURY; CELL; ASTROCYTES;

CLONING; RECEPTORS; FAMILY; GENE

AB Diacylglycerol kinase (DGK) is an enzyme which phosphorylates a second messenger diacylglycerol and consists of a family of isozymes that differ in terms of structural motifs, enzymological property, and cell and tissue distribution. One of the isozymes, DGK zeta was originally shown to be expressed in various kinds of neurons under physiological conditions. However, we unexpectedly found that under pathological conditions, such as cerebral infarction, DGK zeta-immunoreactivity is detected in non-neuronal cells, although it remained to be elucidated in detail which cell types are responsible for the induced expression of DGK zeta in this setting. To further elucidate functional implications of DGK zeta in non-neuronal cells we performed detailed immunohistochemical analysis of DGK zeta using rat brain cryoinjury model. As early as I h after cryoinjury, DGK zeta-immunoreactivity was greatly decreased in the afflicted cerebral cortex and almost disappeared in the necrotic core. On day 7 after cryoinjury, however, DGK zeta-immunoreactivity reappeared in this area. DGK zeta-immunoreactivity was clearly detected in Iba1-immunoreactive cells of an oval or ameboid shape in the scar region, which represent activated microglia and/or macrophages. on the other hand, DGK zeta-immunoreactivity was not detected in Iba1-immunoreactive, resting microglia of ramified and dendritic configuration in the intact cortex. Furthermore, DGK zeta-immunoreactive cells were also positive for a microglia marker GLUT5 in the scar region, but never for an astrocyte marker GFAP. Taken together, the present study reveals that DGK zeta is induced in activated microglia in brain trauma, suggesting the functional significance of DGK zeta in this process. (C) 2009 Elsevier Ireland Ltd. All rights reserved.

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Huang, Yi

TI NMDA receptor antagonist MK-801 reduces neuronal damage and preserves

learning and memory in a rat model of traumatic brain injury

SO NEUROSCIENCE BULLETIN

LA English

DT Article

DE traumatic brain injury; MK-801; learning and memory; caspase-3;

microglia; neuronal nitric oxide synthase

AB Objective NMDA receptor channel plays an important role in the pathophysiological process of traumatic brain injury (TBI). The present study aims to study the pathological mechanism of TBI and the impairment of learning and memory after TBI, and to investigate the mechanism of the protective effect of NMDA receptor antagonist MK-801 on learning and memory disorder after TBI. Methods Forty Sprague-Dawley rats (weighing approximately 200 g) were randomized into 5 groups (n = 8 in each group): control group, model group, low-dose group (MK-801 0.5 mg/kg), middle-dose group (MK-801 2 mg/kg), and high-dose group (MK-801 10 mg/kg). TBI model was established using a weight-drop head injury mode. After 2-month drug treatment, learning and memory ability was evaluated by using Morris water maze test. Then the animals were sacrificed, and brain tissues were taken out for morphological and immunohistochemical assays. Results The ability of learning and memory was significantly impaired in the TBI model animals. Besides, the neuronal caspase-3 expression, neuronal nitric oxide synthase (nNOS)-positive neurons and OX-42-positive microglia were all increased in TBI animals. Meanwhile, the number of neuron synapses was decreased, and vacuoles degeneration could be observed in mitochondria. After MK-801 treatment at 3 different dosages, the ability of learning and memory was markedly improved, as compared to that of the TBI model animals. Moreover, neuronal caspase-3 expression, OX-42-positive microglia and nNOS-positive neurons were all significantly decreased. Meanwhile, the mitochondria degeneration was greatly inhibited. Conclusion MK-801 could significantly inhibit the degeneration and apoptosis of neurons in damaged brain areas. It could also inhibit TBI-induced increase in nNOS-positive neurons and OX-42-positive microglia. Impairment in learning and memory in TBI animals could be repaired by treatment with MK-801.

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TI MECHANISM OF THE ANTI-INFLAMMATORY EFFECT OF 17β-ESTRADIOL ON BRAIN

FOLLOWING TRAUMA-HEMORRHAGE

SO SHOCK

LA English

DT Article

DE CNS; TNF-alpha; female sex hormone; microglia; minocycline

ID CELL CYTOKINE PRODUCTION; ESTROGEN-RECEPTOR-ALPHA; INFLAMMATORY

RESPONSE; MICROGLIAL ACTIVATION; NERVOUS-SYSTEM; EXPRESSION; INJURY;

PATHWAY; RAT; NOREPINEPHRINE

AB Although 17 beta-estradiol (E2) is reported to improve the inflammatory response after trauma-hemorrhage (T-H), it remains unknown whether E2 plays any role in the central nervous system after T-H. Microglial cells, resident central macrophages, are thought to play a central role in exacerbating cell-mediated inflammation. We hypothesized that T-H up-regulates microglial cell-mediated inflammatory response in the brain, and E2 produces central anti-inflammatory effects via negative regulation of microglial cells. Male Sprague-Dawley rats were subjected to sham operation (cannulation plus laparotomy) or T-H (midline laparotomy; mean blood pressure, 35 +/- 5 mmHg for 90 min followed by resuscitation) and immediately killed after resuscitation. Rats received vehicle or E2 (1 mg/kg body weight i.v.) at the onset of resuscitation. In other experiments, minocycline (40 mg/kg body weight i.p.), microglia inhibitor, was administered 1 h before T-H to prevent inflammatory response in the microglia after T-H. The plasma and hypothalamic tumor necrosis factor (TNF-alpha) levels were increased, along with the activation of microglial cells in T-H rats compared with shams. Furthermore, T-H increased microglial TNF-alpha productive capacity in vitro. 17 beta administration after T-H prevented these inflammatory responses. In rats pretreated with minocycline, decreased microglial TNF-alpha. production and hypothalamic TNF-alpha levels were observed, but plasma TNF-alpha levels were not altered after T-H. Thus, T-H induces inflammatory responses even in the hypothalamus, and E2 seems to be a useful adjunct for down-regulating microglial cell-mediated inflammatory response after T-H.

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JI Shock

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TI CC-CHEMOKINE LIGAND 18/PULMONARY ACTIVATION-REGULATED CHEMOKINE

EXPRESSION IN THE CNS WITH SPECIAL REFERENCE TO TRAUMATIC BRAIN INJURIES

AND NEOPLASTIC DISORDERS

SO NEUROSCIENCE

LA English

DT Article

DE CC-chemokine; glia; microglia; macrophages; glioma; source; lesion

ID CENTRAL-NERVOUS-SYSTEM; MONOCYTE CHEMOATTRACTANT PROTEIN-1;

MESSENGER-RNA EXPRESSION; HUMAN-MALIGNANT GLIOMA; RECEPTOR EXPRESSION;

CEREBROSPINAL-FLUID; HUMAN ASTROCYTOMAS; CELL INFILTRATION; DENDRITIC

CELLS; MACROPHAGES

AB Pulmonary activation-regulated chemokine (PARC) now designated CC-chemokine ligand 18 (CCL18) has been shown to play a significant role in the pathogenesis of various tissue injuries and diseases in a proinflammatory or immune suppressive way to limit or support the inflammation or disease. While much is known about the roles of CCL18/PARC in non-neural tissues, its expression in the CNS has remained largely unexplored and controversial. Using reverse transcription polymerase chain reaction (RT-PCR) and double immunohistochemical staining, we analyzed the expression of CCL18/PARC in the human brain with special reference to traumatic brain injuries and tumors. The RT-PCR analysis revealed the expression of CCL18/PARC mRNA both in the traumatic brain and glioma tissues examined. Immunoexpression of CCL18/PARC protein was consistently detected in all cases of traumatic brain injuries examined by immunohistochemical staining. Double immunofluorescence labeling has extended the study that CCL18/PARC positive cells were macrophages/microglia, astrocytes or neurons. The CCL18/PARC expression was localized in macrophage-like cells in two of eight glioblastoma tissues whose cancer cells were CCL18/PARC negative. Unexpectedly, CCL18/PARC mRNA weakly and constitutively expressed by glioblastoma cell line was upregulated after endotoxin stimulation. The present results indicated a significant production of CCL18/PARC in different CNS traumatic and neoplasm tissues by specific cellular elements expressing the chemokine. An anti-inflammatory mechanism jointly exerted by these cells via CCL18/PARC may be involved in the CNS immunity after traumatic injury and tumorigenesis. (C) 2010 IBRO. Published by Elsevier Ltd. All rights reserved.

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TI Long-Term Gliosis and Molecular Changes in the Cervical Spinal Cord of

the Rhesus Monkey after Traumatic Brain Injury

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE injury; microglia; motor recovery; spinal cord; synapse

ID GLUTAMATE TRANSPORTER GLT-1; PROTEIN-KINASE PATHWAYS; CORTICAL IMPACT

INJURY; NOGO RECEPTOR; ARM REPRESENTATION; CEREBRAL-ISCHEMIA;

IMMUNE-RESPONSE; CELL-DEATH; EXPRESSION; RECOVERY

AB Recovery of fine motor skills after traumatic brain injury (TBI) is variable, with some patients showing progressive improvements over time while others show poor recovery. We therefore studied possible cellular mechanisms accompanying the recovery process in a non-human primate model system, in which the lateral frontal motor cortex areas controlling the preferred upper limb were unilaterally lesioned, and the animals eventually regained fine hand motor function. Immunohistochemical staining of the cervical spinal cord, the site of compensatory sprouting and degeneration of corticospinal axons, showed profound increases in immunoreactivities for major histocompatibility complex class II molecule (MHC-II) and extracellular signal-regulated kinases (ERK1/2) up to 12 months post lesion, particularly within the lateral corticospinal tract (LCST). Double immunostaining demonstrated that phosphorylated ERK1/2 colocalized within the MCH-II+ microglia, suggesting a trophic role of long-term microglia activation after TBI at the site of compensatory sprouting. Active sprouting was observed in the LCST as well as in the spinal gray matter of the lesioned animals, as illustrated by increases in growth associated protein 43. Upregulation of Nogo receptor and glutamate transporter expression was also observed in this region after TBI, suggesting possible mechanisms for controlling aberrant sprouting and/or synaptic formation en route and interstitial glutamate concentration changes at the site of axon degeneration, respectively. Taken together, these changes in the non-human primate spinal cord support a long-term trophic/tropic role for reactive microglia, in particular, during functional and structural recovery after TBI.

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TI Adenosine A<sub>1</sub> Receptor Activation as a Brake on the Microglial

Response after Experimental Traumatic Brain Injury in Mice

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE adenosine; A(1) receptor; BV-2 cells; head injury; Iba-1; knockout;

microglia; neurotrauma

ID CONTROLLED CORTICAL IMPACT; STATUS EPILEPTICUS; CEREBRAL-ISCHEMIA;

MOUSE-BRAIN; CELLS; NEURODEGENERATION; ANTAGONISTS; SEIZURES; LACKING;

RATS

AB We reported that adenosine A(1) receptor (A(1)AR) knockout (KO) mice develop lethal status epilepticus after experimental traumatic brain injury (TBI), which is not seen in wild-type (WT) mice. Studies in epilepsy, multiple sclerosis, and neuro-oncology suggest enhanced neuro-inflammation and/or neuronal death in A(1)AR KO. We hypothesized that A(1)AR deficiency exacerbates the microglial response and neuronal damage after TBI. A1AR KO and WT littermates were subjected to mild controlled cortical impact (3m/sec; 0.5mm depth) to left parietal cortex, an injury level below the acute seizure threshold in the KO. At 24 h or 7 days, mice were sacrificed and serial sections prepared. Iba-1 immunostaining was used to quantify microglia at 7 days. To assess neuronal injury, sections were stained with Fluoro-Jade C (FJC) at 24 h to evaluate neuronal death in the hippocampus and cresyl violet staining at 7 days to analyze cortical lesion volumes. We also studied the effects of adenosine receptor agonists and antagonists on H-3-thymidine uptake (proliferation index) by BV-2 cells (immortalized mouse microglial). There was no neuronal death in CA1 or CA3 quantified by FJC. A(1)AR KO mice exhibited enhanced microglial response; specifically, Iba-1 + microglia were increased 20-50% more in A1AR KO versus WT in ipsilateral cortex, CA3, and thalamus, and contralateral cortex, CA1, and thalamus (p<0.05). However, contusion and cortical volumes did not differ between KO and WT. Pharmacological studies in cultured BV-2 cells indicated that A(1)AR activation inhibits microglial proliferation. A(1)AR activation is an endogenous inhibitor of the microglial response to TBI, likely via inhibition of proliferation, and this may represent a therapeutic avenue to modulate microglia after TBI.

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TI Blockade of Acute Microglial Activation by Minocycline Promotes

Neuroprotection and Reduces Locomotor Hyperactivity after Closed Head

Injury in Mice: A Twelve-Week Follow-Up Study

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE diffuse axonal injury; locomotor hyperactivity; microglial activation;

minocycline; traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; AMYLOID PRECURSOR PROTEIN;

AMYOTROPHIC-LATERAL-SCLEROSIS; FLUID PERCUSSION INJURY; EXCITATORY

AMINO-ACIDS; AXONAL INJURY; CEREBRAL EDEMA; MODEL; NEUROTOXICITY;

EXPRESSION

AB Traumatic brain injury (TBI) causes a wide spectrum of consequences, such as microglial activation, cerebral inflammation, and focal and diffuse brain injury, as well as functional impairment. In this study we aimed to investigate the effects of acute treatment with minocycline as an inhibitor of microglial activation on cerebral focal and diffuse lesions, and on the spontaneous locomotor activity following TBI. The weight-drop model was used to induce TBI in mice. Microglial activation and diffuse axonal injury (DAI) were detected by immunohistochemistry using CD11b and beta-amyloid precursor protein (beta-APP) immunolabeling, respectively. Focal injury was determined by the measurement of the brain lesion volume. Horizontal and vertical locomotor activities were measured for up to 12 weeks post-injury by an automated actimeter. Minocycline or vehicle were administered three times post-insult, at 5 min (90mg/kg i.p.), 3 h, and 9 h post-TBI (45 mg/kg i.p.). Minocycline treatment attenuated microglial activation by 59% and reduced brain lesion volume by 58%, yet it did not affect DAI at 24 h post-TBI. More interestingly, minocycline significantly decreased TBI-induced locomotor hyperactivity at 48 h post-TBI, and its effect lasted for up to 8 weeks. Taken together, the results indicate that microglial activation appears to play an important role in the development of TBI-induced focal injury and the subsequent locomotor hyperactivity, and its short-term inhibition provides long-lasting functional recovery after TBI. These findings emphasize the fact that minocycline could be a promising new therapeutic strategy for head-injured patients.

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TI Minozac Treatment Prevents Increased Seizure Susceptibility in a Mouse

"Two-Hit'' Model of Closed Skull Traumatic Brain Injury and

Electroconvulsive Shock-Induced Seizures

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE astrocyte; electroconvulsive shock; microglia; seizure; traumatic brain

injury

ID INDUCED STATUS EPILEPTICUS; FLUID PERCUSSION INJURY; PROINFLAMMATORY

CYTOKINE; POSTTRAUMATIC EPILEPSY; HEAD INJURY; FUNCTIONAL RECOVERY;

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AB The mechanisms linking traumatic brain injury (TBI) to post-traumatic epilepsy (PTE) are not known and no therapy for prevention of PTE is available. We used a mouse closed-skull midline impact model to test the hypotheses that TBI increases susceptibility to seizures in a "two-hit'' injury model, and that suppression of cytokine upregulation after the first hit will attenuate the increased susceptibility to the second neurological insult. Adult male CD-1 mice underwent midline closed skull pneumatic impact. At 3 and 6 h after impact or sham procedure, the mice were injected IP with either Minozac (Mzc), a suppressor of proinflammatory cytokine upregulation, or vehicle (saline). On day 7 after sham operation or TBI, seizures were induced using electroconvulsive shock (ECS), and susceptibility to seizures was measured by the current required for seizure induction. Activation of glia, neuronal injury, and metallothionein-immunoreactive cells were quantified in the hippocampus by immunohistochemical methods. Neurobehavioral function over 14-day recovery was quantified using the Barnes maze. Following TBI there was a significant increase in susceptibility to seizures induced by ECS, and this susceptibility was prevented by suppression of cytokine upregulation with Mzc. Astrocyte activation, metallothionein expression, and neurobehavioral impairment were also increased in the two-hit group subjected to combined TBI and ECS. These enhanced responses in the two-hit group were also prevented by suppression of proinflammatory cytokine upregulation with Mzc. These data implicate glial activation in the mechanisms of epileptogenesis after TBI, and identify a potential therapeutic approach to attenuate the delayed neurological sequelae of TBI.

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TI Gp91<SUP>phox</SUP> (NOX2) in classically activated microglia

exacerbates traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

ID NADPH-OXIDASE; OXIDATIVE STRESS; LIPID-PEROXIDATION; ALZHEIMERS-DISEASE;

CELLS; DAMAGE; NEURONS; SYSTEM; MODEL; MICE

AB Background: We hypothesized that gp91(phox) (NOX2), a subunit of NADPH oxidase, generates superoxide anion (O-2(-)) and has a major causative role in traumatic brain injury (TBI). To evaluate the functional role of gp91(phox) and reactive oxygen species (ROS) on TBI, we carried out controlled cortical impact in gp91(phox) knockout mice (gp91(phox-/-)). We also used a microglial cell line to determine the activated cell phenotype that contributes to gp91(phox) generation.

Methods: Unilateral TBI was induced in gp91(phox-/-) and wild-type (Wt) mice (C57/B6J) (25-30 g). The expression and roles of gp91(phox) after TBI were investigated using immunoblotting and staining techniques. Levels of O2-and peroxynitrite were determined in situ in the mouse brain. The activated phenotype in microglia that expressed gp91(phox) was determined in a microglial cell line, BV-2, in the presence of IFN gamma or IL-4.

Results: Gp91(phox) expression increased mainly in amoeboid-shaped microglial cells of the ipsilateral hemisphere of Wt mice after TBI. The contusion area, number of TUNEL-positive cells, and amount of O-2(-) and peroxynitrite metabolites produced were less in gp91(phox-/-) mice than in Wt. In the presence of IFN gamma, BV-2 cells had increased inducible nitric oxide synthase and nitric oxide levels, consistent with a classical activated phenotype, and drastically increased expression of gp91(phox).

Conclusions: Classical activated microglia promote ROS formation through gp91(phox) and have an important role in brain damage following TBI. Modulating gp91(phox) and gp91(phox)-derived ROS may provide a new therapeutic strategy in combating post-traumatic brain injury.

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TI Genetic regulation of microglia activation, complement expression, and

neurodegeneration in a rat model of traumatic brain injury

SO EXPERIMENTAL BRAIN RESEARCH

LA English

DT Article

DE Traumatic brain injury; Inflammation; Complement; Neurodegeneration;

Strain dependent; T cells; Polymorphonuclear cells

ID COLONY-STIMULATING FACTORS; TUMOR-NECROSIS-FACTOR; CLOSED-HEAD INJURY;

SPINAL-CORD INJURY; MONOCLONAL-ANTIBODY; NERVOUS-SYSTEM; INFLAMMATORY

RESPONSE; MOUSE STRAINS; AMINO-ACIDS; CELL-DEATH

AB Secondary brain damage following traumatic brain injury in part depends on neuroinflammation, a process where genetic factors may play an important role. We examined the response to a standardized cortical contusion in two different inbred rat strains, Dark Agouti (DA) and Piebald Virol Glaxo (PVG). Both are well characterized in models of autoimmune neuroinflammation, where DA is susceptible and PVG resistant. We found that infiltration of polymorphonuclear granulocytes (PMN) at 3-day postinjury was more pronounced in PVG. DA was more infiltrated by T cells at 3-day postinjury, showed an enhanced glial activation at 7-day postinjury and higher expression of C3 complement at 7-day postinjury. Neurodegeneration, assessed by Fluoro-Jade, was also more pronounced in the DA strain at 30-day postinjury. These results demonstrate differences in the response to cortical contusion injury attributable to genetic influences and suggest a link between injury-induced inflammation and neurodegeneration. Genetic factors that regulate inflammation elicited by brain trauma may be important for the development of secondary brain damage.

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TI Beneficial effects of sodium or ethyl pyruvate after traumatic brain

injury in the rat

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Cell Death; Cytochrome oxidase; Ethyl pyruvate; Functional recovery;

Inflammation; Microglia; Neuroprotection; Rats; Sodium pyruvate;

Traumatic brain injury

ID CYTOCHROME-C-OXIDASE; CORTICAL CONTUSION INJURY; WIDE THERAPEUTIC

WINDOW; FOCAL CEREBRAL-ISCHEMIA; HEMORRHAGIC-SHOCK; HUNTINGTONS-DISEASE;

HEAD-INJURY; ANTIINFLAMMATORY MECHANISM; ENDOTHELIAL-CELLS; OXIDATIVE

DAMAGE

AB Sodium pyruvate (SP) treatment initiated within 5 min post-injury is neuroprotective in a rat model of unilateral cortical contusion injury (CCI). The current studies examined: (1) effects of delayed SP treatments (1000 mg/kg, i.p., at 1, 12 and 24 h), (2) effects of single (1 h) or multiple (1, 12 and 24 h) ethyl pyruvate treatments (EP; at 20 or 40 mg/kg, i.p.), and (3) mechanisms of action for pyruvate effects after CCI. In Experiment 1, both SP and EP treatment(s) significantly reduced the number of dead/dying cells in the ipsilateral hippocampus (dentate hilus + CA3(c) and/or CA3(a-b) regions) at 72 h post-CCI. Pyruvate treatment (s) attenuated CCI-induced reductions of cerebral cytochrome oxidase activity at 72 h, significantly improving activity in peri-contusional cortex after multiple SP or EP treatments. Optical density measures of ipsilateral CD11b immuno-staining were significantly increased 72 h post-CCI, but these measures of microglia activation were not different from sham injury values in SP and EP groups with three post-CCI treatments. In Experiment 2, three treatments (1, 12 and 24 h) of SP (1000 mg/kg) or EP (40 mg/kg) significantly improved recovery of beam-walking and neurological scores in the first 3 weeks after CCI, and EP treatments significantly improved spatial working memory 1 week post-CCI. Ipsilateral CA3(b) neuronal loss, but not cortical tissue loss, was significantly reduced 1 month post-CCI with pyruvate treatments begun 1 h post-CCI. Thus, delayed pyruvate treatments after CCI are neuroprotective and improve neurobehavioral recovery; these effects may be mediated by improved metabolism and reduced inflammation. (C) 2010 Elsevier Inc. All rights reserved.

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TI Role of Microglia in Neurotrauma

SO NEUROTHERAPEUTICS

LA English

DT Review

DE Microglia; spinal cord injury; traumatic brain injury; inflammation

ID SPINAL-CORD-INJURY; TRAUMATIC BRAIN-INJURY; TUMOR-NECROSIS-FACTOR;

INTERLEUKIN-1 RECEPTOR ANTAGONIST; PPAR-GAMMA AGONISTS; INTRACEREBRAL

INFLAMMATORY RESPONSE; METABOTROPIC GLUTAMATE RECEPTORS; IMPROVES

FUNCTIONAL RECOVERY; MACROPHAGE-GENE-EXPRESSION; AMYLOID PROTEIN

DEPOSITION

AB Microglia are the primary mediators of the immune defense system of the CNS and are integral to the subsequent inflammatory response. The role of microglia in the injured CNS is under scrutiny, as research has begun to fully explore how postinjury inflammation contributes to secondary damage and recovery of function. Whether microglia are good or bad is under debate, with strong support for a dual role or differential activation of microglia. Microglia release a number of factors that modulate secondary injury and recovery after injury, including pro-and anti-inflammatory cytokines, chemokines, nitric oxide, prosta-glandins, growth factors, and superoxide species. Here we review experimental work on the complex and varied responses of microglia in terms of both detrimental and beneficial effects. Addressed in addition are the effects of microglial activation in two examples of CNS injury: spinal cord and traumatic brain injury. Microglial activation is integral to the response of CNS tissue to injury. In that light, future research is needed to focus on clarifying the signals and mechanisms by which microglia can be guided to promote optimal functional recovery.

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TI Traumatic Brain Injury Exacerbates Neurodegenerative Pathology:

Improvement with an Apolipoprotein E-Based Therapeutic

SO JOURNAL OF NEUROTRAUMA

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DT Article

DE Alzheimer's disease; amyloid; apolipoprotein E; microglia;

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ID AMYLOID PRECURSOR PROTEIN; CLOSED-HEAD INJURY; E-BASED PEPTIDE;

ALZHEIMERS-DISEASE; MOUSE MODEL; MURINE MODEL; SUBARACHNOID HEMORRHAGE;

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AB Cognitive impairment is common following traumatic brain injury (TBI), and neuroinflammatory mechanisms may predispose to the development of neurodegenerative disease. Apolipoprotein E (apoE) polymorphisms modify neuroinflammatory responses, and influence both outcome from acute brain injury and the risk of developing neurodegenerative disease. We demonstrate that TBI accelerates neurodegenerative pathology in double-transgenic animals expressing the common human apoE alleles and mutated amyloid precursor protein, and that pathology is exacerbated in the presence of the apoE4 allele. The administration of an apoE-mimetic peptide markedly reduced the development of neurodegenerative pathology in mice homozygous for apoE3 as well as apoE3/E4 heterozygotes. These results demonstrate that TBI accelerates the cardinal neuropathological features of neurodegenerative disease, and establishes the potential for apoE mimetic therapies in reducing pathology associated with neurodegeneration.

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TI Post-Traumatic Hypoxia Exacerbates Brain Tissue Damage: Analysis of

Axonal Injury and Glial Responses

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE astrocytes; hypoxia; inflammation; macrophages; microglia; traumatic

axonal injury

ID AMYLOID PRECURSOR PROTEIN; INTRAAXONAL NEUROFILAMENT COMPACTION;

CENTRAL-NERVOUS-SYSTEM; CLOSED-HEAD INJURY; INFLAMMATORY RESPONSE;

ULTRASTRUCTURAL-CHANGES; AXOLEMMAL PERMEABILITY; MICROGLIAL ACTIVATION;

REACTIVE ASTROCYTES; SECONDARY INSULTS

AB Traumatic brain injury (TBI) resulting in poor neurological outcome is predominantly associated with diffuse brain damage and secondary hypoxic insults. Post-traumatic hypoxia is known to exacerbate primary brain injury; however, the underlying pathological mechanisms require further elucidation. Using a rat model of diffuse traumatic axonal injury (TAI) followed by a post-traumatic hypoxic insult, we characterized axonal pathology, macrophage/microglia accumulation, and astrocyte responses over 14 days. Rats underwent TAI alone, TAI followed by 30 min of hypoxia (TAI+Hx), hypoxia alone, or sham-operation (n=6/group). Systemic hypoxia was induced by ventilating rats with 12% oxygen in nitrogen, resulting in a similar to 50% reduction in arterial blood oxygen saturation. Brains were assessed for axonal damage, macrophage/microglia accumulation, and astrocyte activation at 1,7, and 14 days post-treatment. Immunohistochemistry with axonal damage markers (beta-amyloid precursor protein [beta-APP] and neurofilament) showed strong positive staining in TAI+Hx rats, which was most prominent in the corpus callosum (retraction bulbs 69.8 +/- 18.67; swollen axons 14.2 +/- 5.25), and brainstem (retraction bulbs 294 +/- 118.3; swollen axons 50.3 +/- 20.45) at 1 day post-injury. Extensive microglia/macrophage accumulation detected with the CD68 antibody was maximal at 14 days post-injury in the corpus callosum (macrophages 157.5 +/- 55.48; microglia 72.71 +/- 20.75), and coincided with regions of axonal damage. Astrocytosis assessed with glial fibrillary acidic protein (GFAP) antibody was also abundant in the corpus callosum and maximal at 14 days, with a trend toward an increase in TAI+Hx animals (18.99 +/- 2.45 versus 13.56 +/- 0.81; p=0.0617). This study demonstrates for the first time that a hypoxic insult following TAI perpetuates axonal pathology and cellular inflammation, which may account for the poor neurological outcomes seen in TBI patients who experience post-traumatic hypoxia.

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TI Increase in Blood-Brain Barrier Permeability, Oxidative Stress, and

Activated Microglia in a Rat Model of Blast-Induced Traumatic Brain

Injury

SO JOURNAL OF NEUROSCIENCE RESEARCH

LA English

DT Article

DE traumatic brain injury; oxidative stress; inflammation; blood-brain

barrier; PK11195

ID BENZODIAZEPINE BINDING-SITES; PERIPHERAL BENZODIAZEPINE; EXERCISE

PERFORMANCE; INDUCED NEUROTRAUMA; NEURONAL DEATH; LUNG INJURY; MOUSE

MODEL; HEAD-INJURY; TIME-COURSE; EXPOSURE

AB Traumatic brain injury (TBI) as a consequence of exposure to blast is increasingly prevalent in military populations, with the underlying pathophysiological mechanisms mostly unknown. In the present study, we utilized an air-driven shock tube to investigate the effects of blast exposure (120 kPa) on rat brains. Immediately following exposure to blast, neurological function was reduced. BBB permeability was measured using IgG antibody and evaluating its immunoreactivity in the brain. At 3 and 24 hr postexposure, there was a transient significant increase in IgG staining in the cortex. At 3 days postexposure, IgG immunoreactivity returned to control levels. Quantitative immunostaining was employed to determine the temporal course of brain oxidative stress following exposure to blast. Levels of 4-hydroxynonenal (4-HNE) and 3-nitrotyrosine (3-NT) were significantly increased at 3 hr postexposure and returned to control levels at 24 hr postexposure. The response of microglia to blast exposure was determined by autoradiographic localization of H-3-PK11195 binding. At 5 days postexposure, increased binding was observed in the contralateral and ipsilateral dentate gyrus. These regions also displayed increased binding at 10 days postexposure; in addition to these regions there was increased binding in the contralateral ventral hippocampus and substantia nigra at this time point. By using antibodies against CD11b/c, microglia morphology characteristic of activated microglia was observed in the hippocampus and substantia nigra of animals exposed to blast. These results indicate that BBB breakdown, oxidative stress, and microglia activation likely play a role in the neuropathology associated with TBI as a result of blast exposure. (C) 2010 Wiley-Liss, Inc.

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TI Pioglitazone attenuates mitochondrial dysfunction, cognitive impairment,

cortical tissue loss, and inflammation following traumatic brain injury

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Traumatic brain injury; Pioglitazone; Mitochondria; Inflammation;

Microglia; Cortical lesion; Cortical impact; Contusion; PPAR; Peroxisome

proliferator-activated receptor

ID ACTIVATED-RECEPTOR-GAMMA; NITRIC-OXIDE SYNTHASE; SPINAL-CORD-INJURY;

AMYOTROPHIC-LATERAL-SCLEROSIS; PPAR-ALPHA AGONIST; NF-KAPPA-B;

CELL-DEATH; OXIDATIVE STRESS; MOUSE MODEL; DOPAMINERGIC

NEURODEGENERATION

AB Following traumatic brain injury (TBI) there is significant neuropathology which includes mitochondrial dysfunction, loss of cortical gray matter, microglial activation, and cognitive impairment. Previous evidence has shown that activation of the peroxisome proliferator-activated receptors (PPARs) provide neuroprotection following traumatic brain and spinal injuries. In the current study we hypothesized that treatment with the PPAR ligand Pioglitazone would promote neuroprotection following a rat controlled cortical impact model of TBI. Animals received a unilateral 1.5 mm controlled cortical impact followed by administration of Pioglitazone at 10 mg/kg beginning 15 min after the injury and subsequently every 24h for 5 days. Beginning 1 day after the injury there was significant impairment in mitochondrial bioenergetic function which was attenuated by treatments with Pioglitazone at 15 min and 24 h ( p< 0.05). In an additional set of animals, cognitive function was assessed using the Morris Water Maze (MWM) and it was observed that over the course of 4 days of testing the injury produced a significant increase in both latency ( p< 0.05) and distance ( p< 0.05) to the platform. Animals treated with Pioglitazone performed similarly to sham animals and did not exhibit any impairment in MWM performance. Sixteen days after the injury tissue sections through the lesion site were quantified to determine the size of the cortical lesion. Vehicle-treated animals had an average lesion size of 5.09 +/- 0.73 mm(3) and treatment with Pioglitazone significantly reduced the lesion size by 55% to 2.27 +/- 0.27 mm(3) ( p< 0.01). Co-administration of the antagonist T0070907 with Pioglitazone blocked the protective effect seen with administration of Pioglitazone by itself. Following the injury there was a significant increase in the number of activated microglia in the area of the cortex adjacent to the site of the lesion ( p< 0.05). Treatment with Pioglitazone prevented the increase in the number of activated microglia and no difference was observed between sham and Pioglitazone-treated animals. From these studies we conclude that following TBI Pioglitazone is capable ameliorating multiple aspects of neuropathology. These studies provide further support for the use of PPAR ligands, specifically Pioglitazone, for neuroprotection. (C) 2010 Elsevier Inc. All rights reserved.

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TI Multiple sites of vasopressin synthesis in the injured brain

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE activated microglia; arginine vasopressin; cerebrovascular endothelium;

macrophages; traumatic brain injury

ID RAT CHOROID-PLEXUS; ARGININE-VASOPRESSIN; EXPRESSION; RECEPTORS;

KINETICS

AB Previous studies have indicated that the primary targets for vasopressin actions on the injured brain are the cerebrovascular endothelium and astrocytes, and that vasopressin amplifies the posttraumatic production of proinflammatory mediators. Here, the controlled cortical impact model of traumatic brain injury in rats was used to identify the sources of vasopressin in the injured brain. Injury increased vasopressin synthesis in the hypothalamus and cerebral cortex adjacent to the posttraumatic lesion. In the cortex, vasopressin was predominantly produced by activated microglia/macrophages, and, to a lesser extent, by the cerebrovascular endothelium. These data further support the pathophysiological role of vasopressin in brain injury. Journal of Cerebral Blood Flow & Metabolism (2011) 31, 47-51; doi:10.1038/jcbfm.2010.188; published online 20 October 2010

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TI Expression and Localization of the Orexin-1 Receptor (OX1R) After

Traumatic Brain Injury in Mice

SO JOURNAL OF MOLECULAR NEUROSCIENCE

LA English

DT Article

DE Orexin-1 receptor (OX1R); Orexin; Traumatic brain injury; Neuron;

Microglia; Immunohistochemistry

ID NARCOLEPSY; MICROGLIA; APOPTOSIS; DEFICIENCY; PEPTIDES; NEURONS

AB Orexins are neuropeptides that have a wide range of physiological effects, and recent studies have suggested that the orexin system may be involved in traumatic brain injury. However, the expression and localization of orexin receptors have not been examined yet under brain injury conditions. In the present study, we used immunohistochemical techniques to investigate the expression of orexin-1 receptor (OX1R) and its time-dependent changes in the mouse brain after controlled cortical impact (CCI) injury. OX1R-like immunoreactivity was first detected 6 h after injury in the surrounding penumbra of the injury. The intensity of this immunoreactivity was increased at 12 h, peaked at day 1, and then decreased from day 2 to day 7. To identify the cellular localization of OX1R, we also performed double-immunohistochemical staining with OX1R and several cell marker antibodies. OX1R-like immunopositive cells were clearly co-localized with immunoreactivity for the neuronal marker NeuN at day 7. It was also expressed on the periphery of cells immunopositive for CD11b, a microglial cell marker, at days 1 and 7. These results suggest that orexin and its receptor may play roles in traumatic brain injury, and that OX1R is induced in neurons and microglial cells after traumatic brain injury.

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Sun, W

AF Moon, Younghye

Kim, Joo Yeon

Choi, So Yoen

Kim, Kyungjin

Kim, Hyun

Sun, Woong

TI Induction of ezrin-radixin-moesin molecules after cryogenic traumatic

brain injury of the mouse cortex

SO NEUROREPORT

LA English

DT Article

DE ezrin-radixin-moesin; macrophage; microglia; traumatic brain injury

ID ADULT SUBVENTRICULAR ZONE; ROSTRAL MIGRATORY STREAM; ERM PROTEINS;

EXPRESSION; CELLS

AB Traumatic brain injury promotes rapid induction of microglial cells and infiltration of peripheral macrophages to the injury sites. Such inflammatory responses are mediated by the activation and migration of immune cells, which are influenced by the actin cytoskeleton remodeling. In this study, we observed that the phosphorylation and expressions of ezrin-radixin-moesin (ERM) proteins, which are linkers for cell surface with actin cytoskeleton, are induced in the activated microglia/macrophages, whereas ERM molecules are only marginally expressed in quiescent microglia in the normal brain. These results suggest that ERM activation in the injury penumbra is implicated in the inflammatory immune responses after traumatic brain injury. NeuroReport 22:304-308 (C) 2011 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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U2 4

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AF Shitaka, Yoshitsugu

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Levy, Marilyn A.

Dikranian, Krikor

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TI Repetitive Closed-Skull Traumatic Brain Injury in Mice Causes Persistent

Multifocal Axonal Injury and Microglial Reactivity

SO JOURNAL OF NEUROPATHOLOGY AND EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Axonal injury; Electron microscopy; Microglia; Mouse; Repetitive

concussion; Silver staining; Traumatic brain injury

ID CONTROLLED CORTICAL IMPACT; AMYLOID PRECURSOR PROTEIN; MILD HEAD-INJURY;

COGNITIVE IMPAIRMENT; EXPLOSIVE BLAST; UNITED-STATES; MOUSE MODEL;

VULNERABILITY; PERFORMANCE; FOOTBALL

AB Repetitive mild or "concussive" traumatic brain injury (TBI) can cause substantial neurologic impairment, but the pathological features of this type of injury are not fully understood. We report an experimental model of TBI in which the closed skulls of anesthetized male C57BL/6J mice are struck with an electromagnetically controlled rubber impactor twice with an interval of 24 hours between impacts. The mice had deficits in Morris water maze performance in the first week after injury that only partially resolved 7 weeks later. By routine histology, there was no apparent bleeding, neuronal cell loss, or tissue disruption, and amyloid precursor protein immunohistochemistry demonstrated very few immunoreactive axonal varicosities. In contrast, silver staining revealed extensive abnormalities in the corpus callosum and bilateral external capsule, the ipsilateral cortex and thalamus, and the contralateral hippocampal CA1 stratum radiatum and stratum oriens. Electron microscopy of white matter regions demonstrated axonal cytoskeletal disruption, intra-axonal organelle compaction, and irregularities in axon caliber. Reactive microglia were observed in the same areas as the injured axons by both electron microscopy and Iba1 immunohistochemistry. Quantitative analyses of silver staining and Iba1 immunohistochemistry at multiple time points demonstrated transient cortical and thalamic abnormalities but persistent white matter pathology as late as 7 weeks after injury. Thus, prominent and long-lasting abnormalities in this TBI model were underestimated using conventional approaches. The model may be useful for mechanistic investigations and preclinical assessment of candidate therapeutics.

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NR 49

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U1 1

U2 24

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JI J. Neuropathol. Exp. Neurol.

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TI Neutralization of interleukin-1β reduces cerebral edema and tissue loss

and improves late cognitive outcome following traumatic brain injury in

mice

SO EUROPEAN JOURNAL OF NEUROSCIENCE

LA English

DT Article

DE behavior; cognition; edema; microglia; traumatic brain injury

ID CONTROLLED CORTICAL IMPACT; AMYLOID PRECURSOR PROTEIN; FLUID PERCUSSION

INJURY; LONG-TERM POTENTIATION; CLOSED-HEAD INJURY; INFLAMMATORY

RESPONSE; RECEPTOR ANTAGONIST; SUBARACHNOID HEMORRHAGE; EXPERIMENTAL

STROKE; MEMORY PROCESSES

AB Increasing evidence suggests that interleukin-1 beta (IL-1 beta) is a key mediator of the inflammatory response following traumatic brain injury (TBI). Recently, we showed that intracerebroventricular administration of an IL-1 beta-neutralizing antibody was neuroprotective following TBI in mice. In the present study, an anti-IL-1 beta antibody or control antibody was administered intraperitoneally following controlled cortical injury (CCI) TBI or sham injury in 105 mice and we extended our histological, immunological and behavioral analysis. First, we demonstrated that the treatment antibody reached target brain regions of brain-injured animals in high concentrations (> 11 nm) remaining up to 8 days post-TBI. At 48 h post-injury, the anti-IL-1b treatment attenuated the TBI-induced hemispheric edema (P < 0.05) but not the memory deficits evaluated using the Morris water maze (MWM). Neutralization of IL-1 beta did not influence the TBI-induced increases (P < 0.05) in the gene expression of the Ccl3 and Ccr2 chemokines, IL-6 or Gfap. Up to 20 days post-injury, neutralization of IL-1 beta was associated with improved visuospatial learning in the MWM, reduced loss of hemispheric tissue and attenuation of the microglial activation caused by TBI (P < 0.05). Motor function using the rotarod and cylinder tests was not affected by the anti-IL-1 beta treatment. Our results suggest an important negative role for IL-1 beta in TBI. The improved histological and behavioral outcome following anti-IL-1 beta treatment also implies that further exploration of IL-1 beta-neutralizing compounds as a treatment option for TBI patients is warranted.

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WC Neurosciences

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PT J

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TI Neurogenesis and Glial Proliferation Are Stimulated Following Diffuse

Traumatic Brain Injury in Adult Rats

SO JOURNAL OF NEUROSCIENCE RESEARCH

LA English

DT Article

DE neurogenesis; traumatic axonal injury rat model; astrocytes; microglia;

polydendrocytes

ID INTERLEUKIN-6 MESSENGER-RNA; SUBVENTRICULAR ZONE CELLS; FOCAL

CEREBRAL-ISCHEMIA; AXONAL INJURY; HIPPOCAMPAL NEUROGENESIS; DENTATE

GYRUS; CORTICAL NEUROGENESIS; COGNITIVE RECOVERY; MAMMALIAN BRAIN;

STEM-CELLS

AB Although increased neurogenesis has been described in rodent models of focal traumatic brain injury (TBI), the neurogenic response occurring after diffuse TBI uncomplicated by focal injury has not been examined to date, despite the pervasiveness of this distinct type of brain injury in the TBI patient population. Here we characterize multiple stages of neurogenesis following a traumatic axonal injury (TAI) model of diffuse TBI as well as the proliferative response of glial cells. TAI was induced in adult rats using an impact-acceleration model, and 5-bromo-20-deoxyuridine (BrdU) was administered on days 1-4 posttrauma or sham operation to label mitotic cells. Using immunohistochemistry for BrdU combined with phenotype-specific markers, we found that proliferation was increased following TAI in the subventricular zone of the lateral ventricles and in the hippocampal subgranular zone, although the ultimate production of new dentate granule neurons at 8 weeks was not significantly enhanced. Also, abundant proliferating and reactive astrocytes, microglia, and polydendrocytes were detected throughout the brain following TAI, indicating that a robust glial response occurs in this model, although very few new cells in the nonneurogenic brain regions became mature neurons. We conclude that diffuse brain injury stimulates early stages of a neurogenic response similar to that described for models of focal TBI. (C) 2011 Wiley-Liss, Inc.

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PU WILEY-BLACKWELL

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PT J

AU Folkersma, H

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TI Widespread and Prolonged Increase in (<i>R</i>)-<SUP>11</SUP>C-PK11195

Binding After Traumatic Brain Injury

SO JOURNAL OF NUCLEAR MEDICINE

LA English

DT Article

DE craniocerebral trauma; humans; microglia; positron emission tomography;

(R)-C-11-PK11195

ID IN-VIVO; MICROGLIAL ACTIVATION; PERIPHERAL BENZODIAZEPINE;

COMPUTERIZED-TOMOGRAPHY; DEMENTIA; RECEPTOR; SYSTEM; SITES; MODEL

AB Our objective was to measure (R)-C-11-PK11195 binding as an indirect marker of neuronal damage after traumatic brain injury (TBI). Methods: Dynamic (R)-C-11-PK11195 PET scans were acquired for 8 patients 6 mo after TBI and for 7 age-matched healthy controls. (R)-C-11-PK11195 binding was assessed using the simplified reference tissue model. Because of widespread traumatic changes in TBI, an anatomic reference region could not be defined. Therefore, supervised cluster analysis was used to generate an appropriate reference tissue input. Results: Increased whole-brain binding of (R)-C-11-PK11195 was observed in TBI patients. Regional analysis indicated that increased (R)-C-11-PK11195 binding was widespread over the brain. Conclusion: Six months after TBI, there was a prolonged and widespread increase in (R)-C-11-PK11195 binding, which is indicative of diffuse neuronal damage.

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Z9 62

U1 0

U2 14

PU SOC NUCLEAR MEDICINE INC

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J9 J NUCL MED

JI J. Nucl. Med.

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PT J

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TI Modulation of traumatic brain injury using progesterone and the role of

glial cells on its neuroprotective actions

SO JOURNAL OF NEUROIMMUNOLOGY

LA English

DT Review

DE Progesterone; Traumatic brain injury; Neuroprotection; Glial cells;

Microglia

ID CORTICAL CONTUSION; NEURONAL LOSS; SEX STEROIDS; INFLAMMATORY RESPONSE;

NERVOUS-SYSTEM; HEAD-INJURY; EDEMA; RECOVERY; HORMONES; ALLOPREGNANOLONE

AB TBI is a complex disease process caused by a cascade of systemic events. Attention is now turning to drugs that act on multiple pathways to enhance survival and functional outcomes. Progesterone has been found to be beneficial in several animal species, different models of brain injury, and in two preliminary human clinical trials. It holds promise as a treatment for TBI. Progesterone's multiple mechanisms of action may work synergistically to prevent the death of neurons and glia, leading to reduced morbidity and mortality. This review highlights the importance of glial cells as mediators of progesterone's actions on the CNS and describes progesterone's pleiotrophic effects on immune enhancement and neuroprotection in TBI. (C) 2011 Elsevier B.V. All rights reserved.

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NR 85

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TI Inflammation after Trauma: Microglial Activation and Traumatic Brain

Injury

SO ANNALS OF NEUROLOGY

LA English

DT Article

ID HEAD-INJURY; NERVOUS-SYSTEM; RHESUS-MONKEY; RAT-BRAIN;

NEURODEGENERATION; ASTROCYTES; STROKE; DEATH; TIME; PET

AB Objective: Patient outcome after traumatic brain injury (TBI) is highly variable. The underlying pathophysiology of this is poorly understood, but inflammation is potentially an important factor. Microglia orchestrate many aspects of this response. Their activation can be studied in vivo using the positron emission tomography (PET) ligand [11C](R)PK11195 (PK). In this study, we investigate whether an inflammatory response to TBI persists, and whether this response relates to structural brain abnormalities and cognitive function.

Methods: Ten patients, studied at least 11 months after moderate to severe TBI, underwent PK PET and structural magnetic resonance imaging (including diffusion tensor imaging). PK binding potentials were calculated in and around the site of focal brain damage, and in selected distant and subcortical brain regions. Standardized neuropsychological tests were administered.

Results: PK binding was significantly raised in the thalami, putamen, occipital cortices, and posterior limb of the internal capsules after TBI. There was no increase in PK binding at the original site of focal brain injury. High PK binding in the thalamus was associated with more severe cognitive impairment, although binding was not correlated with either the time since the injury or the extent of structural brain damage.

Interpretation: We demonstrate that increased microglial activation can be present up to 17 years after TBI. This suggests that TBI triggers a chronic inflammatory response particularly in subcortical regions. This highlights the importance of considering the response to TBI as evolving over time and suggests interventions may be beneficial for longer intervals after trauma than previously assumed. ANN NEUROL 2011;70:374-383

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TI Traumatic Brain Injury in Adult Rats Causes Progressive Nigrostriatal

Dopaminergic Cell Loss and Enhanced Vulnerability to the Pesticide

Paraquat

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE alpha-synuclein; lateral fluid percussion; microglia; paraquat;

Parkinson's disease; traumatic brain injury

ID LIPOPOLYSACCHARIDE-INDUCED NEUROTOXICITY; INTRACEREBRAL INFLAMMATORY

RESPONSE; NONSTEROIDAL ANTIINFLAMMATORY DRUGS; ENVIRONMENTAL

RISK-FACTORS; NIGRA PARS COMPACTA; PARKINSONS-DISEASE; SUBSTANTIA-NIGRA;

MICROGLIAL ACTIVATION; AXONAL INJURY; COMPLEX-I

AB Parkinson's disease (PD) is a neurodegenerative disorder characterized by the loss of nigrostriatal dopaminergic neurons and the accumulation of alpha-synuclein. Both traumatic brain injury (TBI) and pesticides are risk factors for PD, but whether TBI causes nigrostriatal dopaminergic cell loss in experimental models and whether it acts synergistically with pesticides is unknown. We have examined the acute and long-term effects of TBI and exposure to low doses of the pesticide paraquat, separately and in combination, on nigrostriatal dopaminergic neurons in adult male rats. In an acute study, rats received moderate TBI by lateral fluid percussion (LFP) injury, were injected with saline or paraquat (10 mg/kg IP) 3 and 6 days after LFP, were sacrificed 5 days later, and their brains processed for immunohistochemistry. TBI alone increased microglial activation in the substantia nigra, and caused a 15% loss of dopaminergic neurons ipsilaterally. Paraquat increased the TBI effect, causing a 30% bilateral loss of dopaminergic neurons, reduced striatal tyrosine hydroxylase (TH) immunoreactivity more than TBI alone, and induced alpha-synuclein accumulation in the substantia nigra pars compacta. In a long-term study, rats received moderate LFP, were injected with saline or paraquat at 21 and 22 weeks post-injury, and were sacrificed 4 weeks later. At 26 weeks post injury, TBI alone induced a 30% bilateral loss of dopaminergic neurons that was not exacerbated by paraquat. These data suggest that TBI is sufficient to induce a progressive degeneration of nigrostriatal dopaminergic neurons. Furthermore, TBI and pesticide exposure, when occurring within a defined time frame, could combine to increase the PD risk.

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TI Human umbilical cord blood mesenchymal stem cells protect mice brain

after trauma

SO CRITICAL CARE MEDICINE

LA English

DT Article

DE human cord blood mesenchymal stem cells; traumatic brain injury;

microglia; transplantation; functional recovery; brain protection

ID MARROW STROMAL CELLS; BONE-MARROW; GENE-EXPRESSION; IN-VITRO; INJURY;

TRANSPLANTATION; NEUROPROTECTION; GROWTH; INHIBITION; CHALLENGES

AB Objective: To investigate whether human umbilical cord blood mesenchymal stem cells, a novel source of progenitors with multilineage potential: 1) decrease traumatic brain injury sequelae and restore brain function; 2) are able to survive and home to the lesioned region; and 3) induce relevant changes in the environment in which they are infused.

Design: Prospective experimental study.

Setting: Research laboratory.

Subjects: Male C57BI/6 mice.

Interventions: Mice were subjected to controlled cortical impact/sham brain injury. At 24 hrs postinjury, human umbilical cord blood mesenchymal stem cells (150,000/5 mu L) or phosphate-buffered saline (control group) were infused intracerebroventricularly contralateral to the injured side. Immunosuppression was achieved by cyclosporine A (10 mg/kg intraperitoneally).

Measurements and Main Results: After controlled cortical impact, human umbilical cord blood mesenchymal stem cell transplantation induced an early and long-lasting improvement in sensorimotor functions assessed by neuroscore and beam walk tests. One month postinjury, human umbilical cord blood mesenchymal stem cell mice showed attenuated learning dysfunction at the Morris water maze and reduced contusion volume compared with controls. Hoechst positive human umbilical cord blood mesenchymal stem cells homed to lesioned tissue as early as 1 wk after injury in 67% of mice and survived in the injured brain up to 5 wks. By 3 days postinjury, cell infusion significantly increased brain-derived neurotrophic factor concentration into the lesioned tissue, restoring its expression close to the levels observed in sham operated mice. By 7 days postinjury, controlled cortical impact human umbilical cord blood mesenchymal stem cell mice showed a nonphagocytic activation of microglia/macrophages as shown by a selective rise (260%) in CD11b staining (a marker of microglia/macrophage activation/recruitment) associated with a decrease (58%) in CD68 (a marker of active phagocytosis). Thirty-five days postinjury, controlled cortical impact human umbilical cord blood mesenchymal stem cell mice showed a decrease of glial fibrillary acidic protein positivity in the scar region compared with control mice.

Conclusions: These findings indicate that human umbilical cord blood mesenchymal stem cells stimulate the injured brain and evoke trophic events, microglia/macrophage phenotypical switch, and glial scar inhibitory effects that remodel the brain and lead to significant improvement of neurologic outcome. (Crit Care Med 2011; 39: 2501-2510)

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TI Selective CDK inhibitor limits neuroinflammation and progressive

neurodegeneration after brain trauma

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE brain trauma; cell cycle; cyclin-dependent kinases; microglial

activation; neurodegeneration

ID CONTROLLED CORTICAL IMPACT; CELL-CYCLE ACTIVATION; ALZHEIMERS-DISEASE;

SPATIAL MEMORY; INJURY; ROSCOVITINE; DAMAGE; NEURONS; DEATH; MICROGLIA

AB Traumatic brain injury (TBI) induces secondary injury mechanisms, including cell-cycle activation (CCA), which lead to neuronal cell death, microglial activation, and neurologic dysfunction. Here, we show progressive neurodegeneration associated with microglial activation after TBI induced by controlled cortical impact (CCI), and also show that delayed treatment with the selective cyclin-dependent kinase inhibitor roscovitine attenuates posttraumatic neurodegeneration and neuroinflammation. CCI resulted in increased cyclin A and D1 expressions and fodrin cleavage in the injured cortex at 6 hours after injury and significant neurodegeneration by 24 hours after injury. Progressive neuronal loss occurred in the injured hippocampus through 21 days after injury and correlated with a decline in cognitive function. Microglial activation associated with a reactive microglial phenotype peaked at 7 days after injury with sustained increases at 21 days. Central administration of roscovitine at 3 hours after CCI reduced subsequent cyclin A and D1 expressions and fodrin cleavage, improved functional recovery, decreased lesion volume, and attenuated hippocampal and cortical neuronal cell loss and cortical microglial activation. Furthermore, delayed systemic administration of roscovitine improved motor recovery and attenuated microglial activation after CCI. These findings suggest that CCA contributes to progressive neurodegeneration and related neurologic dysfunction after TBI, likely in part related to its induction of microglial activation. Journal of Cerebral Blood Flow & Metabolism (2012) 32, 137-149; doi:10.1038/jcbfm.2011.117; published online 10 August 2011

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TI Neutrophil depletion reduces edema formation and tissue loss following

traumatic brain injury in mice

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Neutrophil; traumatic brain injury; brain edema; controlled cortical

impact; neuroprotection; blood-brain-barrier; cell death; microglia;

neutrophil-depletion; mouse

ID CONTROLLED CORTICAL IMPACT; CLOSED-HEAD INJURY; MIDDLE CEREBRAL-ARTERY;

BARRIER PERMEABILITY; ISCHEMIC BRAIN; POLYMORPHONUCLEAR LEUKOCYTES;

VASCULAR-PERMEABILITY; ADHESION MOLECULES; TNF-ALPHA; RATS

AB Background: Brain edema as a result of secondary injury following traumatic brain injury (TBI) is a major clinical concern. Neutrophils are known to cause increased vascular permeability leading to edema formation in peripheral tissue, but their role in the pathology following TBI remains unclear.

Methods: In this study we used controlled cortical impact (CCI) as a model for TBI and investigated the role of neutrophils in the response to injury. The outcome of mice that were depleted of neutrophils using an anti-Gr-1 antibody was compared to that in mice with intact neutrophil count. The effect of neutrophil depletion on blood-brain barrier function was assessed by Evan's blue dye extravasation, and analysis of brain water content was used as a measurement of brain edema formation (24 and 48 hours after CCI). Lesion volume was measured 7 and 14 days after CCI. Immunohistochemistry was used to assess cell death, using a marker for cleaved caspase-3 at 24 hours after injury, and microglial/macrophage activation 7 days after CCI. Data were analyzed using Mann-Whitney test for non-parametric data.

Results: Neutrophil depletion did not significantly affect Evan's blue extravasation at any time-point after CCI. However, neutrophil-depleted mice exhibited a decreased water content both at 24 and 48 hours after CCI indicating reduced edema formation. Furthermore, brain tissue loss was attenuated in neutropenic mice at 7 and 14 days after injury. Additionally, these mice had a significantly reduced number of activated microglia/macrophages 7 days after CCI, and of cleaved caspase-3 positive cells 24 h after injury.

Conclusion: Our results suggest that neutrophils are involved in the edema formation, but not the extravasation of large proteins, as well as contributing to cell death and tissue loss following TBI in mice.

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TI Microglial activation induced by brain trauma is suppressed by

post-injury treatment with a PARP inhibitor

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Astrocyte; Behavioral; Forelimb; Inflammation; Microglia; Minocycline;

Poly(ADP-ribose) polymerase; traumatic brain injury

ID NF-KAPPA-B; POLY(ADP-RIBOSE) POLYMERASE INHIBITOR; NAD(+) DEPLETION;

NEURONAL DEATH; MOLECULAR-MECHANISMS; THERAPEUTIC TARGETS;

CEREBRAL-ISCHEMIA; INJURY; INFLAMMATION; CNS

AB Background: Traumatic brain injury (TBI) induces activation of microglia. Activated microglia can in turn increase secondary injury and impair recovery. This innate immune response requires hours to days to become fully manifest, thus providing a clinically relevant window of opportunity for therapeutic intervention. Microglial activation is regulated in part by poly(ADP-ribose) polymerase-1 (PARP-1). Inhibition of PARP-1 activity suppresses NF-kB-dependent gene transcription and thereby blocks several aspects of microglial activation. Here we evaluated the efficacy of a PARP inhibitor, INO-1001, in suppressing microglial activation after cortical impact in the rat.

Methods: Rats were subjected to controlled cortical impact and subsequently treated with 10 mg/kg of INO-1001 (or vehicle alone) beginning 20 - 24 hours after the TBI. Brains were harvested at several time points for histological evaluation of inflammation and neuronal survival, using markers for microglial activation (morphology and CD11b expression), astrocyte activation (GFAP), and neuronal survival (NeuN). Rats were also evaluated at 8 weeks after TBI using measures of forelimb dexterity: the sticky tape test, cylinder test, and vermicelli test.

Results: Peak microglial and astrocyte activation was observed 5 to 7 days after this injury. INO-1001 significantly reduced microglial activation in the peri-lesion cortex and ipsilateral hippocampus. No rebound inflammation was observed in rats that were treated with INO-1001 or vehicle for 12 days followed by 4 days without drug. The reduced inflammation was associated with increased neuronal survival in the peri-lesion cortex and improved performance on tests of forelimb dexterity conducted 8 weeks after TBI.

Conclusions: Treatment with a PARP inhibitor for 12 days after TBI, with the first dose given as long as 20 hours after injury, can reduce inflammation and improve histological and functional outcomes.

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TI Delayed mGluR5 activation limits neuroinflammation and neurodegeneration

after traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

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DT Article

DE Traumatic brain injury; microglia; metabotropic glutamate receptor 5;

delayed treatment; neuroprotection

ID METABOTROPIC GLUTAMATE RECEPTORS; AUTOMATED IMAGE REGISTRATION;

CONTROLLED CORTICAL IMPACT; DIFFUSION-TENSOR; LONGITUDINAL CHANGES;

PROGRESSIVE ATROPHY; NEURONAL APOPTOSIS; MICROGLIA; NEUROTOXICITY;

INFLAMMATION

AB Background: Traumatic brain injury initiates biochemical processes that lead to secondary neurodegeneration. Imaging studies suggest that tissue loss may continue for months or years after traumatic brain injury in association with chronic microglial activation. Recently we found that metabotropic glutamate receptor 5 (mGluR5) activation by (RS)-2-chloro-5-hydroxyphenylglycine (CHPG) decreases microglial activation and release of associated pro-inflammatory factors in vitro, which is mediated in part through inhibition of reduced nicotinamide adenine dinucleotide phosphate (NADPH) oxidase. Here we examined whether delayed CHPG administration reduces chronic neuroinflammation and associated neurodegeneration after experimental traumatic brain injury in mice.

Methods: One month after controlled cortical impact traumatic brain injury, C57Bl/6 mice were randomly assigned to treatment with single dose intracerebroventricular CHPG, vehicle or CHPG plus a selective mGluR5 antagonist, 3(( 2-Methyl-4-thiazolyl)ethynyl) pyridine. Lesion volume, white matter tract integrity and neurological recovery were assessed over the following three months.

Results: Traumatic brain injury resulted in mGluR5 expression in reactive microglia of the cortex and hippocampus at one month post-injury. Delayed CHPG treatment reduced expression of reactive microglia expressing NADPH oxidase subunits; decreased hippocampal neuronal loss; limited lesion progression, as measured by repeated T2-weighted magnetic resonance imaging (at one, two and three months) and white matter loss, as measured by high field ex vivo diffusion tensor imaging at four months; and significantly improved motor and cognitive recovery in comparison to the other treatment groups.

Conclusion: Markedly delayed, single dose treatment with CHPG significantly improves functional recovery and limits lesion progression after experimental traumatic brain injury, likely in part through actions at mGluR5 receptors that modulate neuroinflammation.

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TI Critical Role of NADPH Oxidase in Neuronal Oxidative Damage and

Microglia Activation following Traumatic Brain Injury

SO PLOS ONE

LA English

DT Article

ID CONTROLLED CORTICAL IMPACT; AMYLOID PRECURSOR PROTEIN;

CENTRAL-NERVOUS-SYSTEM; SUPEROXIDE-PRODUCTION; SYNAPTIC PROTEINS;

ISCHEMIA; OXYGEN; EDEMA; NEUROPROTECTION; ATTENUATION

AB Background: Oxidative stress is known to play an important role in the pathology of traumatic brain injury. Mitochondria are thought to be the major source of the damaging reactive oxygen species (ROS) following TBI. However, recent work has revealed that the membrane, via the enzyme NADPH oxidase can also generate the superoxide radical (O-2(-)), and thereby potentially contribute to the oxidative stress following TBI. The current study thus addressed the potential role of NADPH oxidase in TBI.

Methodology/Principal Findings:The results revealed that NADPH oxidase activity in the cerebral cortex and hippocampal CA1 region increases rapidly following controlled cortical impact in male mice, with an early peak at 1 h, followed by a secondary peak from 24-96 h after TBI. In situ localization using oxidized hydroethidine and the neuronal marker, NeuN, revealed that the O-2(-) induction occurred in neurons at 1 h after TBI. Pre- or post-treatment with the NADPH oxidase inhibitor, apocynin markedly inhibited microglial activation and oxidative stress damage. Apocynin also attenuated TBI-induction of the Alzheimer's disease proteins beta-amyloid and amyloid precursor protein. Finally, both pre- and post-treatment of apocynin was also shown to induce significant neuroprotection against TBI. In addition, a NOX2-specific inhibitor, gp91ds-tat was also shown to exert neuroprotection against TBI.

Conclusions/Significance: As a whole, the study demonstrates that NADPH oxidase activity and superoxide production exhibit a biphasic elevation in the hippocampus and cortex following TBI, which contributes significantly to the pathology of TBI via mediation of oxidative stress damage, microglial activation, and AD protein induction in the brain following TBI.

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TI Impact of inhibition of erythropoietin treatment-mediated neurogenesis

in the dentate gyrus of the hippocampus on restoration of spatial

learning after traumatic brain injury

SO EXPERIMENTAL NEUROLOGY

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DE Astrocytes; Erythropoietin; Microglia; Neurogenesis; Spatial learning;

Traumatic brain injury

ID CONTROLLED CORTICAL IMPACT; MARROW STROMAL CELLS; NEURAL STEM-CELLS;

FUNCTIONAL RECOVERY; NEURONAL APOPTOSIS; CA3 REGION; ADULT-RAT; ENHANCES

NEUROGENESIS; STRIATAL NEUROGENESIS; COGNITIVE RECOVERY

AB Our previous study demonstrates that delayed (initiated 24 h post injury) erythropoietin (EPO) therapy for traumatic brain injury (TBI) significantly improves spatial learning. In this study, we investigated the impact of inhibition of EPO treatment-mediated neurogenesis on spatial learning after experimental TBI. Young male Wistar rats (318 +/- 7 g) were subjected to unilateral controlled cortical impact injury. TBI rats received delayed EPO treatment (5000 U/kg in saline) administered intraperitoneally once daily at 1,2, and 3 days post injury and intracerebroventricular (icy) infusion of either a mitotic inhibitor cytosine-b-D-arabinofuranoside or vehicle (saline) for 14 days. Another 2 groups of TBI rats were treated intraperitoneally with saline and infused icy with either a mitotic inhibitor Ara-C or saline for 14 days. Animals receiving sham operation were infused icy with either Ara-C infusion or saline. Bromodeoxyuridine (BrdU) was administered to label dividing cells. Spatial learning was assessed using a modified Morris water maze test. Animals were sacrificed at 35 days after injury and brain sections stained for immunohistochemical analyses. As compared to the saline treatment, immunohistochemical analysis revealed that delayed EPO treatment significantly increased the number of BrdU-positive cells and new neurons co-stained with BrdU and NeuN (mature neuron marker) in the dentate gyrus in TBI rats. EPO treatment improved spatial learning after TBI. Ara-C infusion significantly abolished neurogenesis and spatial learning recovery after TBI and EPO treatment. Both EPO and Ara-C reduced the number of astrocytes and microglia/macrophages in the dentate gyrus after TBI. Our findings are highly suggestive for an important role of EPO-amplified dentate gyrus neurogenesis as one of the mechanisms underlying EPO therapeutic treatments after TBI, strongly indicating that strategies promoting endogenous neurogenesis may hold an important therapeutic potential for treatment of TBI. (C) 2012 Elsevier Inc. All rights reserved.

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TI Traumatic brain injury in the neonate, child and adolescent human: An

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DE TBI; Hematoma; Immature or unmyelinated axons; Myelination; Neuronal

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response; The neurovascular unit

ID NONDISRUPTIVE AXONAL INJURY; WHITE-MATTER DEVELOPMENT; SITU DNA

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HUMAN FETAL; AXOLEMMAL PERMEABILITY; CYTOSKELETAL CHANGES;

CEREBRAL-CORTEX

AB In the middle of the last century it had been thought that a good recovery of function and behavior would occur after traumatic brain injury (TBI) in very young human beings. A recent major change in thinking states that early childhood TBI may result in a severe compromise of normal brain growth and development such that TBI, rather, may compromise later normal development resulting in a need for very long term patient care and management. The mechanisms of injury and pathology within the injured brain are reviewed and compared between when injury occurs at or close to the time of birth, in an infant, in a young child, in a child between ages 5 and 10, in young and older adolescents and in young adulthood. Our understanding of pathophysiological responses by cells of the human central nervous system has recently greatly increased but has really only served to illustrate the great complexity of interactions between different types of cell within the growing and developing CNS. The hypothesis is developed that the outcome for a very young patient differs with the relative state of development of injured cells at the locus of injury. And that the potential for either repair, re-instatement of normal cellular and organ function or for continued normal development is much reduced after an early brain insult (EBI) compared with TBI in a slightly older child or young adult patient. The advent of increasingly sophisticated non-invasive imaging technology has allowed assessment of the influence and time course of brain pathology both early and late after TBI. This has generated greater confidence on the part of clinicians in forecasting outcomes for an injured patient. But our increased understanding has still not allowed development of therapeutic strategies that might ameliorate the effect of an injury. It is suggested that an improved integration of major clinical and scientific effort needs to be made to appreciate the import of multiple interactions between cells forming the neurovascular unit in order to improve any potential for post-traumatic recovery after TBI in neonates and young children. (C) 2011 ISDN. Published by Elsevier Ltd. All rights reserved.

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TI Prevention of Traumatic Brain Injury-Induced Neuron Death by Intranasal

Delivery of Nicotinamide Adenine Dinucleotide

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DE hippocampus; intranasal; microglia; nicotinamide adenine dinucleotide;

poly(ADP-ribose) polymerase-1; superoxide; traumatic brain injury

ID POLY(ADP-RIBOSE) POLYMERASE INHIBITOR; TRANSIENT FOCAL ISCHEMIA; FLUID

PERCUSSION INJURY; NAD(+) DEPLETION; MICROGLIAL ACTIVATION; CELL-DEATH;

NEUTROPHIL ACCUMULATION; CEREBRAL-ISCHEMIA; GLOBAL-ISCHEMIA;

NITRIC-OXIDE

AB Traumatic brain injury (TBI) is one of the most devastating injuries experienced by military personnel, as well as the general population, and can result in acute and chronic complications such as cognitive impairments. Since there are currently no effective tools for the treatment of TBI, it is of great importance to determine the mechanisms of neuronal death that characterize this insult. Several studies have indicated that TBI-induced neuronal death arises in part due to excessive activation of poly(ADP-ribose) polymerase-1 (PARP-1), which results in nicotinamide adenine dinucleotide (NAD(+)) depletion and subsequent energy failure. In this study, we investigated whether intranasal administration of NAD(+) could reduce neuronal death after TBI. Rats were subjected to a weight-drop TBI model that induces cortical and hippocampal neuronal death. The intranasal administration of NAD(+) (20 mg/kg) immediately after TBI protected neurons in CA1, CA3, and dentate gyrus of the hippocampus, but not in the cortex. In addition, delayed microglial activation normally seen after TBI was reduced by NAD(+) treatment at 7 days after insult. Neuronal superoxide production and PARP-1 accumulation after TBI were not inhibited by NAD+ treatment, indicating that reactive oxygen species (ROS) production and PARP-1 activation are events that occur upstream of NAD(+) depletion. This study suggests that intranasal delivery of NAD(+) represents a novel, inexpensive, and non-toxic intervention for preventing TBI-induced neuronal death.

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TI Attenuation of Microglial Activation with Minocycline Is Not Associated

with Changes in Neurogenesis after Focal Traumatic Brain Injury in Adult

Mice

SO JOURNAL OF NEUROTRAUMA

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DT Article

DE adult neurogenesis; closed head injury; microglia; minocycline;

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ID SPINAL-CORD-INJURY; CEREBRAL-ARTERY OCCLUSION; CENTRAL-NERVOUS-SYSTEM;

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INTRACEREBRAL HEMORRHAGE; CELLULAR PROLIFERATION; CORTICAL NEUROGENESIS;

GLIAL PROLIFERATION

AB Neurogenesis is stimulated following injury to the adult brain and could potentially contribute to tissue repair. However, evidence suggests that microglia activated in response to injury are detrimental to the survival of new neurons, thus limiting the neurogenic response. The aim of this study was to determine the effect of the anti-inflammatory drug minocycline on neurogenesis and functional recovery after a closed head injury model of focal traumatic brain injury (TBI). Beginning 30 min after trauma, minocycline was administered for up to 2 weeks and bromodeoxyuridine was given on days 1-4 to label proliferating cells. Neurological outcome and motor function were evaluated over 6 weeks using the Neurological Severity Score (NSS) and ledged beam task. Microglial activation was assessed in the pericontusional cortex and hippocampus at 1 week post-trauma, using immunohistochemistry to detect F4/80. Following immunolabeling of bromodeoxyuridine, double-cortin, and NeuN, cells undergoing distinct stages of neurogenesis, including proliferation, neuronal differentiation, neuroblast migration, and long-term survival, were quantified at 1 and 6 weeks in the hippocampal dentate gyrus, as well as in the subventricular zone of the lateral ventricles and the pericontusional cortex. Our results show that minocycline successfully reduced microglial activation and promoted early neurological recovery that was sustained over 6 weeks. We also show for the first time in the closed head injury model, that early stages of neurogenesis were stimulated in the hippocampus and subventricular zone; however, no increase in new mature neurons occurred. Contrary to our hypothesis, despite the attenuation of activated microglia, minocycline did not support neurogenesis in the hippocampus, lateral ventricles, or pericontusional cortex, with none of the neurogenic stages being affected by treatment. These data provide evidence that a general suppression of microglial activation is insufficient to enhance neuronal production, suggesting that further work is required to elucidate the relationship between microglia and neurogenesis after TBI.

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TI Injury and repair in the neurovascular unit

SO NEUROLOGICAL RESEARCH

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DT Review

DE Neuron; Astrocyte; Pericyte; Microglia; Endothelium; Plasticity; Stroke;

Traumatic brain injury; Neurodegeneration

ID BLOOD-BRAIN-BARRIER; CEREBRAL ENDOTHELIAL-CELLS; SUBVENTRICULAR ZONE;

AMYLOID-BETA; FUNCTIONAL RECOVERY; NEUROGENESIS; STROKE; MECHANISMS;

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AB The neurovascular unit provides a conceptual framework for investigating the pathophysiology of how brain cells die after stroke, brain injury, and neurodegeneration. Emerging data now suggest that this concept can be further extended. Cell-cell signaling between neuronal, glial, and vascular elements in the brain not only mediates the mechanisms of acute injury, but integrated responses in these same elements may also be required for recovery as the entire neurovascular unit attempts to reorganize and remodel. Understanding the common signals and substrates of this transition between acute injury and delayed repair in the neurovascular unit may reveal useful paradigms for augmenting neuronal, glial, and vascular plasticity in damaged and diseased brain.

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et al., Ann New York Acad Sci. 2010; Lo, Nat Med. 2010; Moskowitz et

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AU Zhang, ZR

Zhang, ZY

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AF Zhang, Zhiren

Zhang, Zhi-Yuan

Wu, Yuzhang

Schluesener, Hermann J.

TI Lesional Accumulation of CD163<SUP>+</SUP> Macrophages/microglia in Rat

Traumatic Brain Injury

SO BRAIN RESEARCH

LA English

DT Article

DE Traumatic brain injury; CD163; Macrophages; Heme oxygenase-1

ID ANTIINFLAMMATORY MACROPHAGE PHENOTYPE; HEMOGLOBIN SCAVENGER RECEPTOR;

CENTRAL-NERVOUS-SYSTEM; HEME OXYGENASE-1 HO-1; PERIVASCULAR MACROPHAGES;

NEUROVASCULAR UNIT; HUMAN MONOCYTES; MICROGLIA; ACTIVATION; EXPRESSION

AB A robust neuroinflammation, contributing to the development of secondary injury, is a common histopathological feature of traumatic brain injury (TBI). Characterization of leukocytic subpopulations contributing to the early infiltration of the damaged tissue might aid in further understanding of lesion development. Reactive macrophages/microglia can exert protective or damaging effects in TBI. CD163 is considered a marker of M2 (alternatively activated) macrophages. Therefore we investigated the accumulation of CD163(+) macrophages/microglia in the brain of TB! rats. TBI was induced in rats using an open skull weight-drop contusion model and the accumulation of CD163(+) cells was analyzed by immunohistochemistry. In normal rat brains, CD163 was expressed by meningeal, choroid plexus and perivascular macrophages. Significant parenchymal CD163(+) cell accumulation was observed two days post TBI and continuously increased in the investigated survival time. The accumulated CD163(+) cells were mainly distributed to the lesional areas and exhibited macrophage phenotypes with amoeboid morphologic characteristics but not activated microglial phenotypes with hypertrophic morphology and thick processes. Double-labeling experiments showed that most CD163+ cells co-expressed heme oxygenase-1 (HO-1). In addition, in vitro incubating of macrophage RAW264.7 cells or primary peritoneal macrophages with hemoglobin- haptoglobin (Hb-Hp) complex suppressed LPS-induced inflammatory macrophages phenotype and induced CD163 and HO-1 upregulation, indicating that CD163(+) macrophages/microglia in TBI might have anti-inflammatory effects. And further study is necessary to identify functions of these cells in TBI. (C) 2012 Elsevier B.V. All rights reserved.

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AU Carthew, HL

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Vink, R

AF Carthew, H. L.

Ziebell, J. M.

Vink, R.

TI SUBSTANCE P-INDUCED CHANGES IN CELL GENESIS FOLLOWING DIFFUSE TRAUMATIC

BRAIN INJURY

SO NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury; substance P; neurogenesis; microglia;

hippocampus; subventricular zone

ID FOCAL CEREBRAL-ISCHEMIA; ADULT MAMMALIAN BRAIN; HIPPOCAMPAL

NEUROGENESIS; NEURAL PROGENITOR; MURINE MICROGLIA; STEM-CELLS; RATS;

PATHOPHYSIOLOGY; PROLIFERATION; INFLAMMATION

AB Inhibition of substance P (SP) activity through the use of NK1 receptor antagonists has been shown to be a promising neuroprotective therapy following traumatic brain injury (TBI). Conversely, recent research has implicated SP in the stimulation of neurogenesis, suggesting that the neuropeptide has the potential to promote recovery following TBI. This study characterised the effects of SP and the NK1 antagonist, n-acetyl tryptophan (NAT), on cell proliferation following diffuse TBI. Adult male Sprague-Dawley rats were injured using the impact acceleration model of TBI and randomly assigned to one of five treatment groups: sham, vehicle control, NAT alone, SP alone or SP with NAT. Cellular proliferation was assessed with immunostaining for bromodeoxyuridine (BrdU) and cell-specific markers. Infusion of SP (+/- NAT) promoted cellular proliferation in the subventricular zone and dentate gyrus following TBI. This increase was largely associated with microglial proliferation and did not correspond with functional improvements. These results suggest that NAT treatment results in neuro-protection following TBI, mediated in part via inhibition of microglia. (c) 2012 IBRO. Published by Elsevier Ltd. All rights reserved.

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TC 25

Z9 26

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TI Microglia activation along the corticospinal tract following traumatic

brain injury in the rat: A neuroanatomical study

SO BRAIN RESEARCH

LA English

DT Article

DE Microglia; Traumatic brain injury; Corticospinal neuron; Electron

microscopy; Light microscopy; Rat

ID CLOSED-HEAD INJURY; NERVOUS-SYSTEM; MICE; INTERLEUKIN-1-BETA; RECEPTORS;

RECOVERY; PROTEIN

AB Traumatic injury to the brain often manifests itself symptomatically and structurally long after the traumatic event. The cellular basis of this complex response is not completely understood. However, we hypothesized that microglia might contribute to the brain-wide process. To test this hypothesis, we employed optical and electron microscopy to study the microglia in rat brains up to 2 months after digitally controlled cortical impact (CCI) to produce traumatic brain injury (TBI). We also used antibodies against ED-1 and Iba-1, respectively, as markers for activated and resting microglia. ED-1 positive microglial cells are observed accompanying the entire corticospinal tract (CST) on the injured side, but not the control, contralateral side of the brain at 2 months. In this case, ED-1 and Iba-1 were observed to co-localize uniquely on the injured side of the brain. At earlier times following CCI, ultrastructural studies reveal that microglial cells have very irregular shapes and have many processes that intermingle with degenerating nerve axons of the CST in the hindbrain pyramids. These cells appear to be engulfing degenerating myelinated axons. The debris within the cells is converted to lipofuscin, the antigen for the ED-1 antibody, and remains in the cell cytoplasm throughout the life of the cell. We conclude, as hypothesized, that microglia are critical cellular components. Based on observed close association with myelin degeneration, interdigitating activated microglia may be contributing to damage control. Finally, based on the close neuroanatomical relationship between the lesioned corticospinal tract and the wide distribution of activated microglia, primary signals from CST neurons per se, may be directing microglial responses along the entire damaged rat neuroaxis. The role of persistent activation of microglia has not been determined. Published by Elsevier B.V.

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PT J

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TI The immunology of traumatic brain injury: a prime target for Alzheimer's

disease prevention

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Review

DE traumatic brain injury; flavonoids; microglia phenotype; Alzheimer's

disease; cytokines

ID SEVERE HEAD-INJURY; GINKGO-BILOBA EXTRACT; NEURONAL CELL-DEATH; GREEN

TEA-EGCG; MICROGLIAL ACTIVATION; AMYLOID-BETA; INFLAMMATORY RESPONSE;

COGNITIVE IMPAIRMENT; DIETARY FLAVONOIDS; SECONDARY INSULTS

AB A global health problem, traumatic brain injury (TBI) is especially prevalent in the current era of ongoing world military conflicts. Its pathological hallmark is one or more primary injury foci, followed by a spread to initially normal brain areas via cascades of inflammatory cytokines and chemokines resulting in an amplification of the original tissue injury by microglia and other central nervous system immune cells. In some cases this may predispose individuals to later development of Alzheimer's disease (AD). The inflammatory-based progression of TBI has been shown to be active in humans for up to 17 years post TBI. Unfortunately, all neuroprotective drug trials have failed, and specific treatments remain less than efficacious. These poor results might be explained by too much of a scientific focus on neurons without addressing the functions of microglia in the brain, which are at the center of proinflammatory cytokine generation. To address this issue, we provide a survey of the TBI-related brain immunological mechanisms that may promote progression to AD. We discuss these immune and microglia-based inflammatory mechanisms involved in the progression of post-trauma brain damage to AD. Flavonoid-based strategies to oppose the antigen-presenting cell-like inflammatory phenotype of microglia will also be reviewed. The goal is to provide a rationale for investigations of inflammatory response following TBI which may represent a pathological link to AD. In the end, a better understanding of neuroinflammation could open therapeutic avenues for abrogation of secondary cell death and behavioral symptoms that may mediate the progression of TBI to later AD.

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TI Primary Microglia Isolation from Mixed Glial Cell Cultures of Neonatal

Rat Brain Tissue

SO JOVE-JOURNAL OF VISUALIZED EXPERIMENTS

LA English

DT Article

DE Immunology; Issue 66; Neuroscience; Physiology; Molecular Biology; Cell

Culture; isolation; microglia; mixed glial cell; traumatic brain injury;

neurodegenerative disease

ID ACTIVATION; GLUTAMATE; INFLAMMATION; MECHANISMS

AB Microglia account for approximately 12% of the total cellular population in the mammalian brain. While neurons and astrocytes are considered the major cell types of the nervous system, microglia play a significant role in normal brain physiology by monitoring tissue for debris and pathogens and maintaining homeostasis in the parenchyma via phagocytic activity 1,2. Microglia are activated during a number of injury and disease conditions, including neurodegenerative disease, traumatic brain injury, and nervous system infection (3). Under these activating conditions, microglia increase their phagocytic activity, undergo morpohological and proliferative change, and actively secrete reactive oxygen and nitrogen species, pro-inflammatory chemokines and cytokines, often activating a paracrine or autocrine loop (4-6). As these microglial responses contribute to disease pathogenesis in neurological conditions, research focused on microglia is warranted.

Due to the cellular heterogeneity of the brain, it is technically difficult to obtain sufficient microglial sample material with high purity during in vivo experiments. Current research on the neuroprotective and neurotoxic functions of microglia require a routine technical method to consistently generate pure and healthy microglia with sufficient yield for study. We present, in text and video, a protocol to isolate pure primary microglia from mixed glia cultures for a variety of downstream applications. Briefly, this technique utilizes dissociated brain tissue from neonatal rat pups to produce mixed glial cell cultures. After the mixed glial cultures reach confluency, primary microglia are mechanically isolated from the culture by a brief duration of shaking. The microglia are then plated at high purity for experimental study.

The principle and protocol of this methodology have been described in the literature (7,8). Additionally, alternate methodologies to isolate primary microglia are well described (9-12). Homogenized brain tissue may be separated by density gradient centrifugation to yield primary microglia (12). However, the centrifugation is of moderate length (45 min) and may cause cellular damage and activation, as well as, cause enriched microglia and other cellular populations. Another protocol has been utilized to isolate primary microglia in a variety of organisms by prolonged (16 hr) shaking while in culture (9-11). After shaking, the media supernatant is centrifuged to isolate microglia. This longer two-step isolation method may also perturb microglial function and activation. We chiefly utilize the following microglia isolation protocol in our laboratory for a number of reasons: (1) primary microglia simulate in vivo biology more faithfully than immortalized rodent microglia cell lines, (2) nominal mechanical disruption minimizes potential cellular dysfunction or activation, and (3) sufficient yield can be obtained without passage of the mixed glial cell cultures.

It is important to note that this protocol uses brain tissue from neonatal rat pups to isolate microglia and that using older rats to isolate microglia can significantly impact the yield, activation status, and functional properties of isolated microglia. There is evidence that aging is linked with microglia dysfunction, increased neuroinflammation and neurodegenerative pathologies, so previous studies have used ex vivo adult microglia to better understand the role of microglia in neurodegenerative diseases where aging is important parameter. However, ex vivo microglia cannot be kept in culture for prolonged periods of time. Therefore, while this protocol extends the life of primary microglia in culture, it should be noted that the microglia behave differently from adult microglia and in vitro studies should be carefully considered when translated to an in vivo setting.

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TI Progenitor Cells: Therapeutic Targets after Traumatic Brain Injury

SO TRANSLATIONAL STROKE RESEARCH

LA English

DT Review

DE Traumatic brain injury; TBI; Stem cells; Progenitor cells; Bone marrow

derived mononuclear cells; MAPC; MSC; Microglia; Inflammatory reflex;

Spleen

ID MOUSE SPINAL-CORD; INFLAMMATORY RESPONSE; ISCHEMIA-REPERFUSION;

TIME-COURSE; ACTIVATION; MICROGLIA; IDENTIFICATION; RECEPTOR; DAMAGE;

NERVE

AB Traumatic brain injuries and their associated treatments carry high cost in both financial impact and morbidity to human life. Recent studies and trials present promising results in reducing secondary injury in the days and weeks following the primary insult. A number of studies, both pre-clinical and clinical, have found that different populations of stem/progenitor cells result in a reduction of inflammation, maintenance of the blood brain barrier, and an overall improved prognosis. The mechanism of action of these cellular therapies appears to rely upon the ability of the cells to influence microglia/macrophage phenotype and alter the state of the inflammatory response. The spleen has become an area of intense interest as an arena where therapeutic cells interact with reactive macrophages to cause system-level changes in immune activity. Additionally, the spleen enacts anti-inflammatory responses originating in the CNS, delivered through vagal activity with a recently described mechanism culminating in acetylcholine release. This review provides a summary of recent findings as to the mechanisms of action observed in current cellular therapies.

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TI Intravenous multipotent adult progenitor cell therapy after traumatic

brain injury: modulation of the resident microglia population

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Multipotent adult progenitor cells; Traumatic brain injury; Stem cells;

Splenocytes; Blood brain barrier; Microglia

ID CENTRAL-NERVOUS-SYSTEM; INFLAMMATORY RESPONSE; SPINAL-CORD

AB Introduction: We have demonstrated previously that the intravenous delivery of multipotent adult progenitor cells (MAPC) after traumatic brain injury affords neuroprotection via interaction with splenocytes, leading to an increase in systemic anti-inflammatory cytokines. We hypothesize that the observed modulation of the systemic inflammatory milieu is related to T regulatory cells and a subsequent increase in the locoregional neuroprotective M2 macrophage population.

Methods: C57B6 mice were injected with intravenous MAPC 2 and 24 hours after controlled cortical impact injury. Animals were euthanized 24, 48, 72, and 120 hours after injury. In vivo, the proportion of CD4(+)/CD25(+)/FOXP3(+) T-regulatory cells were measured in the splenocyte population and plasma. In addition, the brain CD86(+) M1 and CD206(+) M2 macrophage populations were quantified. A series of in vitro co-cultures were completed to investigate the need for direct MAPC:splenocyte contact as well as the effect of MAPC therapy on M1 and M2 macrophage subtype apoptosis and proliferation.

Results: Significant increases in the splenocyte and plasma T regulatory cell populations were observed with MAPC therapy at 24 and 48 hours, respectively. In addition, MAPC therapy was associated with an increase in the brain M2/M1 macrophage ratio at 24, 48 and 120 hours after cortical injury. In vitro cultures of activated microglia with supernatant derived from MAPC:splenocyte co-cultures also demonstrated an increase in the M2/M1 ratio. The observed changes were secondary to an increase in M1 macrophage apoptosis.

Conclusions: The data show that the intravenous delivery of MAPC after cortical injury results in increases in T regulatory cells in splenocytes and plasma with a concordant increase in the locoregional M2/M1 macrophage ratio. Direct contact between the MAPC and splenocytes is required to modulate activated microglia, adding further evidence to the central role of the spleen in MAPC-mediated neuroprotection.

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TI Prevention of traumatic brain injury-induced neuronal death by

inhibition of NADPH oxidase activation

SO BRAIN RESEARCH

LA English

DT Article

DE Traumatic brain injury; Apocynin; Hippocampus; NADPH oxidase; Microglia;

Reactive oxygen species; Blood-brain barrier disruption

ID POLY(ADP-RIBOSE) POLYMERASE; NEUTROPHIL ACCUMULATION; CEREBRAL-ISCHEMIA;

OXIDATIVE STRESS; APOCYNIN; ZINC; SUPEROXIDE; RATS; HYPERTHERMIA;

REPERFUSION

AB The present study aimed to evaluate the therapeutic potential of apocynin, an NADPH oxidase assembly inhibitor, on traumatic brain injury. Rat traumatic brain injury (TBI) was performed using a weight drop model. Apocynin (100 mg/kg) was injected into the intraperitoneal space 15 min before TBI. Reactive oxygen species (ROS) in the hippocampal CA3 pyramidal neurons were detected by dihydroethidium (dHEt) at 3 h after TBI. Oxidative injury was detected by 4-hydroxy-2-nonenal (4HNE) at 6 h after TBI. Blood-brain barrier disruption was detected by IgG extravasation and neuronal death was evaluated with Fluoro Jade-B staining 24h after TBI. Microglia activation was detected by CD11b immunohistochemistry in the hippocampus at 1 week after TBI. ROS production was inhibited by apocynin administration in the hippocampal CA3 pyramidal neurons. This pre-treatment with apocynin decreased the blood-brain barrier disruption, the number of degenerating neurons in the hippocampal CA3 region and microglial activation after TBI. The present study indicates that apocynin pre-treatment prevents TBI-induced ROS production, thus decreasing BBB disruption, neuronal death and microglial activation. Therefore, the present study suggests that inhibition of NADPH oxidase by apocynin may have a high therapeutic potential to reduce traumatic brain injury-induced neuronal death. (C) 2012 Elsevier B.V. All rights reserved.

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AU Kumar, A

Loane, DJ

AF Kumar, Alok

Loane, David J.

TI Neuroinflammation after traumatic brain injury: Opportunities for

therapeutic intervention

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Review

DE Traumatic brain injury; Neuroinflammation; Neuroprotection; Microglia;

Chronic neurodegeneration

ID TUMOR-NECROSIS-FACTOR; CLOSED-HEAD INJURY; INTRACEREBRAL INFLAMMATORY

RESPONSE; AMYLOID PROTEIN DEPOSITION; FIBRILLARY ACIDIC PROTEIN;

EXCITATORY AMINO-ACIDS; SPINAL-CORD-INJURY; MICROGLIAL ACTIVATION;

GENE-EXPRESSION; CEREBRAL-CORTEX

AB Traumatic brain injury (TBI) remains one of the leading causes of mortality and morbidity worldwide, yet despite extensive efforts to develop neuroprotective therapies for this devastating disorder there have been no successful outcomes in human clinical trials to date. Following the primary mechanical insult TBI results in delayed secondary injury events due to neurochemical, metabolic and cellular changes that account for many of the neurological deficits observed after TBI. The development of secondary injury represents a window of opportunity for therapeutic intervention to prevent progressive tissue damage and loss of function after injury. To establish effective neuroprotective treatments for TBI it is essential to fully understand the complex cellular and molecular events that contribute to secondary injury. Neuroinflammation is well established as a key secondary injury mechanism after TBI, and it has been long considered to contribute to the damage sustained following brain injury. However, experimental and clinical research indicates that neuroinflammation after TBI can have both detrimental and beneficial effects, and these likely differ in the acute and delayed phases after injury. The key to developing future anti-inflammatory based neuroprotective treatments for TBI is to minimize the detrimental and neurotoxic effects of neuroinflammation while promoting the beneficial and neurotrophic effects, thereby creating optimal conditions for regeneration and repair after injury. This review outlines how post-traumatic neuroinflammation contributes to secondary injury after TBI, and discusses the complex and varied responses of the primary innate immune cells of the brain, microglia, to injury. In addition, emerging experimental anti-inflammatory and multipotential drug treatment strategies for TBI are discussed, as well as some of the challenges faced by the research community to translate promising neuroprotective drug treatments to the clinic. (C) 2012 Elsevier Inc. All rights reserved.

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TI A cannabinoid type 2 receptor agonist attenuates blood-brain barrier

damage and neurodegeneration in a murine model of traumatic brain injury

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LA English

DT Article

DE controlled cortical impact; blood-brain barrier; cannabinoid-2 receptor;

endocannabinoid; traumatic brain injury; microglia

ID CONTROLLED CORTICAL IMPACT; CEREBRAL ISCHEMIC/REPERFUSION INJURY; FLUID

PERCUSSION INJURY; SPINAL-CORD-INJURY; CB2 RECEPTOR; ENDOCANNABINOID

SYSTEM; MICROGLIAL ACTIVATION; INFLAMMATORY RESPONSE; CELL-MIGRATION;

MOUSE MODEL

AB After traumatic brain injury (TBI), inflammation participates in both the secondary injury cascades and the repair of the CNS, both of which are influenced by the endocannabinoid system. This study determined the effects of repeated treatment with a cannabinoid type 2 receptor (CB2R) agonist on bloodbrain barrier integrity, neuronal degeneration, and behavioral outcome in mice with TBI. We also looked for the presence of a prolonged treatment effect on the macrophage/microglial response to injury. C57BL/6 mice underwent controlled cortical impact (CCI) and received repeated treatments with a CB2R agonist, 0-1966, or vehicle. After euthanasia at 6 hr or 1, 2, 3, or 7 days postinjury, brains were removed for histochemical analysis. Bloodbrain barrier permeability changes were evaluated by using sodium fluorescein (NaF). Perilesional degenerating neurons, injury volumes, and macrophage/microglia cells were quantified by stereological methods. Rota-rod and open-field testing were performed to evaluate motor function and natural exploratory behavior in mice. 0-1966 Treatment resulted in a significant reduction in NaF uptake and number of degenerating neurons compared with the vehicle-treated group. 0-1966-Treated mice demonstrated improvement on rota-rod and open-field testing compared with vehicle-treated mice. These changes in CCI mice treated with 0-1966 were associated with a prolonged reduction in macrophage/microglia cell counts. In conclusion, repeated treatments with a CB2R agonist, 0-1966, result in attenuated bloodbrain barrier disruption and neuronal degeneration. In addition, repeated treatment with 0-1966 shows prolonged treatment effects on behavior and the macrophage/microglia cell response over several days. (c) 2012 Wiley Periodicals, Inc.

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TI MORPHOLOGICAL AND GENETIC ACTIVATION OF MICROGLIA AFTER DIFFUSE

TRAUMATIC BRAIN INJURY IN THE RAT

SO NEUROSCIENCE

LA English

DT Article

DE classical activation; alternate activation; anti-inflammatory;

neuroplasticity

ID CLOSED-HEAD INJURY; NEURONAL INJURY; NEUTROPHIL INFILTRATION;

ALTERNATIVE ACTIVATION; AXON REGENERATION; MYELOID CELLS; SPINAL-CORD;

LATE-ONSET; EXPRESSION; MACROPHAGES

AB Traumatic brain injury (TBI) survivors experience long-term post-traumatic morbidities. In diffuse brain-injured rats, a chronic sensory sensitivity to whisker stimulation models the agitation of TBI survivors and provides anatomical landmarks across the whisker-barrel circuit to evaluate post-traumatic neuropathology. As a consequence of TBI, acute and chronic microglial activation can contribute to degenerative and reparative events underlying post-traumatic morbidity. Here we hypothesize that a temporal sequence of microglial activation states contributes to the circuit pathology responsible for post-traumatic morbidity, and test the hypothesis by examining microglial morphological activation and neuroinflammatory markers for activation states through gene expression and receptor-binding affinity. Adult male, Sprague-Dawley rats were subjected to a single moderate midline fluid percussion injury (FPI) or sham injury. Microglial activation was determined by immunohistochemistry, quantitative real-time PCR and receptor autoradiography in the primary somatosensory barrel field (S1BF) and ventral posterior medial nucleus (VPM) of the thalamus at 7 and 28 days following FPI. Morphological changes indicative of microglial activation, including swollen cell body with thicker, shrunken processes, were evident in S1BF and VPM at 7 and 28 days post-injury. Principally at 7 days post-injury in VPM, general inflammatory gene expression (major histocompatibility complex I, major histocompatibility complex II, translocator protein 18 kDa [TSPO]) is increased above sham level and TSPO gene expression confirmed by receptor autoradiography. Further, CD45, a marker of classical activation, and TGF-beta I, an acquired deactivation marker, were elevated significantly above sham at 7 days post-injury. Daily administration of the anti-inflammatory ibuprofen (20 mg/kg, i.p.) significantly reduced the expression of these genes. Evidence for alternative activation (arginase 1) was not observed. Thus, these data demonstrate concomitant classical activation and acquired deactivation phenotypes of microglia in diffuse TBI in the absence of overt contusion or cavitation. Antiinflammatory treatment may further alleviate the neuropathological burden of post-traumatic inflammation. (C) 2012 IBRO. Published by Elsevier Ltd. All rights reserved.

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TI Inflammation and white matter degeneration persist for years after a

single traumatic brain injury

SO BRAIN

LA English

DT Article

DE inflammation; diffuse axonal injury; traumatic brain injury; axonal

pathology; microglia

ID AMYLOID PRECURSOR PROTEIN; DIFFUSE AXONAL INJURY; LONG-TERM

ACCUMULATION; HEAD-INJURY; ALZHEIMERS-DISEASE; MICROGLIAL ACTIVATION;

RISK-FACTOR; BETA ACCUMULATION; NEUROINFLAMMATION; DIAGNOSIS

AB A single traumatic brain injury is associated with an increased risk of dementia and, in a proportion of patients surviving a year or more from injury, the development of hallmark Alzheimer's disease-like pathologies. However, the pathological processes linking traumatic brain injury and neurodegenerative disease remain poorly understood. Growing evidence supports a role for neuroinflammation in the development of Alzheimer's disease. In contrast, little is known about the neuroinflammatory response to brain injury and, in particular, its temporal dynamics and any potential role in neurodegeneration. Cases of traumatic brain injury with survivals ranging from 10 h to 47 years post injury (n = 52) and age-matched, uninjured control subjects (n = 44) were selected from the Glasgow Traumatic Brain Injury archive. From these, sections of the corpus callosum and adjacent parasaggital cortex were examined for microglial density and morphology, and for indices of white matter pathology and integrity. With survival of epsilon 3 months from injury, cases with traumatic brain injury frequently displayed extensive, densely packed, reactive microglia (CR3/43- and/or CD68-immunoreactive), a pathology not seen in control subjects or acutely injured cases. Of particular note, these reactive microglia were present in 28% of cases with survival of > 1 year and up to 18 years post-trauma. In cases displaying this inflammatory pathology, evidence of ongoing white matter degradation could also be observed. Moreover, there was a 25% reduction in the corpus callosum thickness with survival > 1 year post-injury. These data present striking evidence of persistent inflammation and ongoing white matter degeneration for many years after just a single traumatic brain injury in humans. Future studies to determine whether inflammation occurs in response to or, conversely, promotes white matter degeneration will be important. These findings may provide parallels for studying neurodegenerative disease, with traumatic brain injury patients serving as a model for longitudinal investigations, in particular with a view to identifying potential therapeutic interventions.

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TI Strain influences on inflammatory pathway activation, cell infiltration

and complement cascade after traumatic brain injury in the rat

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE Traumatic brain injury; Inbred rat strains; Complement; Genetic;

Neutrophils; Macrophages; Microglia; NK cells; CXCL1; Neurofilament

light

ID MAJOR HISTOCOMPATIBILITY COMPLEX; EXPERIMENTAL AUTOIMMUNE

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AB Increasing evidence suggests that genetic background affects outcome of traumatic brain injuries (TB!). Still, there is limited detailed knowledge on what pathways/processes are affected by genetic heterogeneity. The inbred rat strains DA and PVC differ in neuronal survival following TBI. We here carried out global expressional profiling to identify differentially regulated pathways governing the response to an experimental controlled brain contusion injury. One of the most differentially regulated molecular networks concerned immune cell trafficking. Subsequent characterization of the involved cells using flow cytometry demonstrated greater infiltration of neutrophils and monocytes, as well as a higher degree of microglia activation in DA compared to PVG rats. In addition, DA rats displayed a higher number of NK cells and a higher ratio of CD161bright compared to CD161dim NK cells. Local expression of complement pathway molecules such as Cl and C3 was higher in DA and both the key complement component C3 and membrane-attack complex (MAC) could be demonstrated on axons and nerve cells. A stronger activation of the complement system in DA was associated with higher cerebrospinal fluid levels of neurofilament-light, a biomarker for nerve/axonal injury. In summary, we demonstrate substantial differences between DA and PVC rats in activation of inflammatory pathways; in particular, immune cell influx and complement activation associated with neuronal/axonal injury after TBI. These findings suggest genetic influences acting on inflammatory activation to be of importance in TBI and motivate further efforts using experimental forward genetics to identify genes/pathways that affect outcome. (C) 2012 Elsevier Inc. All rights reserved.

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TI Microglia activation as a biomarker for traumatic brain injury

SO FRONTIERS IN NEUROLOGY

LA English

DT Review

DE head trauma; microglia; inflammatory response; secondary cell death;

anti-inflammatory therapy; brain imaging

ID NERVE GROWTH-FACTOR; SPINAL-CORD-INJURY; GENE-EXPRESSION; TNF-ALPHA; RAT

MODEL; CELL-DEATH; S-NITROSOGLUTATHIONE; CEREBRAL-CORTEX; RHESUS-MONKEY;

UP-REGULATION

AB Traumatic brain injury (TBI) has become the signature wound of wars in Afghanistan and Iraq. Injury may result from a mechanical force, a rapid acceleration-deceleration movement, or a blast wave. A cascade of secondary cell death events ensues after the initial injury. In particular, multiple inflammatory responses accompany TBI. A series of inflammatory cytokines and chemokines spreads to normal brain areas juxtaposed to the core impacted tissue. Among the repertoire of immune cells involved, microglia is a key player in propagating inflammation to tissues neighboring the core site of injury. Neuroprotective drug trials in TBI have failed, likely due to their sole focus on abrogating neuronal cell death and ignoring the microglia response despite these inflammatory cells' detrimental effects on the brain. Another relevant point to consider is the veracity of results of animal experiments due to deficiencies in experimental design, such as incomplete or inadequate method description, data misinterpretation, and reporting may introduce bias and give false-positive results. Thus, scientific publications should follow strict guidelines that include randomization, blinding, sample-size estimation, and accurate handling of all data (Lando et al., 2012). A prolonged state of inflammation after brain injury may linger for years and predispose patients to develop other neurological disorders, such as Alzheimer's disease.TBI patients display progressive and long-lasting impairments in their physical, cognitive, behavioral, and social performance. Here, we discuss inflammatory mechanisms that accompany TBI in an effort to increase our understanding of the dynamic pathological condition as the disease evolves over time and begin to translate these findings for defining new and existing inflammation-based biomarkers and treatments for TBI.

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AF Gatson, Joshua W.

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Minei, Joseph P.

TI Resveratrol decreases inflammation in the brain of mice with mild

traumatic brain injury

SO JOURNAL OF TRAUMA AND ACUTE CARE SURGERY

LA English

DT Article

DE TBI; Resveratrol; microglia; IL-6; IL-12; mice

ID COGNITIVE DEFICITS; LIPID-PEROXIDATION; AXONAL INJURY; ADULT-RAT;

NEUROGENESIS; ACTIVATION; CELLS; MODEL

AB BACKGROUND: Following a mild traumatic brain injury (TBI) event, the secondary brain injury that persists after the initial blow to the head consists of excitotoxicity, decreased cerebral glucose levels, oxidant injury, mitochondrial dysfunction, inflammation, and neuronal cell death. To date, there are no effective interventions used at decreasing secondary brain injury after mild TBI.

METHODS: In this study, male mice were treated with either placebo or resveratrol (100 mg/kg) at 5 minutes and 12 hours after mild TBI. The mice were injured using the controlled cortical impact device. In this closed-head model, a midline incision was made to access the skull and the impactor tip was aligned on the sagittal suture midway between the bregma and lambda sutures. The mice were injured at a depth of 2.0 mm, velocity of 4 m/s, and a delay time of 100 milliseconds. At 72 hours following injury, the animals were intracardially perfused with 0.9% saline followed by 10% phosphate-buffered formalin. The whole brain was removed, sliced, and stained for microglial activation (Iba1). In addition, using the enzyme-linked immunosorbent assay, tissue levels of interleukin 6 (IL-6) and IL-12 were measured in the cerebral cortex and hippocampus.

RESULTS: In this study, we found that in the placebo treatment group, there was a significant increase in Iba1 staining in the brain. The levels of microglial activation was reduced by resveratrol in the cerebral cortex (p < 0.001), corpus callosum (p < 0.001), and dentate gyrus (p < 0.005) brain regions after mild TBI. In addition to Iba1, resveratrol decreased the brain levels of IL-6 (p < 0.0001) and IL-12 (p < 0.004), which were observed in the hippocampus of the placebo group. In our model, no increase of IL-6 or IL-12 was observed in the cerebral cortex following TBI.

CONCLUSION: Resveratrol given acutely after TBI results in a decrease in neuroinflammation. These results suggest that resveratrol may be beneficial in reducing secondary brain injury after experiencing a mild TBI. (J Trauma Acute Care Surg. 2013; 74: 470-475. Copyright (C) 2013 by Lippincott Williams & Wilkins)

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TI Review: The long-term consequences of microglial activation following

acute traumatic brain injury

SO NEUROPATHOLOGY AND APPLIED NEUROBIOLOGY

LA English

DT Review

DE chronic traumatic encephalopathy; microglia; neuroinflammation;

traumatic brain injury

ID INTRACEREBRAL INFLAMMATORY RESPONSE; GLIAL-NEURONAL INTERACTIONS;

AMYLOID PROTEIN DEPOSITION; REPETITIVE HEAD-INJURY; ACTIVE AMATEUR

BOXERS; ALZHEIMERS-DISEASE; DEMENTIA-PUGILISTICA; PRECURSOR PROTEIN;

RISK-FACTORS; ENCEPHALOPATHY

AB The brain is vulnerable to a number of acute insults, with traumatic brain injury being among the commonest. Neuroinflammation is a common response to acute injury and microglial activation is a key component of the inflammatory response. In the acute and subacute phase it is likely that this response is protective and forms an important part of the normal tissue reaction. However, there is considerable literature demonstrating an association between acute traumatic brain injury to the brain and subsequent cognitive decline. This article will review the epidemiological literature relating to both single and repetitive head injury. It will focus on the neuropathological features associated with long-term complications of a single blunt force head injury, repetitive head injury and blast head injury, with particular reference to chronic traumatic encephalopathy, including dementia pugilistica. Neuroinflammation has been postulated as a key mechanism linking acute traumatic brain injury with subsequent neurodegenerative disease, and this review will consider the response to injury in the acute phase and how this may be detrimental in the longer term, and discuss potential genetic factors which may influence this cellular response. Finally, this article will consider future directions for research and potential future therapies.

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TI EXACERBATED INFLAMMATORY RESPONSES RELATED TO ACTIVATED MICROGLIA AFTER

TRAUMATIC BRAIN INJURY IN PROGRANULIN-DEFICIENT MICE

SO NEUROSCIENCE

LA English

DT Article

DE progranulin; traumatic brain injury; neuroinflammation; microglia; CD68;

TGF beta 1

ID AMYOTROPHIC-LATERAL-SCLEROSIS; GRANULIN-EPITHELIN PRECURSOR;

GROWTH-FACTOR-BETA; FRONTOTEMPORAL LOBAR DEGENERATION; OLIGOMERIC MATRIX

PROTEIN; SPINAL-CORD-INJURY; ALZHEIMERS-DISEASE; NEURODEGENERATIVE

DISEASE; GENETIC-VARIABILITY; SURFACE EXPRESSION

AB Progranulin (PGRN), a multifunctional growth factor, appears to play a role in neurodegenerative diseases accompanied by neuroinflammation. In this study, we investigated the role of PGRN in neuroinflammation, especially in the activation of microglia, by means of experimental traumatic brain injury (TBI) in the cerebral cortex of mice. The expression of GRN mRNA was increased in association with neuroinflammation after TBI. Double-immunohistochemical study showed that PGRN-immunoreactive (-IR) cells were mainly overlapped with CD68-IR cells, suggesting that the main source of PGRN was CD68-positive activated microglia. To investigate the role of PGRN in inflammatory responses related to activated microglia, we compared the immunoreactivity and expression of ionized calcium-binding adaptor molecule 1 (Iba1), CD68, and CD11b as markers for activated microglia between wild-type (WT) and GRN-deficient (KO) mice. The number of Iba1- and CD11b-IR cells and gene expression of Iba1 and CD11b were not significantly different between WT and KO mice, while the number of CD68-IR cells and CD68 expression in KO mice were significantly greater than those in WT mice. Double-immunohistochemical study showed that CD68-IR microglia were also IR for TGF beta 1, and TGF beta 1 expression and Smad3 phosphorylation in KO mice were elevated compared to WT mice. Moreover, double-immunostaining between phospho-Smad3 and glial fibrillary acidic protein suggested increased TGF beta 1-Smad3 signal mainly by astrocytes. The levels of protein carbonyl groups, which reflect protein oxidation, and laminin immunoreactivity, which is associated with angiogenesis, were also significantly increased in KO mice compared to WT mice. These results suggest that PGRN is produced in CD68-positive microglia and suppresses excessive inflammatory responses related to activated microglia after TBI in mice. (c) 2012 IBRO. Published by Elsevier Ltd. All rights reserved.

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TI DISTRIBUTION OF MEMBRANE PROGESTERONE RECEPTOR ALPHA IN THE MALE MOUSE

AND RAT BRAIN AND ITS REGULATION AFTER TRAUMATIC BRAIN INJURY

SO NEUROSCIENCE

LA English

DT Article

DE membrane progesterone receptor (mPR); neurons; astrocytes;

oligodendrocytes; microglia; traumatic brain injury (TBI)

ID BINDING-PROTEIN 25-DX; NONCLASSICAL MECHANISMS; NEUROPROTECTIVE FACTOR;

PLASMA-MEMBRANE; SEXUAL-BEHAVIOR; EXPRESSION; NEUROSTEROIDS;

HYPOTHALAMUS; ESTRADIOL; HORMONE

AB Progesterone has been shown to exert pleiotropic actions in the brain of both male and females. In particular, after traumatic brain injury (TBI), progesterone has important neuroprotective effects. In addition to intracellular progesterone receptors, membrane receptors of the hormone such as membrane progesterone receptor (mPR) may also be involved in neuroprotection. Three mPR subtypes (mPR alpha, mPR beta and mPR gamma) have been described and mPR alpha is best characterized pharmacologically. In the present study we investigated the distribution, cellular localization and the regulation of mPR alpha in male mouse and rat brain. We showed by reverse transcription-PCR that mPR alpha is expressed at similar levels in the male and female mouse brain suggesting that its expression may not be influenced by steroid levels. Treatment of males by estradiol or progesterone did not modify the level of expression of mPR alpha as shown by Western blot analysis. In situ hybridization and immunohistochemistry analysis showed a wide expression of mPR alpha in particular in the olfactory bulb, striatum, cortex, thalamus, hypothalamus, septum, hippocampus and cerebellum. Double immunofluorescence and confocal microscopy analysis showed that mPR alpha is expressed by neurons but not by oligodendrocytes and astrocytes. In the rat brain, the distribution of mPR alpha was similar to that observed in mouse brain; and after TBI, mPR alpha expression was induced in oligodendrocytes, astrocytes and reactive microglia. The wide neuroanatomical distribution of mPR alpha suggests that this receptor may play a role beyond neuroendocrine and reproductive functions. However, in the absence of injury its role might be restricted to neurons. The induction of mPR alpha after TBI in microglia, astrocytes and oligodendrocytes, points to a potential role in mediating the modulatory effects of progesterone in inflammation, ion and water homeostasis and myelin repair in the injured brain. (c) 2012 IBRO. Published by Elsevier Ltd. All rights reserved.

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TI Activation of mGluR5 and Inhibition of NADPH Oxidase Improves Functional

Recovery after Traumatic Brain Injury

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE functional recovery; microglia; metabotropic glutamate receptor 5; NADPH

oxidase; neuroprotection; traumatic brain injury

ID METABOTROPIC GLUTAMATE RECEPTORS; CELL-DEATH; NEURONAL APOPTOSIS;

OXIDATIVE STRESS; MICROGLIA; LIPOPOLYSACCHARIDE; NEUROTOXICITY;

EXPRESSION; NEUROINFLAMMATION; NEUROPROTECTION

AB Traumatic brain injury (TBI) induces microglial activation, which can contribute to secondary tissue loss. Activation of mGluR5 reduces microglial activation and inhibits microglial-mediated neurodegeneration in vitro, and is neuroprotective in experimental models of CNS injury. In vitro studies also suggest that the beneficial effects of mGluR5 activation involve nicotinamide adenine dinucleotide phosphate (NADPH) oxidase inhibition in activated microglia. We hypothesized that activation of mGluR5 by the selective agonist CHPG after TBI in mice is neuroprotective and that its therapeutic actions are mediated by NADPH oxidase inhibition. Vehicle, CHPG, or CHPG plus the mGluR5 antagonist (MPEP), were administered centrally, 30 minutes post-TBI, and functional recovery and lesion volume was assessed. CHPG significantly attenuated post-traumatic sensorimotor and cognitive deficits, and reduced lesion volumes; these effects were blocked by MPEP, thereby indicating neuroprotection involved selective activation of mGluR5. CHPG treatment also reduced NF kappa B activity and nitrite production in lipopolysaccharide-stimulated microglia and the protective effects of CHPG treatment were abrogated in NADPH oxidase deficient microglial cultures (gp91(phox-/-)). To address whether the neuroprotective effects of CHPG are mediated via the inhibition of NADPH oxidase, we administered the NADPH oxidase inhibitor apocynin with or without CHPG treatment after TBI. Both apocynin or CHPG treatment alone improved sensorimotor deficits and reduced lesion volumes when compared with vehicle-treated mice; however, the combined CHPG + apocynin treatment was not superior to CHPG alone. These data suggest that the neuroprotective effects of activating mGluR5 receptors after TBI are mediated, in part, via the inhibition of NADPH oxidase.

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TI Etanercept attenuates traumatic brain injury in rats by reducing early

microglial expression of tumor necrosis factor-α

SO BMC NEUROSCIENCE

LA English

DT Article

DE Traumatic brain injury; Microglia; Tumor necrosis factor-alpha;

Astrocyte; Neuron

ID CLOSED-HEAD INJURY; CEREBROSPINAL-FLUID; TNF-ALPHA; CYTOKINE EXPRESSION;

NEUROTROPHIC FACTOR; INTERLEUKIN-6; PLASMA; DEFICIENT; ELEVATION;

ISCHEMIA

AB Background: Tumor necrosis factor-alpha (TNF-alpha) is elevated early in injured brain after traumatic brain injury (TBI), in humans and in animals. Etanercept (a TNF-alpha antagonist with anti-inflammatory effects) attenuates TBI in rats by reducing both microglial and astrocytic activation and increased serum levels of TNF-alpha. However, it is not known whether etanercept improves outcomes of TBI by attenuating microglia-associated, astrocytes-associated, and/or neurons-associated TNF-alpha expression in ischemic brain. A well clinically relevant rat model, where a lateral fluid percussion is combined with systemic administration of etanercept immediately after TBI, was used. The neurological severity score and motor function was measured on all rats preinjury and on day 3 after etanercept administration. At the same time, the neuronal and glial production of TNF-alpha was measured by Immunofluorescence staining. In addition, TNF alpha contents of ischemic cerebral homogenates was measured using commercial enzyme-linked immunosorbent assay kits.

Results: In addition to inducing brain ischemia as well as neurological and motor deficits, TBI caused significantly higher numbers of microglia-TNF-alpha double positive cells, but not neurons-TNF-alpha or astrocytes-TNF-alpha double positive cells in the injured brain areas than did the sham operated controls, when evaluated 3 days after TBI. The TBI-induced cerebral ischemia, neurological motor deficits, and increased numbers of microglia-TNF-alpha double positive cells and increased TNF-alpha levels in the injured brain were all significantly attenuated by etanercept therapy.

Conclusion: This finding indicates that early microglia overproduction of TNF-alpha in the injured brain region after TBI contributes to cerebral ischemia and neurological motor deficits, which can be attenuated by etanercept therapy. Studies in this model could provide insight into the mechanisms underlying neurological motor disturbance in brain-injured patients.

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TI Post-injury conditioning with lipopolysaccharide or lipooligosaccharide

reduces inflammation in the brain

SO JOURNAL OF NEUROIMMUNOLOGY

LA English

DT Article

DE Traumatic brain injury; Inflammation; Neuroprotection; LPS; LOS;

Microglia

ID NERVOUS-SYSTEM; INTERFERON-GAMMA; BARRIER PERMEABILITY; ADULT

NEUROGENESIS; INJURED BRAIN; INTERLEUKIN-1; CYTOKINES; CELLS; RAT;

ACTIVATION

AB Background: Traumatic brain injury (TBI) is a leading cause of mortality and disability in the Western world. The first stage of TBI results from the mechanical damage from an impact or blast. A second stage occurs as an inflammatory response to the primary injury and presents an opportunity for clinical intervention. In this study, we investigated the effect of pre- and post-injury treatment with lipopolysaccharide (LPS) from Escherichia coli and lipooligosaccharide (LOS) from Neisseria meningitidis on levels of cerebral inflammatory cells, circulating blood cells, and pro- and anti-inflammatory cytokine levels in a rat model of neuroinflammation induced by intrastriatal injection of IL-1 beta to mimic the second stage of TBI.

Methods: LPS or LOS was administered intravenously (IV) or intranasally (IN) 2 h pre- or post-injection of IL-1 beta. The rats were euthanized 12 h following IL-1 beta injection. Brain sections were immunostained with antibody to ED-1, a microglia cell marker. Cells in whole blood were assessed with a VetScan HM2 analyzer, and cytokine levels in sera were analyzed with a Bio-Plex system.

Results: Pre- and post-injury IV administration of LPS or LOS significantly reduced microglia in the brain, and IN pre-treatment with LPS or LOS showed a statistical trend towards reducing microglia. Pre- and post-treatment IV with LOS increased circulating levels of IL-2 and IL-4, whereas IN post-treatment with LPS reduced levels of the inflammatory cytokines, TNF-alpha and IFN-gamma.

Conclusions: The findings strongly support continued investigation of post-conditioning with LPS or LOS as potential neuroprotective treatments for neuroinflammation from TBI. Published by Elsevier B.V.

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TI The p38α MAPK Regulates Microglial Responsiveness to Diffuse Traumatic

Brain Injury

SO JOURNAL OF NEUROSCIENCE

LA English

DT Article

ID PROINFLAMMATORY CYTOKINE PRODUCTION; ATTENUATES SYNAPTIC DYSFUNCTION;

PROTEIN-KINASE INHIBITOR; MACROSIALIN MOUSE CD68; UP-REGULATION; NEURON

DEGENERATION; AXONAL INJURY; IN-VIVO; ACTIVATION; RECEPTOR

AB Neuropathology after traumatic brain injury (TBI) is the result of both the immediate impact injury and secondary injury mechanisms. Unresolved post-traumatic glial activation is a secondary injury mechanism that contributes to a chronic state of neuroinflammation in both animal models of TBI and human head injury patients. We recently demonstrated, using in vitro models, that p38 alpha MAPK signaling in microglia is a key event in promoting cytokine production in response to diverse disease-relevant stressors and subsequent inflammatory neuronal dysfunction. From these findings, we hypothesized that the p38 alpha signaling pathway in microglia could be contributing to the secondary neuropathologic sequelae after a diffuse TBI. Mice where microglia were p38 alpha-deficient (p38 alpha KO) were protected against TBI-induced motor deficits and synaptic protein loss. In wild-type (WT) mice, diffuse TBI produced microglia morphological activation that lasted for at least 7 d; however, p38 alpha KO mice failed to activate this response. Unexpectedly, we found that the peak of the early, acute phase cytokine and chemokine levels was increased in injured p38 alpha KO mice compared with injured WT mice. The increased cytokine levels in the p38 alpha KO mice could not be accounted for by more infiltration of macrophages or neutrophils, or increased astrogliosis. By 7 d after injury, the cytokine and chemokine levels remained elevated in injured WT mice but not in p38 alpha KO mice. Together, these data suggest that p38 alpha balances the inflammatory response by acutely attenuating the early proinflammatory cytokine surge while perpetuating the chronic microglia activation after TBI.

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TI Lesional accumulation of heme oxygenase-1 <SUP>+</SUP>

microglia/macrophages in rat traumatic brain injury

SO NEUROREPORT

LA English

DT Article

DE heme oxygenase-1; macrophages; microglia; traumatic brain injury

ID EXPRESSION; MACROPHAGES/MICROGLIA; INFLAMMATION; INDUCTION; SYSTEM; HO-1

AB Heme oxygenase-1 (HO-1) is an inducible rate-limiting enzyme for heme degradation. Here, we studied the HO-1 expression in an open-skull weight-drop-induced traumatic brain injury, with a focus on the early phase, most amenable to therapy. In normal rat brains of our study, HO-1 (+) cells were rarely observed. Significant parenchymal accumulation of HO-1 (+) non-neuron cells was observed 18 h post-traumatic brain injury and increased continuously during the investigating time. We also observed that the accumulated HO-1 (+) non-neuron cells were mainly distributed in the perilesional areas and showed activated microglia/macrophage phenotypes with ramified or amoeboid morphologic characteristics. Further double-labeling experiments showed that most HO-1 (+) non-neuron cells coexpressed CD68 and CD163, but not glial fibrillary acid protein. Our data suggest that HO-1 expression defines a subtype of activated microglia/macrophages involved in the early processes following traumatic brain injury. NeuroReport 24:281-286 (C) 2013 Wolters Kluwer Health vertical bar Lippincott Williams & Wilkins.

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TI Traumatic brain injury in aged animals increases lesion size and

chronically alters microglial/macrophage classical and alternative

activation states

SO NEUROBIOLOGY OF AGING

LA English

DT Article

DE Microglia/macrophage; Alternative activation; Traumatic brain injury;

Aging; NADPH oxidase; Neurodegeneration; Ym1

ID NADPH OXIDASE; OXIDATIVE STRESS; MICROGLIAL ACTIVATION; INFLAMMATORY

RESPONSE; COGNITIVE DEFICITS; SYNAPTIC PROTEINS; MANNOSE RECEPTOR;

EXPRESSION; NEUROINFLAMMATION; HIPPOCAMPUS

AB Traumatic brain injury (TBI) causes chronic microglial activation that contributes to subsequent neurodegeneration, with clinical outcomes declining as a function of aging. Microglia/macrophages (MG/M phi) have multiple phenotypes, including a classically activated, proinflammatory (M1) state that might contribute to neurotoxicity, and an alternatively activated (M2) state that might promote repair. In this study we used gene expression, immunohistochemical, and stereological analyses to show that TBI in aged versus young mice caused larger lesions associated with an M1/M2 balance switch and increased numbers of reactive (bushy and hypertrophic) MG/M phi in the cortex, hippocampus, and thalamus. Chitinase3- like 3 (Ym1), an M2 phenotype marker, displayed heterogeneous expression after TBI with amoeboid-like Ym1-positive MG/M phi at the contusion site and ramified Ym1-positive MG/M phi at distant sites; this distribution was age-related. Aged-injured mice also showed increased MG/M phi expression of major histocompatibility complex II and NADPH oxidase, and reduced antioxidant enzyme expression which was associated with lesion size and neurodegeneration. Thus, altered relative M1/M2 activation and an nicotinamide adenine dinucleotide phosphate oxidase (NADPH oxidase)-mediated shift in redox state might contribute to worse outcomes observed in older TBI animals by creating a more proinflammatory M1 MG/M phi activation state. (C) 2013 Elsevier Inc. All rights reserved.

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TI Immediate splenectomy down-regulates the MAPK-NF-κB signaling pathway in

rat brain after severe traumatic brain injury

SO JOURNAL OF TRAUMA AND ACUTE CARE SURGERY

LA English

DT Article

DE Traumatic brain injury; splenectomy; microglia; inflammation;

MAPK-NF-kappa B

ID INFLAMMATION; ASTROCYTES; CHEMOKINES; EXPRESSION

AB BACKGROUND: The treatment of severe traumatic brain injury (TBI) remains a difficult process. One key to improving treatment efficacy is to reduce secondary brain injury. Local and systemic inflammatory responses play an important role in secondary injury after TBI, which if unchecked can lead to fatal cerebral edema. Previous studies focused mainly on local brain tissue, whereas little is known about the contribution of peripheral organs in the pathogenesis of TBI. We previously showed that immediate splenectomy decreases mortality and improves cognitive function in rats after severe TBI by inhibiting the release of proinflammatory cytokines both systematically and locally in the injured brain. In this study, we further investigated the molecular mechanisms responsible for the effect of the spleen on local brain inflammation after TBI.

METHODS: We established a severe TBI model with rats and performed splenectomy to study the effect of the spleen on mitogen-activated protein kinase (MAPK)-NF-kappa B activation in the brain tissue. The expression of p38 MAPK, extracellular regulated protein kinases (ERK), and NF-kappa B protein in the trauma region was examined by Western blotting. The neuron-like PC-12 cell line and microglia-like BV-2 cell line were used for in vitro experiments to test the effects of spleen supernatant after TBI. Cell apoptosis (annexin V/propidium iodide staining), NF-kappa B nuclear translocation (immunofluorescence microscopy), and MAPK signaling (phosphorylation of p-p38 and p-ERK) were examined.

RESULTS: We found that TBI significantly up-regulated MAPK signaling in the injured brain region, whereas immediate splenectomy suppressed MAPK activation. In vitro, the spleen supernatant from rats after TBI also resulted in increased MAPK activation and NF-kappa B nuclear translocation in microglia-like BV-2 cells, whereas the application of interleukin (IL)-1R antagonist (IL-1Ra) significantly reduced the expression of p-p38 and p-ERK as well as NF-kappa B nuclear translocation. In addition, spleen supernatant after TBI induced apoptosis in neuron-like PC-12 cells, and IL-1Ra could effectively reduce apoptosis.

CONCLUSION: Our study demonstrates that immediate splenectomy down-regulates the MAPK-NF-kappa B signaling pathway in rat brain after severe TBI. We also provide experimental evidence for the potential use of IL-1Ra to alleviate brain inflammation after TBI. (Copyright (C) 2013 by Lippincott Williams & Wilkins)

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TI Diffuse Traumatic Axonal Injury in the Optic Nerve Does Not Elicit

Retinal Ganglion Cell Loss

SO JOURNAL OF NEUROPATHOLOGY AND EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Microglia/macrophage; Retinal ganglion cell survival; Traumatic axonal

injury; Traumatic brain injury; Visual system; YFP-16 transgenic mice

ID C-JUN EXPRESSION; BRAIN-INJURY; NEURITE TRANSECTION; NEUROTROPHIC

FACTOR; NEURONAL SURVIVAL; CALCIUM-CHANNEL; STRETCH-INJURY; MOUSE MODEL;

TIME-COURSE; ADULT-RAT

AB Much of the morbidity after traumatic brain injury (TBI) is associated with traumatic axonal injury (TAI). Although most TAI studies focus on corpus callosum white matter, the visual system has received increased interest. To assess visual system TAI, we developed a mouse model of optic nerve TAI. It is unknown, however, whether this TAI causes retinal ganglion cell (RGC) death. To address this issue, YFP (yellow fluorescent protein)-16 transgenic mice were subjected to mild TBI and followed from 2 to 28 days. Neither TUNEL (terminal deoxynucleotidyl transferase dUTP nick end labeling)-positive or cleaved caspase-3Yimmunoreactive RGCs were observed from 2 to 28 days after TBI. Quantification of immunoreactivity of Brn3a, an RGC marker, demonstrated no RGC loss; parallel electron microscopic analysis confirmed RGC viability. Persistent RGC survival was also consistent with the finding of reorganization in the proximal axonal segments after TAI, wherein microglia/macrophages remained inactive. In contrast, activated microglia/macrophages closely enveloped the distal disconnected, degenerating axonal segments at 7 to 28 days after injury, thereby confirming that this model consistently evoked TAI followed by disconnection. Collectively, these data provide novel insight into the evolving pathobiology associated with TAI that will form a foundation for future studies exploring TAI therapy and its downstream consequences.

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TI Lacosamide Improves Outcome in a Murine Model of Traumatic Brain Injury

SO NEUROCRITICAL CARE

LA English

DT Article

DE Traumatic brain injury; Lacosamide; Neuroprotection; Inflammation;

Microglial activation

ID CLOSED-HEAD INJURY; SPINAL-CORD-INJURY; SUBARACHNOID HEMORRHAGE;

ANTICONVULSANT PROPHYLAXIS; FUNCTIONAL RECOVERY; SODIUM-CHANNELS; ONSET

SEIZURES; PHENYTOIN; PRESSURE; LEVETIRACETAM

AB Use of antiepileptic drugs (AED's) is common in the neurocritical care setting. However, there remains a great deal of controversy regarding the optimal agent. Studies associating the prophylactic use of AED's with poor outcomes are heavily biased by the prevalent use of phenytoin, an agent highly associated with deleterious effects. In the current study, we evaluate lacosamide for neuroprotective properties in a murine model of closed head injury.

Mice were subjected to moderate closed head injury using a pneumatic impactor, and then treated with either low-dose (6 mg/kg) or high-dose (30 mg/kg) lacosamide or vehicle at 30 min post-injury, and twice daily for 3 days after injury. Motor and cognitive functional assessments were performed following injury using rotarod and Morris Water Maze, respectively. Neuronal injury and microglial activation were measured by flourojade-B, NeuN, and F4/80 staining at 1 and 7 days post-injury. Timm's staining was also performed to assess lacosamide effects on mossy fiber axonal sprouting. To evaluate possible mechanisms of lacosamide effects on the inflammatory response to injury, an RNA expression array was used to evaluate for alterations in differential gene expression patterns in injured mice following lacosamide or vehicle treatments.

High-dose lacosamide was associated with improved functional outcome on both the rotarod and Morris Water Maze. High-dose lacosamide was also associated with a reduction of neuronal injury at 24 h post-injury. However, the reduction in neuronal loss observed early did not result in greater neuronal density at 31 days post-injury based on unbiased stereology of NeuN staining. High-dose lacosamide was also associated with a significant reduction in microglial activation at 7 days post-injury. The therapeutic effects of lacosamide are associated with a delay in injury-related changes in RNA expression of a subset of inflammatory mediator genes typically seen at 24 h post-injury.

Administration of lacosamide improves functional performance, and reduces histological evidence of acute neuronal injury and neuroinflammation in a murine model of closed head injury. Lacosamide effects appear to be mediated via a reduction or delay in the acute inflammatory response to injury. Prior clinical and animal studies have found antiepileptic treatment following injury to be detrimental, though these studies are biased by the common use of older medications such as phenytoin. Our current results as well as prior work on levetiracetam suggest the newer AED's may be beneficial in the setting of acute brain injury.

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TI Tumor necrosis factor in traumatic brain injury: effects of genetic

deletion of p55 or p75 receptor

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE apoptosis; inflammation; microglia; pathophysiology; traumatic brain

injury; tumor necrosis factor

ID CLOSED-HEAD INJURY; CONTROLLED CORTICAL IMPACT; FACTOR-ALPHA;

CELL-DEATH; TNF-ALPHA; CONTUSION VOLUME; MICE; BCL-2; ACTIVATION;

ISCHEMIA

AB The role of tumor necrosis factor (TNF) and its receptors after traumatic brain injury (TBI) remains unclear. We evaluated the effects of genetic deletion of either p55 or p75 TNF receptor on neurobehavioral outcome, histopathology, DNA damage and apoptosis-related cell death/survival gene expression (bcl-2/bax), and microglia/macrophage (M/M) activation in wild-type (WT) and knockout mice after TBI. Injured p55 (-/-) mice showed a significant attenuation while p75 (-/-) mice showed a significant worsening of sensorimotor deficits compared with WT mice over 4 weeks postinjury. At the same time point, contusion volume in p55 (-/-) mice (11.1 +/- 3.3 mm(3)) was significantly reduced compared with WT (19.7 +/- 3.4 mm(3)) and p75 (-/-) mice (20.9 +/- 3.2 mm(3)). At 4 hours postinjury, bcl-2/bax ratio mRNA expression was increased in p55 (-/-) compared with p75 (-/-) mice and was associated with reduced DNA damage terminal deoxynucleotidyl transferaseYmediated dUTP nick end labeling (TUNEL-positivity), reduced CD11b expression and increased Ym1 expression at 24 hours postinjury in p55 (-/-) compared with p75 (-/-) mice, indicative of a protective M/M response. These data suggest that TNF may exacerbate neurobehavioral deficits and tissue damage via p55 TNF receptor whose inhibition may represent a specific therapeutic target after TBI.

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JI J. Cereb. Blood Flow Metab.

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TI Autologous bone marrow mononuclear cells therapy attenuates activated

microglial/macrophage response and improves spatial learning after

traumatic brain injury

SO JOURNAL OF TRAUMA AND ACUTE CARE SURGERY

LA English

DT Article

DE Behavior (rodent); blood-brain barrier; brain trauma; microglia; rats

ID MOUSE SPINAL-CORD; TIME-COURSE; MICROGLIA; RATS; INFLAMMATION;

EXPRESSION

AB BACKGROUND: Autologous bone marrow-derived mononuclear cells (AMNCs) have shown therapeutic promise for central nervous system insults such as stroke and traumatic brain injury (TBI). We hypothesized that intravenous injection of AMNC provides neuroprotection, which leads to cognitive improvement after TBI.

METHODS: A controlled cortical impact (CCI) rodent TBI model was used to examine blood-brain barrier (BBB) permeability, neuronal and glial apoptosis, as well as cognitive behavior. Two groups of rats underwent CCI with AMNC treatment (CCI-autologous) or without AMNC treatment (CCI-alone), consisting of 2 million AMNC per kilogram body weight harvested from the tibia and intravenously injected 72 hours after injury. CCI-alone animals underwent sham harvests and received vehicle injections.

RESULTS: Ninety-six hours after injury, AMNC significantly reduced the BBB permeability in injured animals, and there was an increase in apoptosis of proinflammatory activated microglia in the ipsilateral hippocampus. At 4 weeks after injury, we observed significant improvement in probe testing of CCI-Autologous group in comparison to CCI-Alone in the Morris Water Maze paradigm.

CONCLUSION: Our data demonstrate that the intravenous injection of AMNC after TBI leads to neuroprotection by preserving early BBB integrity, increasing activated microglial apoptosis and improving cognitive function. (Copyright (C) 2013 by Lippincott Williams & Wilkins)

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J9 J TRAUMA ACUTE CARE

JI J. Trauma Acute Care Surg.

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TI Changes of the GPR17 receptor, a new target for neurorepair, in neurons

and glial cells in patients with traumatic brain injury

SO PURINERGIC SIGNALLING

LA English

DT Article

DE Activated microglia; Adult neural precursors; Human brain injury; Lesion

repair; Reactive astrocytes

ID LEUKOTRIENE; ANTAGONIST; EXPRESSION; BINDING

AB Unveiling the mechanisms participating in the damage and repair of traumatic brain injury (TBI) is fundamental to develop new therapies. The P2Y-like GPR17 receptor has recently emerged as a sensor of damage and a key actor in lesion remodeling/repair in the rodent brain, but its role in humans is totally unknown. Here, we characterized GPR17 expression in brain specimens from seven intensive care unit TBI patients undergoing neurosurgery for contusion removal and from 28 autoptic TBI cases (and 10 control subjects of matched age and gender) of two university hospitals. In both neurosurgery and autoptic samples, GPR17 expression was strong inside the contused core and progressively declined distally according to a spatio-temporal gradient. Inside and around the core, GPR17 labeled dying neurons, reactive astrocytes, and activated microglia/macrophages. In peri-contused parenchyma, GPR17 decorated oligodendrocyte precursor cells (OPCs) some of which had proliferated, indicating re-myelination attempts. In autoptic cases, GPR17 expression positively correlated with death for intracranial complications and negatively correlated with patients' post-traumatic survival. Data indicate lesion-specific sequential involvement of GPR17 in the (a) death of irreversibly damaged neurons, (b) activation of microglia/macrophages remodeling the lesion, and (c) activation/proliferation of multipotent parenchymal progenitors (both reactive astrocytes and OPCs) starting repair processes. Data validate GPR17 as a target for neurorepair and are particularly relevant to setting up new therapies for TBI patients.

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AF Bedi, Supinder S.

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TI Immunomagnetic enrichment and flow cytometric characterization of mouse

microglia

SO JOURNAL OF NEUROSCIENCE METHODS

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DT Article

DE Traumatic brain injury; Microglia; Neuroinflammation; Flow cytometry

ID SPINAL-CORD-INJURY; CENTRAL-NERVOUS-SYSTEM; CELL PERMEABILIZATION;

ACTIVATION; RESPONSES; BRAIN; IDENTIFICATION; MACROPHAGES; VIVO

AB Background: The inflammatory response after a CNS injury is regulated by microglia/macrophages. Changes in the ratio of M1 classically activated pro-inflammatory cells versus M2 alternatively activated anti-inflammatory cells reveal the direction of the immune response. These cells are routinely identified and enumerated by morphology and cell-surface markers using immunohistochemistry.

New method: We used a controlled cortical impact (CCI) mouse model for traumatic brain injury (TBI), then isolated microglia/macrophages from neural cell suspensions using magnetic beads conjugated to CD11b monoclonal antibody to obtain the entire myeloid population. Polarization states of CD11b(+)CD45(lo) microglia were evaluated by expression of M1 surface marker F-c gamma RII/III and M2 surface marker CD206.

Results: After TBI, we observed an increase in M1:M2 ratio in the ipsilateral hemisphere when compared to the contralateral side, indicating that 24h after CCI, a shift in microglia polarization occurs localized to the hemisphere of injury. Comparison with existing method(s): The major impetus for developing and refining the methods was the need to accurately quantify microglial activation states without reliance on manual morphometric counting of serial immunohistochemistry slides. Flow cytometric analysis of enriched cell suspensions provides quantitative measurement of microglial polarization states complementary to existing methods, but for entire populations of cells.

Conclusions: In summary, we used immunomagnetic beads to isolate myeloid cells from injured brain, then stained surface antigens to flow cytometrically identify and categorize microglia as either classically activated M1 or alternatively activated M2, generating a ratio of M1:M2 cells which is useful in studying attempts to reduce or redirect neuroinflammation. (C) 2013 Elsevier B.V. All rights reserved.

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TI ApoE and outcome after traumatic brain injury

SO CLINICAL LIPIDOLOGY

LA English

DT Review

DE apoE; microglia; neuroprotection; traumatic brain injury

ID DENSITY-LIPOPROTEIN RECEPTOR; APOLIPOPROTEIN-E EPSILON-4;

CENTRAL-NERVOUS-SYSTEM; MOUSE CORTICAL-NEURONS; D-ASPARTATE RECEPTOR;

CLOSED-HEAD INJURY; E-DEFICIENT MICE; ALZHEIMERS-DISEASE; INTRACEREBRAL

HEMORRHAGE; E GENOTYPE

AB There is increasing evidence to support the role of genetic influences in determining functional outcomes in subjects with traumatic brain injury. In particular, apoE polymorphism has been identified as one of the important determinants of prognosis after traumatic brain injury (TBI). It is likely that apoE plays a central role in determining the neuroinflammatory response to various neurological injuries such as stroke, subarachnoid hemorrhage and TBI by modulating the function of microglia, which are a major source of inflammatory mediators in the CNS. There is evidence to support the role of apoE mimetic peptides as a novel therapeutic strategy in preclinical models of TBI. Ultimately, continued research focused on the interplay between lipid biology and acute brain injury responses may have the potential to define new neuroprotective strategies.

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PT J

AU Lim, SW

Wang, CC

Wang, YH

Chio, CC

Niu, KC

Kuo, JR

AF Lim, Sher-Wei

Wang, Che-Chuan

Wang, Yun-Han

Chio, Chung-Ching

Niu, Ko-Chi

Kuo, Jinn-Rung

TI Microglial activation induced by traumatic brain injury is suppressed by

postinjury treatment with hyperbaric oxygen therapy

SO JOURNAL OF SURGICAL RESEARCH

LA English

DT Article

DE Traumatic brain injury; Hyperbaric oxygen therapy; Microglia; Tumor

necrosis factor-alpha; Anti-inflammation

ID LATERAL FLUID-PERCUSSION; MITOCHONDRIAL-FUNCTION; CEREBRAL OXYGENATION;

RAT MODEL; INFLAMMATION; MECHANISMS; PHYSIOLOGY; NEUROTRAUMA;

EXPRESSION; CONTUSIONS

AB Background: The mechanisms underlying the protective effects of hyperbaric oxygen (HBO) therapy on traumatic brain injury (TBI) are unclear. TBI initiates a neuroinflammatory cascade characterized by activation of microglia and increased production of proinflammatory cytokines. In this study, we attempted to ascertain whether the occurrence of neuroinflammation exhibited during TBI can be reduced by HBO.

Methods: TBI was produced by the fluid percussion technique in rats. HBO (100% O-2 at 2.0 absolute atmospheres) was then used at 1 h (HBO I) or 8 h (HBO II) after TBI. Neurobehavior was evaluated by the inclined plane test on the 72 h after TBI and then the rats were killed. The infarction area was evaluated by Triphenyltetrazolium chloride. Immunofluorescence staining was used to evaluate neuronal apoptosis (TUNEL + NeuN), microglial cell aggregation count (OX42 + DAPI), and tumor necrosis factor-alpha (TNF-alpha) expression in microglia cell (OX42 + TNF-alpha).

Results: The maximum grasp angle in the inclined plane test and cerebral infarction of the rats after TBI were significantly attenuated by HBO therapy regardless of whether the rats were treated with HBO 1 or 8 h after TBI compared with the controls. TBI-induced microglial activation, TNF-alpha expression, and neuronal apoptosis were also significantly reduced by HBO therapy.

Conclusions: Our results demonstrate that treatment of TBI during the acute phase of injury can attenuate microgliosis and proinflammatory cytokine TNF-alpha expression resulting in a neuroprotective effect. Even treating TBI with HBO after 8 h had a therapeutic effect. (C) 2013 Elsevier Inc. All rights reserved.

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AU Mayer, CL

Huber, BR

Peskind, E

AF Mayer, Cynthia L.

Huber, Bertrand R.

Peskind, Elaine

TI Traumatic Brain Injury, Neuroinflammation, and Post-Traumatic Headaches

SO HEADACHE

LA English

DT Review

DE traumatic brain injury; inflammation; glia; microglia; astrocytes;

satellite cells; blood brain barrier; cytokines; post-traumatic

headaches; inflammatory mediators

ID SATELLITE GLIAL-CELLS; CORTICAL SPREADING DEPRESSION; DIFFUSE AXONAL

INJURY; TUMOR-NECROSIS-FACTOR; NERVOUS-SYSTEM; INFLAMMATORY RESPONSE;

BARRIER DISRUPTION; SENSORY GANGLIA; IMMUNE-SYSTEM; AMYLOID-BETA

AB Concussions following head and/or neck injury are common, and although most people with mild injuries recover uneventfully, a subset of individuals develop persistent post-concussive symptoms that often include headaches. Post-traumatic headaches vary in presentation and may progress to become chronic and in some cases debilitating. Little is known about the pathogenesis of post-traumatic headaches, although shared pathophysiology with that of the brain injury is suspected. Following primary injury to brain tissues, inflammation rapidly ensues; while this inflammatory response initially provides a defensive/reparative function, it can persist beyond its beneficial effect, potentially leading to secondary injuries because of alterations in neuronal excitability, axonal integrity, central processing, and other changes. These changes may account for the neurological symptoms often observed after traumatic brain injury, including headaches. This review considers selected aspects of the inflammatory response following traumatic brain injury, with an emphasis on the role of glial cells as mediators of maladaptive post-traumatic inflammation.

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TI The neuroinflammatory response in humans after traumatic brain injury

SO NEUROPATHOLOGY AND APPLIED NEUROBIOLOGY

LA English

DT Article

DE microglia; neuroinflammation; neurotrauma; traumatic axonal injury

ID GLIAL-NEURONAL INTERACTIONS; SITU DNA FRAGMENTATION; ALZHEIMERS-DISEASE;

HEAD-INJURY; MICROGLIAL ACTIVATION; SYSTEMIC INFECTION; TDP-43

PROTEINOPATHY; WHITE-MATTER; RISK-FACTORS; INTERLEUKIN-1

AB Aims: Traumatic brain injury is a significant cause of morbidity and mortality worldwide. An epidemiological association between head injury and long-term cognitive decline has been described for many years and recent clinical studies have highlighted functional impairment within 12 months of a mild head injury. In addition chronic traumatic encephalopathy is a recently described condition in cases of repetitive head injury. There are shared mechanisms between traumatic brain injury and Alzheimer's disease, and it has been hypothesized that neuroinflammation, in the form of microglial activation, may be a mechanism underlying chronic neurodegenerative processes after traumatic brain injury. Methods: This study assessed the microglial reaction after head injury in a range of ages and survival periods, from < 24-h survival through to 47-year survival. Immunohistochemistry for reactive microglia (CD68 and CR3/43) was performed on human autopsy brain tissue and assessed ` blind' by quantitative image analysis. Head injury cases were compared with age matched controls, and within the traumatic brain injury group cases with diffuse traumatic axonal injury were compared with cases without diffuse traumatic axonal injury. Results: A major finding was a neuroinflammatory response that develops within the first week and persists for several months after traumatic brain injury, but has returned to control levels after several years. In cases with diffuse traumatic axonal injury the microglial reaction is particularly pronounced in the white matter. Conclusions: These results demonstrate that prolonged microglial activation is a feature of traumatic brain injury, but that the neuroinflammatory response returns to control levels after several years.

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TI INCREASED LYSOSOMAL BIOGENESIS IN ACTIVATED MICROGLIA AND EXACERBATED

NEURONAL DAMAGE AFTER TRAUMATIC BRAIN INJURY IN PROGRANULIN-DEFICIENT

MICE

SO NEUROSCIENCE

LA English

DT Article

DE progranulin; traumatic brain injury; microglia; lysosome

ID FRONTOTEMPORAL LOBAR DEGENERATION; HOST-DEFENSE; GRANULIN; PROTEIN;

GENE; PRECURSOR; INFLAMMATION; MODEL; NEURODEGENERATION; NEUROPROTECTION

AB Progranulin (PGRN) is known to play a role in the pathogenesis of neurodegenerative diseases. Recently, it has been demonstrated that patients with the homozygous mutation in the GRN gene present with neuronal ceroid lipofuscinosis, and there is growing evidence that PGRN is related to lysosomal function. In the present study, we investigated the possible role of PGRN in the lysosomes of activated microglia in the cerebral cortex after traumatic brain injury (TBI). We showed that the mouse GRN gene has two possible coordinated lysosomal expression and regulation (CLEAR) sequences that bind to transcription factor EB (TFEB), a master regulator of lysosomal genes. PGRN was colocalized with Lamp1, a lysosomal marker, and Lamp1-positive areas in GRN-deficient (KO) mice were significantly expanded compared with wild-type (WT) mice after TBI. Expression of all the lysosome-related genes examined in KO mice was significantly higher than that in WT mice. The number of activated microglia with TFEB localized to the nucleus was also significantly increased in KO as compared with WT mice. Since the TFEB translocation is regulated by the mammalian target of rapamycin complex 1 (mTORC1) activity in the lysosome, we compared ribosomal S6 kinase 1 (S6K1) phosphorylation that reflects mTORC1 activity. S6K1 phosphorylation in KO mice was significantly lower than that in WT mice. In addition, the number of nissl-positive and fluoro-jade B-positive cells around the injury was significantly decreased and increased, respectively, in KO as compared with WT mice. These results suggest that PGRN localized in the lysosome is involved in the activation of mTORC1, and its deficiency leads to increased TFEB nuclear translocation with a resultant increase in lysosomal biogenesis in activated microglia and exacerbated neuronal damage in the cerebral cortex after TBI. (c) 2013 IBRO. Published by Elsevier Ltd. All rights reserved.

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TI Beneficial Effects of Minocycline and Botulinum Toxin-Induced Constraint

Physical Therapy Following Experimental Traumatic Brain Injury

SO NEUROREHABILITATION AND NEURAL REPAIR

LA English

DT Article

DE controlled cortical impact; constraint-induced movement therapy (CIMT);

botox; rehabilitation; microglia; glial fibrillary acidic protein (GFAP)

ID INDUCED MOVEMENT THERAPY; MICROGLIAL ACTIVATION; ANIMAL-MODELS; STROKE;

METAANALYSIS; CHILDREN; RECOVERY; TARGETS; ADULT; LIMB

AB Background. Effective recovery from functional impairments caused by traumatic brain injury (TBI) requires appropriate rehabilitation therapy. Multiple pathways are involved in secondary injury and recovery suggesting a role for multimodal approaches. Objective. Here, we examined the efficacy of the anti-inflammatory agent minocycline and botulinum toxin (botox)-induced limb constraint with structured physical therapy, delivered alone or in combination, after a severe TBI produced by a controlled cortical impact in rats. Methods. Minocycline was administered at 25 mg/kg daily for 2 weeks beginning 1 day after TBI or sham surgery. For constraint/physical therapy, botox-type A was injected into the nonaffected forearm muscle 1 day after injury and 2 weeks of physical therapy commenced at 5 days after injury. Functional evaluations were conducted 8 weeks after injury. Results. Minocycline, either as a monotherapy or as combination treatment with botox/physical therapy significantly reduced impairments of spatial learning and memory in the water maze test, whereas botox/physical therapy reduced forelimb motor asymmetry and improved manual dexterity in the cylinder and vermicelli handling tests, A synergistic effect between the 2 treatments was observed when rats performed tasks requiring dexterity. Inflammation was attenuated in the peri-contusion cortex and hippocampus in all TBI groups receiving mono or combination therapies, though there was no significant difference in lesion size among groups. Conclusion. These data provide a rationale for incorporating anti-inflammatory treatment during rehabilitation therapy.

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TI Propofol Limits Microglial Activation after Experimental Brain Trauma

through Inhibition of Nicotinamide Adenine Dinucleotide Phosphate

Oxidase

SO ANESTHESIOLOGY

LA English

DT Article

ID NADPH OXIDASE; CELL-DEATH; NITRIC-OXIDE; PROVIDES NEUROPROTECTION;

INFLAMMATORY RESPONSES; COGNITIVE DEFICITS; PREFRONTAL CORTEX; SPATIAL

MEMORY; CDK INHIBITOR; MOUSE MODEL

AB Background: Microglial activation is implicated in delayed tissue damage after traumatic brain injury (TBI). Activation of microglia causes up-regulation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, with the release of reactive oxygen species and cytotoxicity. Propofol appears to have antiinflammatory actions. The authors evaluated the neuroprotective effects of propofol after TBI and examined in vivo and in vitro whether such actions reflected modulation of NADPH oxidase.

Methods: Adult male rats were subjected to moderate lateral fluid percussion TBI. Effect of propofol on brain microglial activation and functional recovery was assessed up to 28 days postinjury. By using primary microglial and BV2 cell cultures, the authors examined propofol modulation of lipopolysaccharide and interferon-gamma-induced microglial reactivity and neurotoxicity.

Results: Propofol improved cognitive recovery after TBI in novel object recognition test (48 +/- 6% for propofol [n = 15] vs. 30 +/- 4% for isoflurane [n = 14]; P = 0.005). The functional improvement with propofol was associated with limited microglial activation and decreased cortical lesion volume and neuronal loss. Propofol also attenuated lipopolysaccharide- and interferon-gamma-induced microglial activation in vitro, with reduced expression of inducible nitric oxide synthase, nitric oxide, tumor necrosis factor-alpha, interlukin-1 beta, reactive oxygen species, and NADPH oxidase. Microglial-induced neurotoxicity in vitro was also markedly reduced by propofol. The protective effect of propofol was attenuated when the NADPH oxidase subunit p22(phox) was knocked down by small interfering RNA. Moreover, propofol reduced the expression of p22(phox) and gp91(phox), two key components of NADPH oxidase, after TBI.

Conclusion: The neuroprotective effects of propofol after TBI appear to be mediated, in part, through the inhibition of NADPH oxidase.

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TI Intravenous Multipotent Adult Progenitor Cell Therapy Attenuates

Activated Microglial/Macrophage Response and Improves Spatial Learning

After Traumatic Brain Injury

SO STEM CELLS TRANSLATIONAL MEDICINE

LA English

DT Article

DE Adult stem cells; Neuroimmune; Rat model; Stem/progenitor cell

ID MARROW MONONUCLEAR-CELLS; MOUSE SPINAL-CORD; INFLAMMATORY RESPONSE;

TNF-ALPHA; MICROGLIA; NEUROINFLAMMATION; ASTROCYTES; EXPRESSION; DAMAGE;

RATS

AB We previously demonstrated that the intravenous delivery of multipotent adult progenitor cells (MAPCs) after traumatic brain injury (TBI) in rodents provides neuroprotection by preserving the blood-brain barrier and systemically attenuating inflammation in the acute time frame following cell treatment; however, the long-term behavioral and anti-inflammatory effects of MAPC administration after TBI have yet to be explored. We hypothesized that the intravenous injection of MAPCs after TBI attenuates the inflammatory response (as measured by microglial morphology) and improves performance at motor tasks and spatial learning (Morris water maze [MWM]). MAPCs were administered intravenously 2 and 24 hours after a cortical contusion injury (CCI). We tested four groups at 120 days after TBI: sham (uninjured), injured but not treated (CCI), and injured and treated with one of two concentrations of MAPCs, either 2 million cells per kilogram (CCI-2) or 10 million cells per kilogram (CCI-10). CCI-10 rats showed significant improvement in left hind limb deficit on the balance beam. On the fifth day of MWM trials, CCI-10 animals showed a significant decrease in both latency to platform and distance traveled compared with CCI. Probe trials revealed a significant decrease in proximity measure in CCI-10 compared with CCI, suggesting improved memory retrieval. Neuroinflammation was quantified by enumerating activated microglia in the ipsilateral hippocampus. We observed a significant decrease in the number of activated microglia in the dentate gyrus in CCI-10 compared with CCI. Our results demonstrate that intravenous MAPC treatment after TBI in a rodent model offers long-term improvements in spatial learning as well as attenuation of neuroinflammation.

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Xuan AG, 2012, J NEUROINFLAMM, V9, DOI 10.1186/1742-2094-9-202

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PU WILEY

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AU Wang, GH

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Leak, RK

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TI Microglia/macrophage polarization dynamics in white matter after

traumatic brain injury

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE inflammation; macrophage; microglia; polarization; white matter injury

ID SPINAL-CORD; NEURODEGENERATIVE DISEASES; ACTIVATION PATTERNS; AXONAL

INJURY; CELL-DEATH; MICROGLIA; INFLAMMATION; RAT; NEUROPROTECTION;

IDENTIFICATION

AB Mononuclear phagocytes are a population of multi-phenotypic cells and have dual roles in brain destruction/reconstruction. The phenotype-specific roles of microglia/macrophages in traumatic brain injury (TBI) are, however, poorly characterized. In the present study, TBI was induced in mice by a controlled cortical impact (CCI) and animals were killed at 1 to 14 days post injury. Real-time polymerase chain reaction (RT-PCR) and immunofluorescence staining for M1 and M2 markers were performed to characterize phenotypic changes of microglia/macrophages in both gray and white matter. We found that the number of M1-like phagocytes increased in cortex, striatum and corpus callosum (CC) during the first week and remained elevated until at least 14 days after TBI. In contrast, M2-like microglia/macrophages peaked at 5 days, but decreased rapidly thereafter. Notably, the severity of white matter injury (WMI), manifested by immunohistochemical staining for neurofilannent SMI-32, was strongly correlated with the number of M1-like phagocytes. In vitro experiments using a conditioned medium transfer system confirmed that M1 microglia-conditioned media exacerbated oxygen glucose deprivation-induced oligodendrocyte death. Our results indicate that microglia/macrophages respond dynamically to TBI, experiencing a transient M2 phenotype followed by a shift to the M1 phenotype. The M1 phenotypic shift may propel WMI progression and represents a rational target for TBI treatment.

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JI J. Cereb. Blood Flow Metab.

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WC Endocrinology & Metabolism; Hematology; Neurosciences

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TI Astragaloside Improves Outcomes of Traumatic Brain Injury in Rats by

Reducing Microglia Activation

SO AMERICAN JOURNAL OF CHINESE MEDICINE

LA English

DT Article

DE Traumatic Brain Injury; Astragaloside; Tumor Necrosis Factor-Alpha;

Microglia

ID TUMOR-NECROSIS-FACTOR; FACTOR-ALPHA; INHIBITION; MOTOR

AB Astragaloside (AST) is traditionally prescribed for the prevention and treatment of cerebrovascular diseases. We directly tested the therapeutic effects of AST in a rat model of traumatic brain injury (TBI). One hour after the onset of TBI rats were given Saline (1 ml/kg) or AST (20-80 mg/kg) via i.p. injection. AST causes the attenuation of TBI-induced cerebral contusion, neuronal apoptosis, and neurological motor dysfunction. TBI-induced microglial activation evidenced by the morphological transformation of microglia (or ameboid microglia) and the microglial overexpression of tumor necrosis factor-alpha was reduced by AST. Our results indicate that AST may protect against brain contusion and neuronal apoptosis after TBI by attenuating microglia activation in male rats.

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PU WORLD SCIENTIFIC PUBL CO PTE LTD

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AU Xue, FT

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TI Positive Allosteric Modulators (PAMs) of Metabotropic Glutamate Receptor

5 (mGluR5) Attenuate Microglial Activation

SO CNS & NEUROLOGICAL DISORDERS-DRUG TARGETS

LA English

DT Article

DE Metabotropic glutamate receptor 5; microglial activation;

neuroinflammation; neuroprotection; positive allosteric modulator;

traumatic brain injury

ID PROTEIN-COUPLED RECEPTORS; IN-VIVO; BRAIN-INJURY; DRUG DEVELOPMENT;

CELL-DEATH; RAT; DISCOVERY; VITRO; SUBTYPE-5; APOPTOSIS

AB Traumatic brain injury causes progressive neurodegeneration associated with chronic microglial activation. Recent studies show that neurodegeneration and neuroinflammation after traumatic brain injury can be inhibited as late as one month in animals by the activation of the metabotropic glutamate receptor 5 in microglia using (RS)-2-chloro-5-hydroxy-phenylglycine. However, the therapeutic potential of this agonist is limited due to its relatively weak potency and brain permeability. To address such concerns, we evaluated the anti-inflammatory activities of several positive allosteric modulators using various in vitro assays, and found that 3,3'-difluorobenzaldazine, 3-cyano-N-(1,3-diphenyl-1H-pyrazol-5-yl) benzamide and 4-nitro-N-(1-(2-fluorophenyl)-3-phenyl-1H-pyrazol-5-yl) benzamide showed significantly improved potency which makes them potential lead compounds for further development of positive allosteric modulators for the treatment of traumatic brain injury.

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CR Allen JW, 1999, NEUROPHARMACOLOGY, V38, P1243, DOI 10.1016/S0028-3908(99)00044-1

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U2 2

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J9 CNS NEUROL DISORD-DR

JI CNS Neurol. Disord.-Drug Targets

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WC Neurosciences; Pharmacology & Pharmacy

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PT J

AU Miremami, JD

Talauliker, PM

Harrison, JL

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Lifshitz, Jonathan

TI Neuropathology in sensory, but not motor, brainstem nuclei of the rat

whisker circuit after diffuse brain injury

SO SOMATOSENSORY AND MOTOR RESEARCH

LA English

DT Article

DE Microglial activation; neuropathology; rat; traumatic brain injury;

vibrissal circuitry

ID POST-CONCUSSION SYNDROME; TRAUMATIC AXONAL INJURY; AXOLEMMAL

PERMEABILITY; NEUROLOGICAL RECOVERY; SOMATOSENSORY CORTEX;

HEAD-INJURIES; LATE-ONSET; RESPONSES; DEGENERATION; ACTIVATION

AB Neurological dysfunction after traumatic brain injury (TBI) is associated with pathology in cortical, subcortical, and brainstem nuclei. Our laboratory has reported neuropathology and microglial activation in the somatosensory barrel cortex (S1BF) and ventral posterior medial thalamus (VPM) after diffuse TBI in the rat, which correlated with post-injury whisker sensory sensitivity. The present study extends our previous work by evaluating pathology in whisking-associated sensory and motor brainstem nuclei. Brains from adult, male rats were recovered over 1 month after midline fluid percussion or sham injury. The principal trigeminal nucleus (PrV, sensory nucleus) and facial nucleus (VIIN, motor nucleus) were examined for neuropathology (silver histochemistry) and microglial activation (Iba1). Significant neuropathology in PrV was evident at 2 and 7 days post-injury compared to sham. Iba1-labeled microglia showed swollen somata and thickened processes over 1 month post-injury. In contrast, the VIIN showed non-significant neuropathology and reduced labeling of activated Iba1 microglia over 1 month post-injury. Together with our previous data, neuropathology and neuroinflammation in the whisker somatosensory pathway may contribute to post-injury sensory sensitivity more than the motor pathway. Whether these findings are direct results of the mechanical injury or consequences of progressive degeneration remains to be determined.

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TI The problem of axonal injury in the brains of veterans with histories of

blast exposure

SO ACTA NEUROPATHOLOGICA COMMUNICATIONS

LA English

DT Article

DE Traumatic brain injury; Diffuse axonal injury; APP; Axon bulbs; Opiate;

Microglia

ID AMYLOID PRECURSOR PROTEIN; POSTTRAUMATIC-STRESS-DISORDER; WHITE-MATTER;

POSTCONCUSSIVE SYMPTOMS; EXPERIMENTAL-MODELS; HEAD-INJURY; BETA-APP;

MILD; ACCUMULATION; DEGENERATION

AB Introduction: Blast injury to brain, a hundred-year old problem with poorly characterized neuropathology, has resurfaced as health concern in recent deployments in Iraq and Afghanistan. To characterize the neuropathology of blast injury, we examined the brains of veterans for the presence of amyloid precursor protein (APP)-positive axonal swellings typical of diffuse axonal injury (DAI) and compared them to healthy controls as well as controls with opiate overdose, anoxic-ischemic encephalopathy, and non-blast TBI (falls and motor vehicle crashes).

Results: In cases with blast history, we found APP (+) axonal abnormalities in several brain sites, especially the medial dorsal frontal white matter. In white matter, these abnormalities were featured primarily by clusters of axonal spheroids or varicosities in a honeycomb pattern with perivascular distribution. Axonal abnormalities colocalized with IBA1 (+) reactive microglia and had an appearance that was distinct from classical DAI encountered in TBI due to motor vehicle crashes. Opiate overdose cases also showed APP (+) axonal abnormalities, but the intensity of these lesions was lower compared to cases with blast histories and there was no clear association of such lesions with microglial activation.

Conclusions: Our findings demonstrate that many cases with history of blast exposure are featured by APP (+) axonopathy that may be related to blast exposure, but an important role for opiate overdose, antemortem anoxia, and concurrent blunt TBI events in war theater or elsewhere cannot be discounted.

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TI Temporal patterns of cortical proliferation of glial cell populations

after traumatic brain injury in mice

SO ASN NEURO

LA English

DT Article

DE astrocytes; BrdU; microglia; neurogenesis; NG2; proliferation; traumatic

brain injury (TBI)

ID CENTRAL-NERVOUS-SYSTEM; ADULT-RAT; ENHANCED NEUROGENESIS; SUBVENTRICULAR

ZONE; EXPRESSING CELLS; IMPACT INJURY; SPINAL-CORD; SCAR; ASTROCYTES;

MICROGLIA

AB TBI (traumatic brain injury) triggers an inflammatory cascade, gliosis and cell proliferation following cell death in the pericontusional area and surrounding the site of injury. In order to better understand the proliferative response following CCI (controlled cortical impact) injury, we systematically analyzed the phenotype of dividing cells at several time points post-lesion. C57BL/6 mice were subjected to mild to moderate CCI over the left sensory motor cortex. At different time points following injury, mice were injected with BrdU (bromodeoxyuridine) four times at 3-h intervals and then killed. The greatest number of proliferating cells in the pericontusional region was detected at 3 dpi (days post-injury). At 1 dpi, NG2(+) cells were the most proliferative population, and at 3 and 7 dpi the Iba-1(+) microglial cells were proliferating more. A smaller, but significant number of GFAP(+) (glial fibrillary acidic protein) astrocytes proliferated at all three time points. Interestingly, at 3 dpi we found a small number of proliferating neuroblasts [DCX+ (doublecortin)] in the injured cortex. To determine the cell fate of proliferative cells, mice were injected four times with BrdU at 3 dpi and killed at 28 dpi. Approximately 70% of proliferative cells observed at 28 dpi were GFAP+ astrocytes. In conclusion, our data suggest that the specific glial cell types respond differentially to injury, suggesting that each cell type responds to a specific pattern of growth factor stimulation at each time point after injury.

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TI Neuroprotective Strategies for Traumatic Brain Injury: Improving

Clinical Translation

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Review

DE clinical trial design; multipotential neuroprotective approaches;

microglial and astrocyte activation; programmed cell death; experimental

head injury; translational challenges; caspase-dependent and

AIF-mediated cell death; autophagy

ID APOPTOSIS-INDUCING FACTOR; CELL-CYCLE ACTIVATION; CYCLOSPORINE-A;

FUNCTIONAL DEFICITS; ALZHEIMERS-DISEASE; PROVIDES NEUROPROTECTION;

LIMITS NEUROINFLAMMATION; COGNITIVE RECOVERY; CORTICAL CONTUSION;

GENDER-DIFFERENCES

AB Traumatic brain injury (TBI) induces secondary biochemical changes that contribute to delayed neuroinflammation, neuronal cell death, and neurological dysfunction. Attenuating such secondary injury has provided the conceptual basis for neuroprotective treatments. Despite strong experimental data, more than 30 clinical trials of neuroprotection in TBI patients have failed. In part, these failures likely reflect methodological differences between the clinical and animal studies, as well as inadequate pre-clinical evaluation and/or trial design problems. However, recent changes in experimental approach and advances in clinical trial methodology have raised the potential for successful clinical translation. Here we critically analyze the current limitations and translational opportunities for developing successful neuroprotective therapies for TBI.

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TI Progressive Neurodegeneration After Experimental Brain Trauma:

Association With Chronic Microglial Activation

SO JOURNAL OF NEUROPATHOLOGY AND EXPERIMENTAL NEUROLOGY

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DT Article

DE Chronic microglial activation; NADPH oxidase; Progressive

neurodegeneration; Traumatic brain injury

ID INTRACEREBRAL INFLAMMATORY RESPONSE; CONTROLLED CORTICAL IMPACT; NADPH

OXIDASE; LIMITS NEUROINFLAMMATION; MEDIATED NEUROTOXICITY; OXIDATIVE

STRESS; INJURY; EXPRESSION; LIPOPOLYSACCHARIDE; MORPHOLOGY

AB Recent clinical studies indicate that traumatic brain injury (TBI) produces chronic and progressive neurodegenerative changes leadingto late neurologic dysfunction, but little is known about the mechanisms underlying such changes. Microglial-mediated neuroinflammationis an important secondary injury mechanism after TBI. In human studies, microglial activation has been found to persist for many years after the initial brain trauma, particularly after moderate to severe TBI. In the present study, adult C57Bl/6 mice were subjected to single moderate-level controlled cortical impact and were followed up by longitudinal T2-weighted magnetic resonance imaging in combination with stereologic histologic assessment of lesion volume expansion, neuronal loss, and microglial activation for up to 1 year after TBI. Persistent microglial activation was observed in the injured cortex through 1year after injury and was associated with progressive lesion expansion, hippocampal neurodegeneration, and loss of myelin. Notably, highly activated microglia that expressed major histocompatibility complex class II (CR3/43), CD68, and NADPH oxidase (NOX2) were detected at the margins of the expanding lesion at 1 year after injury; biochemical markers of neuroinflammation and oxidative stress were significantly elevated at this time point. These data support emerging clinical TBI findings and provide a mechanistic link between TBI-induced chronic microglial activation and progressive neurodegeneration.

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TC 340

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U1 1

U2 35

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PA JOURNALS DEPT, 2001 EVANS RD, CARY, NC 27513 USA

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J9 J NEUROPATH EXP NEUR

JI J. Neuropathol. Exp. Neurol.

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PG 16

WC Clinical Neurology; Neurosciences; Pathology

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SC Neurosciences & Neurology; Pathology

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PT J

AU Kabadi, SV

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Loane, DJ

Luo, T

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AF Kabadi, Shruti V.

Stoica, Bogdan A.

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Luo, Tao

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TI CR8, a novel inhibitor of CDK, limits microglial activation,

astrocytosis, neuronal loss, and neurologic dysfunction after

experimental traumatic brain injury

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE cell cycle; CR8; cyclin-dependent kinases; neurodegeneration;

neuroinflammation; traumatic brain injury

ID CELL-CYCLE ACTIVATION; ADULT NEUROGENESIS; NEUROINFLAMMATION;

NEUROPROTECTION; DEATH; RAT; NEURODEGENERATION; PROLIFERATION;

CONTRIBUTES; EXPRESSION

AB Central nervous system injury causes a marked increase in the expression of cell cycle-related proteins. In this study, we show that cell cycle activation (CCA) is detected in mature neurons at 24 hours after rat lateral fluid percussion (LFP)-induced traumatic brain injury (TBI), as reflected by increased expression of cyclin G1, phosphorylated retinoblastoma (phospho-Rb), E2F1 and proliferating cell nuclear antigen (PCNA). These changes were associated with progressive cortical, hippocampal, and thalamic neuronal loss and microglial and astrocyte activation. Notably, we detected 5-bromo-2'-deoxyuridine (BrdU)-positive neurons, microglia, and astrocytes at 7 days, but not at 24 hours, suggesting that cell cycle reaches the S phase in these cell types at the latter time point. A delayed systemic post-LFP administration at 3 hours of CR8 a potent second-generation cyclin-dependent kinase (CDK) inhibitor reduced CCA; cortical, hippocampal, and thalamic neuronal loss; and cortical microglial and astrocyte activation, Furthermore, CR8 treatment attenuated sensorimotor and cognitive deficits, alleviated depressive-like symptoms, and decreased lesion volume. These findings underscore the contribution of CCA to progressive neurodegeneration and chronic rieuroinflamrnation following TBI, and demonstrate the neuroprotective potential of cell cycle inhibition in a clinically relevant experimental TBI model.

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FX This work was supported by a grant from the National Institutes of

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J9 J CEREBR BLOOD F MET

JI J. Cereb. Blood Flow Metab.

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WC Endocrinology & Metabolism; Hematology; Neurosciences

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AU Vilalta, A

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AF Vilalta, Anna

Brown, Guy C.

TI Deoxyglucose prevents neurodegeneration in culture by eliminating

microglia

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Neurodegeneration; Glycolysis; Neuroinflammation; Brain trauma; Brain

ischemia; Alzheimer's disease; Energetics; Glia; Microglia; Glucose

ID NEURONAL DEATH; AMYLOID-BETA; 2-DEOXY-D-GLUCOSE; INHIBITION; STRESS;

NEUROTOXICITY; PHAGOCYTOSIS; RESTRICTION; GLYCOLYSIS; INJURY

AB Background: 2-Deoxy-D-glucose is an inhibitor of glycolysis, which is protective in animal models of brain pathology, but the mechanisms of this protection are unclear. We examined whether, when and how deoxyglucose protects neurons in co-culture with astrocytes and microglia. Microglia are brain macrophages, which can damage neurons in inflammatory conditions.

Methods: Deoxyglucose was added to primary cultures of microglia and astrocytes from rat cortex, or neurons and glia from rat cerebellum, or the BV-2 microglial cell line, and cell death and cell functions were evaluated.

Results: Surprisingly, addition of deoxyglucose induced microglial loss and prevented spontaneous neuronal loss in long-term cultures of neurons and glia, while elimination of microglia by L-leucine-methyl ester prevented the deoxyglucose-induced neuroprotection. Deoxyglucose also prevented neuronal loss induced by addition of amyloid beta or disrupted neurons (culture models of Alzheimer's disease and brain trauma respectively). However, deoxyglucose greatly increased the neuronal death induced by hypoxia. Addition of deoxyglucose to pure microglia induced necrosis and loss, preceded by rapid ATP depletion and followed by phagocytosis of the microglia. Deoxyglucose did not kill astrocytes or neurons.

Conclusions: We conclude that deoxyglucose causes microglial loss by ATP depletion, and this can protect neurons from neurodegeneration, except in conditions of hypoxia. Deoxyglucose may thus be beneficial in brain pathologies mediated by microglia, including brain trauma, but not where hypoxia is involved.

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FU European Union [251867]

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number: 251867).

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NR 36

TC 33

Z9 34

U1 0

U2 19

PU BMC

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EI 1742-2094

J9 J NEUROINFLAMM

JI J. Neuroinflamm.

PD MAR 26

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VL 11

AR 58

DI 10.1186/1742-2094-11-58

PG 10

WC Immunology; Neurosciences

WE Science Citation Index Expanded (SCI-EXPANDED)

SC Immunology; Neurosciences & Neurology

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PT J

AU Paveliev, M

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Rauvala, H

Khiroug, L

AF Paveliev, Mikhail

Kislin, Mikhail

Molotkov, Dmitry

Yuryev, Mikhail

Rauvala, Heikki

Khiroug, Leonard

TI Acute Brain Trauma in Mice Followed By Longitudinal Two-photon Imaging

SO JOVE-JOURNAL OF VISUALIZED EXPERIMENTS

LA English

DT Article

DE Medicine; Issue 86; Trauma; Nervous System; animal models; Brain trauma;

in vivo multiphoton microscopy; dendrite; astrocyte; microglia; second

harmonic generation

ID TRANSGENIC MICE; CRANIAL WINDOW; LONG-TERM; IN-VIVO; INJURY; NEOCORTEX;

RESPONSES; CORTEX; IMPACT; MODEL

AB Although acute brain trauma often results from head damage in different accidents and affects a substantial fraction of the population, there is no effective treatment for it yet. Limitations of currently used animal models impede understanding of the pathology mechanism. Multiphoton microscopy allows studying cells and tissues within intact animal brains longitudinally under physiological and pathological conditions. Here, we describe two models of acute brain injury studied by means of two-photon imaging of brain cell behavior under posttraumatic conditions. A selected brain region is injured with a sharp needle to produce a trauma of a controlled width and depth in the brain parenchyma. Our method uses stereotaxic prick with a syringe needle, which can be combined with simultaneous drug application. We propose that this method can be used as an advanced tool to study cellular mechanisms of pathophysiological consequences of acute trauma in mammalian brain in vivo. In this video, we combine acute brain injury with two preparations: cranial window and skull thinning. We also discuss advantages and limitations of both preparations for multisession imaging of brain regeneration after trauma.

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U2 5

PU JOURNAL OF VISUALIZED EXPERIMENTS

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J9 JOVE-J VIS EXP

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PG 8

WC Multidisciplinary Sciences

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Faden, Alan I.

TI PARP-1 Inhibition Attenuates Neuronal Loss, Microglia Activation and

Neurological Deficits after Traumatic Brain Injury

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE microglial activation; controlled cortical impact; neuroprotection;

PJ34; PARP-1

ID APOPTOSIS-INDUCING FACTOR; POLYMERASE-1-DEPENDENT CELL-DEATH; FOCAL

CEREBRAL-ISCHEMIA; NITRIC-OXIDE SYNTHASE; POLY(ADP-RIBOSE) POLYMERASE-1;

NAD(+) DEPLETION; NEUROPROTECTION; MICE; PJ34; IDENTIFICATION

AB Traumatic brain injury (TBI) causes neuronal cell death as well as microglial activation and related neurotoxicity that contribute to subsequent neurological dysfunction. Poly (ADP-ribose) polymerase (PARP-1) induces neuronal cell death through activation of caspase-independent mechanisms, including release of apoptosis inducing factor (AIF), and microglial activation. Administration of PJ34, a selective PARP-1 inhibitor, reduced cell death of primary cortical neurons exposed to N-Methyl-N'-Nitro-N-Nitrosoguanidine (MNNG), a potent inducer of AIF-dependent cell death. PJ34 also attenuated lipopolysaccharide and interferon-gamma-induced activation of BV2 or primary microglia, limiting NF-kappa B activity and iNOS expression as well as decreasing generation of reactive oxygen species and TNF alpha. Systemic administration of PJ34 starting as late as 24 h after controlled cortical impact resulted in improved motor function recovery in mice with TBI. Stereological analysis demonstrated that PJ34 treatment reduced the lesion volume, attenuated neuronal cell loss in the cortex and thalamus, and reduced microglial activation in the TBI cortex. PJ34 treatment did not improve cognitive performance in a Morris water maze test or reduce neuronal cell loss in the hippocampus. Overall, our data indicate that PJ34 has a significant, albeit selective, neuroprotective effect after experimental TBI, and its therapeutic effect may be from multipotential actions on neuronal cell death and neuroinflammatory pathways.

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TI Macrophagic and microglial responses after focal traumatic brain injury

in the female rat

SO JOURNAL OF NEUROINFLAMMATION

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DT Article

DE Controlled cortical impact; Inflammation; Macrophage; Microglia; MRI;

Rat; Traumatic brain injury

ID TUMOR-ASSOCIATED MACROPHAGES; SPINAL-CORD-INJURY; CYTOKINE EXPRESSION;

NERVOUS-SYSTEM; ESTROUS-CYCLE; PROGESTERONE; ACTIVATION; RECOVERY;

NEUROINFLAMMATION; POLARIZATION

AB Background: After central nervous system injury, inflammatory macrophages (M1) predominate over anti-inflammatory macrophages (M2). The temporal profile of M1/M2 phenotypes in macrophages and microglia after traumatic brain injury (TBI) in rats is unknown. We subjected female rats to severe controlled cortical impact (CCI) and examined the postinjury M1/M2 time course in their brains.

Methods: The motor cortex (2.5 mm left laterally and 1.0 mm anteriorly from the bregma) of anesthetized female Wistar rats (ages 8 to 10 weeks; N = 72) underwent histologically moderate to severe CCI with a 5-mm impactor tip. Separate cohorts of rats had their brains dissociated into cells for flow cytometry, perfusion-fixed for immunohistochemistry (IHC) and ex vivo magnetic resonance imaging or flash-frozen for RNA and protein analysis. For each analytical method used, separate postinjury times were included for 24 hours; 3 or 5 days; or 1, 2, 4 or 8 weeks.

Results: By IHC, we found that the macrophagic and microglial responses peaked at 5 to 7 days post-TBI with characteristics of mixed populations of M1 and M2 phenotypes. Upon flow cytometry examination of immunological cells isolated from brain tissue, we observed that peak M2-associated staining occurred at 5 days post-TBI. Chemokine analysis by multiplex assay showed statistically significant increases in macrophage inflammatory protein 1 alpha and keratinocyte chemoattractant/growth-related oncogene on the ipsilateral side within the first 24 hours after injury relative to controls and to the contralateral side. Quantitative RT-PCR analysis demonstrated expression of both M1- and M2-associated markers, which peaked at 5 days post-TBI.

Conclusions: The responses of macrophagic and microglial cells to histologically severe CCI in the female rat are maximal between days 3 and 7 postinjury. The response to injury is a mixture of M1 and M2 phenotypes.

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TI Delayed Increases in Microvascular Pathology after Experimental

Traumatic Brain Injury Are Associated with Prolonged Inflammation,

Blood-Brain Barrier Disruption, and Progressive White Matter Damage

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE BBB; CD 68; chronic TBI; GFAP; ICAM-1; microbleeds; microglia

ID CORTICAL IMPACT INJURY; MILD COGNITIVE IMPAIRMENT; CEREBRAL MICROBLEEDS;

MICROGLIAL ACTIVATION; DISEASE; ASTROCYTES; IRON; RAT; DEGENERATION;

DYSFUNCTION

AB Traumatic brain injury (TBI) is a significant risk factor for chronic traumatic encephalopathy (CTE), Alzheimer's disease (AD), and Parkinson's disease (PD). Cerebral microbleeds, focal inflammation, and white matter damage are associated with many neurological and neurodegenerative disorders including CTE, AD, PD, vascular dementia, stroke, and TBI. This study evaluates microvascular abnormalities observed at acute and chronic stages following TBI in rats, and examines pathological processes associated with these abnormalities. TBI in adult rats was induced by controlled cortical impact (CCI) of two magnitudes. Brain pathology was assessed in white matter of the corpus callosum for 24 h to 3 months following injury using immunohistochemistry (IHC). TBI resulted in focal microbleeds that were related to the magnitude of injury. At the lower magnitude of injury, microbleeds gradually increased over the 3 month duration of the study. IHC revealed TBI-induced focal abnormalities including blood-brain barrier (BBB) damage (IgG), endothelial damage (intercellular adhesion molecule 1 [ICAM-1]), activation of reactive microglia (ionized calcium binding adaptor molecule 1 [Iba1]), gliosis (glial fibrillary acidic protein [GFAP]) and macrophage-mediated inflammation (cluster of differentiation 68 [CD68]), all showing different temporal profiles. At chronic stages (up to 3 months), apparent myelin loss (Luxol fast blue) and scattered deposition of microbleeds were observed. Microbleeds were surrounded by glial scars and co-localized with CD68 and IgG puncta stainings, suggesting that localized BBB breakdown and inflammation were associated with vascular damage. Our results indicate that evolving white matter degeneration following experimental TBI is associated with significantly delayed microvascular damage and focal microbleeds that are temporally and regionally associated with development of punctate BBB breakdown and progressive inflammatory responses. Increased understanding of mechanisms underlying delayed microvascular damage following TBI could provide novel insights into chronic pathological responses to TBI and potential common mechanisms underlying TBI and neurodegenerative diseases.

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TI Bone Marrow Mesenchymal Stromal Cells Drive Protective M2 Microglia

Polarization After Brain Trauma

SO NEUROTHERAPEUTICS

LA English

DT Article

DE Traumatic brain injury; Microglia; Macrophages; Mesenchymal stromal

cells; Immunomodulation

ID SPINAL-CORD-INJURY; FOCAL CEREBRAL-ISCHEMIA; TUMOR-NECROSIS-FACTOR;

STEM-CELLS; ALTERNATIVE ACTIVATION; MACROPHAGE PLASTICITY; FUNCTIONAL

RECOVERY; RESIDENT MICROGLIA; SURFACE EXPRESSION; PHENOTYPE MARKERS

AB Microglia/macrophages (M) are major contributors to postinjury inflammation, but they may also promote brain repair in response to specific environmental signals that drive classic (M1) or alternative (M2) polarization. We investigated the activation and functional changes of M in mice with traumatic brain injuries and receiving intracerebroventricular human bone marrow mesenchymal stromal cells (MSCs) or saline infusion. MSCs upregulated Ym1 and Arginase-1 mRNA (p < 0.001), two M2 markers of protective M polarization, at 3 and 7 d postinjury, and increased the number of Ym1(+) cells at 7 d postinjury (p < 0.05). MSCs reduced the presence of the lysosomal activity marker CD68 on the membrane surface of CD11b-positive M (p < 0.05), indicating reduced phagocytosis. MSC-mediated induction of the M2 phenotype in M was associated with early and persistent recovery of neurological functions evaluated up to 35 days postinjury (p < 0.01) and reparative changes of the lesioned microenvironment. In vitro, MSCs directly counteracted the proinflammatory response of primary murine microglia stimulated by tumor necrosis factor-alpha + interleukin 17 or by tumor necrosis factor-alpha + interferon-gamma and induced M2 proregenerative traits, as indicated by the downregulation of inducible nitric oxide synthase and upregulation of Ym1 and CD206 mRNA (p < 0.01). In conclusion, we found evidence that MSCs can drive the M transcriptional environment and induce the acquisition of an early, persistent M2-beneficial phenotype both in vivo and in vitro. Increased Ym1 expression together with reduced in vivo phagocytosis suggests M selection by MSCs towards the M2a subpopulation, which is involved in growth stimulation and tissue repair.

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TI Is neuroinflammation in the injured spinal cord different than in the

brain? Examining intrinsic differences between the brain and spinal cord

SO EXPERIMENTAL NEUROLOGY

LA English

DT Review

DE Astrocyte; Macrophage; Microglia; Neutrophil; Traumatic brain injury;

Spinal cord injury; Monocyte; Secondary cell death; Translational;

Anti-cd11d; Blood brain barrier; Blood spinal cord barrier; Innate;

Immune; B cell; T cell; Autoimmune; Adaptive immune response; Protective

autoimmunity; Alternative activation; Ly6c; Ly6g; Gr1; SUR-1

ID EXPERIMENTAL ALLERGIC ENCEPHALOMYELITIS; CENTRAL-NERVOUS-SYSTEM;

ADAPTIVE IMMUNE-RESPONSES; MYELIN BASIC-PROTEIN; BLOOD-BRAIN; BARRIER

PERMEABILITY; INFLAMMATORY RESPONSE; MACROPHAGE ACTIVATION;

MONOCLONAL-ANTIBODY; PEROXYNITRITE SCAVENGER

AB The field of neuroimmunology is rapidly advancing. There is a growing appreciation for heterogeneity, both in inflammatory composition and region-specific inflammatory responses. This understanding underscores the importance of developing targeted immunomodulatory therapies for treating neurological disorders. Concerning neurotrauma, there is a dearth of publications directly comparing inflammatory responses in the brain and spinal cord after injury. The question therefore remains as to whether inflammatory cells responding to spinal cord vs. brain injury adopt similar functions and are therefore amenable to common therapies. In this review, we address this question while revisiting and modernizing the conclusions from publications that have directly compared inflammation across brain and spinal cord injuries. By examining molecular differences, anatomical variations, and inflammatory cell phenotypes between the injured brain and spinal cord, we provide insight into how neuroinflammation relates to neurotrauma and into fundamental differences between the brain and spinal cord. (C) 2014 Elsevier Inc. All rights reserved.

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TI Selective CDK inhibitors: promising candidates for future clinical

traumatic brain injury trials

SO NEURAL REGENERATION RESEARCH

LA English

DT Review

DE cell cycle inhibition; lateral fluid percussion; Roscovitine, CR-8;

behavior; microglial activation; neurodegeneration

ID CYCLIN-DEPENDENT KINASES; CELL-CYCLE; LIMITS NEUROINFLAMMATION;

ALZHEIMERS-DISEASE; NEURONAL LOSS; ROSCOVITINE; ACTIVATION;

NEURODEGENERATION; NEUROPROTECTION; PROLIFERATION

AB Traumatic brain injury induces secondary injury that contributes to neuroinflammation, neuronal loss, and neurological dysfunction. One important injury mechanism is cell cycle activation which causes neuronal apoptosis and glial activation. The neuroprotective effects of both non-selective (Flavopiridol) and selective (Roscovitine and CR-8) cyclin-dependent kinase inhibitors have been shown across multiple experimental traumatic brain injury models and species. Cyclin-dependent kinaseinhibitors, administered as a single systemic dose up to 24 hours after traumatic brain injury, provide strong neuroprotection-reducing neuronal cell death, neuroinflammation and neurological dysfunction. Given their effectiveness and long therapeutic window, cyclin-dependent kinase inhibitors appear to be promising candidates for clinical traumatic brain injury trials.

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TI Melatonin reduced microglial activation and alleviated neuroinflammation

induced neuron degeneration in experimental traumatic brain injury:

Possible involvement of mTOR pathway

SO NEUROCHEMISTRY INTERNATIONAL

LA English

DT Article

DE Traumatic brain injury; Melatonin; Microglia activation; Inflammation;

Mammalian target of rapamycin

ID CENTRAL-NERVOUS-SYSTEM; CLOSED-HEAD INJURY; MAMMALIAN TARGET; FUNCTIONAL

RECOVERY; OXIDATIVE STRESS; UP-REGULATION; KAPPA-B; RAPAMYCIN; CELLS;

MODEL

AB This study was designed to detect the modulation manner of melatonin on microglial activation and explore herein possible involvement of mammalian target of rapamycin (mTOR) pathway following traumatic brain injury (TBI). ICR mice were divided into four groups: sham group, TBI group, TBI + sal group and TBI + Melatonin group. A weight-drop model was employed to cause TBI. Neurological severity score (NSS) tests were performed to measure behavioral outcomes. Nissl staining was conducted to observe the neuronal degeneration and wet-to-dry weight ratio indicated brain water content. Immunofluorescence was designed to investigate microglial activation. Enzyme-linked immunosorbent assay (ELISA) was employed to evaluate proinflammatory cytokine levels (interleukin-beta (IL-1 beta), tumor necrosis factor-alpha (TNF-alpha)). Western blotting was engaged to analyze the protein content of mammalian target of rapamycin (mTOR), p70 ribosomal S6 kinase (p70S6K) and S6 ribosomal protein (S6RP). Melatonin administration was associated with markedly restrained microglial activation, decreased release of proinflammatory cytokines and increased the number of surviving neurons at the site of pen-contusion. Meanwhile, melatonin administration resulted in dephosphorylated mTOR pathway. In conclusion, this study presents a new insight into the mechanisms responsible for the anti-neuroinflammation of melatonin, with possible involvement of mTOR pathway. (C) 2014 Elsevier Ltd. All rights reserved.

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NR 68

TC 83

Z9 86

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PT J

AU Balu, R

AF Balu, Ramani

TI Inflammation and Immune System Activation After Traumatic Brain Injury

SO CURRENT NEUROLOGY AND NEUROSCIENCE REPORTS

LA English

DT Review

DE Inflammation; Traumatic brain injury; Microglia; Damage-associated

molecular patterns; Cytokine; Pattern recognition receptor

ID CENTRAL-NERVOUS-SYSTEM; PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY;

PLACEBO-CONTROLLED TRIAL; CEREBRAL EDEMA; NATALIZUMAB; INFLAMMASOMES;

NEUTRALIZATION; SURVEILLANCE; MECHANISMS; AUTOIMMUNE

AB Treatment options for managing traumatic brain injury remain limited. Therapies that limit the development of secondary brain injury-the delayed injury that can occur days to weeks after initial presentation-would have a major impact on outcomes and reduce the medical, social, and economic burden of this devastating disease. A growing body of evidence suggests that inflammation and activation of the immune system is a central driver of secondary brain injury. This article reviews the evidence for inflammation mediating secondary injury after head trauma and outlines potential approaches for immunomodulatory therapies after traumatic brain injury.

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AU Bennett, RE

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AF Bennett, Rachel E.

Brody, David L.

TI Acute Reduction of Microglia Does Not Alter Axonal Injury in a Mouse

Model of Repetitive Concussive Traumatic Brain Injury

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE axon injury; concussion; microglia

ID COLLEGIATE FOOTBALL PLAYERS; HEAD-INJURY; COGNITIVE IMPAIRMENT;

RECURRENT CONCUSSION; WHITE-MATTER; BETA; ACTIVATION; ENCEPHALOPATHY;

VULNERABILITY; INFLAMMATION

AB The pathological processes that lead to long-term consequences of multiple concussions are unclear. Primary mechanical damage to axons during concussion is likely to contribute to dysfunction. Secondary damage has been hypothesized to be induced or exacerbated by inflammation. The main inflammatory cells in the brain are microglia, a type of macrophage. This research sought to determine the contribution of microglia to axon degeneration after repetitive closed-skull traumatic brain injury (rcTBI) using CD11b-TK (thymidine kinase) mice, a valganciclovir-inducible model of macrophage depletion. Low-dose (1 mg/mL) valganciclovir was found to reduce the microglial population in the corpus callosum and external capsule by 35% after rcTBI in CD11b-TK mice. At both acute (7 days) and subacute (21 days) time points after rcTBI, reduction of the microglial population did not alter the extent of axon injury as visualized by silver staining. Further reduction of the microglial population by 56%, using an intermediate dose (10mg/mL), also did not alter the extent of silver staining, amyloid precursor protein accumulation, neurofilament labeling, or axon injury evident by electron microscopy at 7 days postinjury. Longer treatment of CD11b-TK mice with intermediate dose and treatment for 14 days with high-dose (50mg/mL) valganciclovir were both found to be toxic in this injury model. Altogether, these data are most consistent with the idea that microglia do not contribute to acute axon degeneration after multiple concussive injuries. The possibility of longer-term effects on axon structure or function cannot be ruled out. Nonetheless, alternative strategies directly targeting injury to axons may be a more beneficial approach to concussion treatment than targeting secondary processes of microglial-driven inflammation.

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U2 16

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JI J. Neurotrauma

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TI Novel mGluR5 Positive Allosteric Modulator Improves Functional Recovery,

Attenuates Neurodegeneration, and Alters Microglial Polarization after

Experimental Traumatic Brain Injury

SO NEUROTHERAPEUTICS

LA English

DT Article

DE Traumatic brain injury; Metabotropic glutamate receptor 5; Positive

allosteric modulator; Neuroprotection; Functional recovery; Microglial

activation

ID METABOTROPIC GLUTAMATE RECEPTORS; IN-VIVO ACTIVITY; ALTERNATIVE

ACTIVATION; CELL-DEATH; NEURONAL APOPTOSIS; SUBTYPE 5; INFLAMMATION;

ANTAGONISTS; INHIBITION; TARGETS

AB Traumatic brain injury (TBI) causes microglial activation and related neurotoxicity that contributes to chronic neurodegeneration and loss of neurological function. Selective activation of metabotropic glutamate receptor 5 (mGluR5) by the orthosteric agonist (RS)-2-chloro-5-hydroxyphenylglycine (CHPG), is neuroprotective in experimental models of TBI, and has potent anti-inflammatory effects in vitro. However, the therapeutic potential of CHPG is limited due to its relatively weak potency and brain permeability. Highly potent, selective and brain penetrant mGluR5 positive allosteric modulators (PAMs) have been developed and show promise as therapeutic agents. We evaluated the therapeutic potential of a novel mGluR5 PAM, VU0360172, after controlled cortical impact (CCI) in mice. Vehicle, VU0360172, or VU0360172 plus mGluR5 antagonist (MTEP), were administered systemically to CCI mice at 3 h post-injury; lesion volume, hippocampal neurodegeneration, microglial activation, and functional recovery were assessed through 28 days post-injury. Anti-inflammatory effects of VU0360172 were also examined in vitro using BV2 and primary microglia. VU0360172 treatment significantly reduced the lesion, attenuated hippocampal neurodegeneration, and improved motor function recovery after CCI. Effects were mediated by mGluR5 as co-administration of MTEP blocked the protective effects of VU0360172. VU0360172 significantly reduced CD68 and NOX2 expression in activated microglia in the cortex at 28 days post-injury, and also suppressed pro-inflammatory signaling pathways in BV2 and primary microglia. In addition, VU0360172 treatment shifted the balance between M1/M2 microglial activation states towards an M2 pro-repair phenotype. This study demonstrates that VU0360172 confers neuroprotection after experimental TBI, and suggests that mGluR5 PAMs may be promising therapeutic agents for head injury.

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TI Traumatic White Matter Injury and Glial Activation: From Basic Science

to Clinics

SO GLIA

LA English

DT Review

DE traumatic brain injury; traumatic axonal injury; diffuse axonal injury;

glial activation; neuroinflammation; microglia; astrocyte; biomarkers;

magnetic resonance imaging; neuroimaging; diffusion tensor imaging

ID DIFFUSE AXONAL INJURY; CORTICAL IMPACT INJURY; TENSOR IMAGING DETECTS;

PERCUSSION BRAIN INJURY; CENTRAL-NERVOUS-SYSTEM; ALPHA-II-SPECTRIN;

HEAD-INJURY; RAT MODEL; MICROGLIAL ACTIVATION; MULTIPLE-SCLEROSIS

AB An improved understanding and characterization of glial activation and its relationship with white matter injury will likely serve as a novel treatment target to curb post injury inflammation and promote axonal remyelination after brain trauma. Traumatic brain injury (TBI) is a significant public healthcare burden and a leading cause of death and disability in the United States. Particularly, traumatic white matter (WM) injury or traumatic axonal injury has been reported as being associated with patients' poor outcomes. However, there is very limited data reporting the importance of glial activation after TBI and its interaction with WM injury. This article presents a systematic review of traumatic WM injury and the associated glial activation, from basic science to clinical diagnosis and prognosis, from advanced neuroimaging perspective. It concludes that there is a disconnection between WM injury research and the essential role of glia which serve to restore a healthy environment for axonal regeneration following WM injury. Particularly, there is a significant lack of non-invasive means to characterize the complex pathophysiology of WM injury and glial activation in both animal models and in humans. An improved understanding and characterization of the relationship between glia and WM injury will likely serve as a novel treatment target to curb post injury inflammation and promote axonal remyelination.

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TI Contributions of the immune system to the pathophysiology of traumatic

brain injury - evidence by intravital microscopy

SO FRONTIERS IN CELLULAR NEUROSCIENCE

LA English

DT Review

DE brain trauma; secondary brain damage; inflammation; leukocytes;

microglia; innate immune answer; in vivo imaging; intravital microscopy

ID CONTROLLED CORTICAL IMPACT; CENTRAL-NERVOUS-SYSTEM; CEREBRAL-BLOOD-FLOW;

SPINAL-CORD-INJURY; INTRACEREBRAL INFLAMMATORY RESPONSE; IN-VIVO;

FLUORESCENCE MICROSCOPY; LEUKOCYTE RECRUITMENT; BARRIER PERMEABILITY;

CONTUSION VOLUME

AB Traumatic brain injury (TBI) results in immediate brain damage that is caused by the mechanical impact and is non-reversible. This initiates a cascade of delayed processes which cause additional-secondary-brain damage. Among these secondary mechanisms, the inflammatory response is believed to play an important role, mediating actions that can have both protective and detrimental effects on the progression of secondary brain damage. Histological data generated extensive information; however, this is only a snapshot of processes that are, in fact, very dynamic. In contrast, in vivo microscopy provides detailed insight into the temporal and spatial patterns of cellular dynamics. In this review, we aim to summarize data which was generated by in vivo microscopy, specifically investigating the immune response following brain trauma, and its potential effects on secondary brain damage.

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Huang, Huei-Sheng

TI Involvement of TG-interacting factor in microglial activation during

experimental traumatic brain injury

SO JOURNAL OF NEUROCHEMISTRY

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DT Article

DE microglial activation; TG-interacting factor; traumatic brain injury

ID HOMEODOMAIN PROTEIN TGIF; PEPTIDE INHIBITOR; UBIQUITIN LIGASE;

GENE-EXPRESSION; INFLAMMATION; CELLS; MODEL; JNK; ANGIOGENESIS;

SUPERFAMILY

AB Traumatic brain injury (TBI) is a complex injury involving several physiological alterations, potentially leading to neurological impairment. Previous mouse studies using high-density oligonucleotide array analysis have confirmed the upregulation of transforming growth-interacting factor (TGIF) mRNA in TBI. TGIF is a transcriptional corepressor of transforming growth factor beta (TGF-) signaling which plays a protective role in TBI. However, the functional roles of TGIF in TBI are not well understood. In this study, we used confocal microscopy after immunofluorescence staining to demonstrate the increase of TGIF levels in the activated microglia of the pericontusional cortex of rats with TBI. Intracerebral knockdown of TGIF in the pericontusional cortex significantly downregulated TGIF expression, attenuated microglial activation, reduced the volume of damaged brain tissue, and facilitated recovery of limb motor function. Collectively, our results indicate that TGIF is involved in TBI-induced microglial activation, resulting in secondary brain injury and motor dysfunction.

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TI Merging pathology with biomechanics using CHIMERA (Closed-Head Impact

Model of Engineered Rotational Acceleration): a novel, surgery-free

model of traumatic brain injury

SO MOLECULAR NEURODEGENERATION

LA English

DT Article

DE Traumatic brain injury; Animal model of traumatic brain injury; Animal

model of closed head injury; Diffuse axonal injury; Microglia

activation; Neuroinflammation; Tau hyperphosphorylation; Head

kinematics; Head injury biomechanics; Impact-acceleration traumatic

brain injury model; Surgery-free animal model of traumatic brain injury;

Traumatic brain injury biomechanics

ID DIFFUSE AXONAL INJURY; PROFESSIONAL FOOTBALL; ANGULAR-ACCELERATION;

MOUSE MODEL; RAT MODEL; CONCUSSION; MICE; EPIDEMIOLOGY; MECHANISMS;

PATHOPHYSIOLOGY

AB Background: Traumatic brain injury (TBI) is a major health care concern that currently lacks any effective treatment. Despite promising outcomes from many preclinical studies, clinical evaluations have failed to identify effective pharmacological therapies, suggesting that the translational potential of preclinical models may require improvement. Rodents continue to be the most widely used species for preclinical TBI research. As most human TBIs result from impact to an intact skull, closed head injury (CHI) models are highly relevant, however, traditional CHI models suffer from extensive experimental variability that may be due to poor control over biomechanical inputs. Here we describe a novel CHI model called CHIMERA (Closed-Head Impact Model of Engineered Rotational Acceleration) that fully integrates biomechanical, behavioral, and neuropathological analyses. CHIMERA is distinct from existing neurotrauma model systems in that it uses a completely non-surgical procedure to precisely deliver impacts of prescribed dynamic characteristics to a closed skull while enabling kinematic analysis of unconstrained head movement. In this study, we characterized head kinematics as well as functional, neuropathological, and biochemical outcomes up to 14d following repeated TBI (rTBI) in adult C57BL/6 mice using CHIMERA.

Results: Head kinematic analysis showed excellent repeatability over two closed head impacts separated at 24h. Injured mice showed significantly prolonged loss of righting reflex and displayed neurological, motor, and cognitive deficits along with anxiety-like behavior. Repeated TBI led to diffuse axonal injury with extensive microgliosis in white matter from 2-14d post-rTBI. Injured mouse brains also showed significantly increased levels of TNF-alpha and IL-1 beta and increased endogenous tau phosphorylation.

Conclusions: Repeated TBI using CHIMERA mimics many of the functional and pathological characteristics of human TBI with a reliable biomechanical response of the head. This makes CHIMERA well suited to investigate the pathophysiology of TBI and for drug development programs.

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TI Widespread microglial activation in patients deceased from traumatic

brain injury

SO BRAIN INJURY

LA English

DT Article

DE Human; microglial activation; traumatic brain injury; Ricinus communis

lectin

ID PARENCHYMAL MICROGLIA/MACROPHAGES; REGIONAL VULNERABILITY;

PARKINSONS-DISEASE; EXPRESSION; INFLAMMATION; MONOCYTES; CELLS; MICE;

CNS; STIMULATION

AB Primary objective: The role of microglial activation in traumatic brain injury (TBI) has been extensively described in established animal models. In contrast, very few studies have analysed this process in human patients, the majority being focused on the local reaction in the contused parenchyma. In this work, the main objective was the analysis of microglial activation in brain regions distant from the primary lesion. Research design: Morphological changes of microglia were evaluated in the cerebral cortex of patients deceased from TBI in comparison with control subjects.

Methods and procedures: Cortical samples from five cases with TBI and 10 controls were evaluated using Ricinus communis lectin histochemistry and conventional Hematoxylin- eosin staining.

Main outcomes and results: It was observed that microglial cells from patients with TBI presented shorter and thicker cellular projections compared with controls. Moreover, the percentage of histological area reactive to lectin was statistically higher in samples from subjects with TBI. These signs of microglial activation were observed in all of the analysed cortical areas, thus indicating a generalized effect on the whole cerebral cortex. The results are consistent with previous imaging PET studies performed in living patients with the C-11-PK11195 radiotracer.

Conclusions: The findings indicate that TBI induces a widespread activation of brain microglia which affects all cortical areas, including those distant from the contusion site.

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TI Study of restorative processes in brain laceration in the first seven

days after traumatic brain injury

SO ROMANIAN JOURNAL OF MORPHOLOGY AND EMBRYOLOGY

LA English

DT Article

DE traumatic brain injury; cerebral laceration; gliosis; microglia

ID CENTRAL-NERVOUS-SYSTEM; COLONY-STIMULATING FACTOR; INTRACEREBRAL

HEMORRHAGE; MICROGLIA; ASTROCYTES; CELLS; INFLAMMATION; PARENCHYMA;

DISEASE

AB Traumatic brain injuries represent the main cause of death and invalidity all over the world. Persons surviving a severe traumatic brain injury often present long-term disabilities, sensitive and motor deficits, cognitive, vegetative or mental disorders. Brain injuries are directly caused by the traumatic agent, and indirectly caused by the action of cells involved in the restorative process. The main cells involved in the restorative process are microglias and astrocytes. By using an experimental model, we investigated the reaction of these cells in the first week after a severe brain injury, followed by brain laceration. Of the two cell types, the most rapid and intense reaction was held by the macroglias, also known as resident macrophages of the central nervous system. Alongside the activation of local microglias, in the restorative process there were also involved blood monocytes that turned into macrophages. 24 hours after the injury, the number of macrophage cells/mm(2) at brain wound level increased 2-4 times, after three days -10-12 times, and after seven days - over 20 times. The astrocyte reaction was slower, their activation being signaled no sooner than three days from injury, when their number in the perilesional brain parenchyma increased approximately two times, while after seven days -approximately 4-5 times. Both astrocytes and macrophages (microglias), besides their beneficial effects in restoring traumatic brain injuries, may have unfavorable effects upon the nervous cells in the immediate proximity of the injury. Destruction of vascular network by the traumatic agent, and the extremely slow restore of vascularization, partially explain brain neurons death on extend areas.

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TI Microglial Activation as a Compelling Target for Treating Acute

Traumatic Brain Injury

SO CURRENT MEDICINAL CHEMISTRY

LA English

DT Article

DE Brain; cytokines; free radicals; microglia; traumatic injury

ID NECROSIS-FACTOR-ALPHA; HYPERBARIC-OXYGEN THERAPY; IMPROVES FUNCTIONAL

RECOVERY; GLYCINE-PROLINE-GLUTAMATE; MESENCHYMAL STEM-CELLS; TNF-ALPHA;

PROINFLAMMATORY CYTOKINE; DOUBLECORTIN EXPRESSION;

MITOCHONDRIAL-FUNCTION; CEREBRAL-ISCHEMIA

AB Microglia and several inflammatory cytokines and neurotrophic growth factors are involved in traumatic brain injury (TBI). Tumor necrosis factor-alpha (TNF-alpha) can be released by microglia, astrocytes, and neurons. TNF-alpha has been reported to be both proneurogenic and antineurogenic, depending upon the model, method, and cell-derived region. There are two subtypes of microglia: M1 and M2. The former (or M1 subtype of non-phagocytic microglia) is able to secrete higher levels of TNF-alpha but lower levels of interleukin (IL)-10 (IL-10), an anti-inflammatory cytokine. Both the proinflammatory and the pro-apoptotic function can also be promoted by activation of tumor necrosis factor-receptor 1 (TNF-R1). In contrast, M2 activation produces lower levels of TNF-alpha but higher levels of IL-10. Pro-growth and survival pathways can be promoted by the activation of TNF-R2. During the acute stage of TBI, both M1 subtype of microglia and TNF-R1 are activated to cause higher levels of TNF-alpha but lower levels of IL-10, which lead to suppressed neurogenesis, neuronal loss and organ dysfunction (so-called microglial activation I). In contrast, activation of both M2 subtype of microglia and TNF-R2 is able to promote neurogenesis and tissue recovery (so-called microglial activation II). The severity of TBI depends upon the net effects between microglial activation I and microglial activation II. Indeed, by using rodent models of TBI, therapeutic evaluation studies reveal that several agents or strategies attenuate contused brain volume and neurological deficits by inhibiting microglial activation I but inducing microglial activation II. For example, etanercept therapy might attenuate contused brain volume and neurological deficits by inactivating the M1 subtype and TNF-R1 to reduce the microglial activation I response, but it might promote neurogenesis and functional recovery by activating the M2 subtype and TNF-R2. Therefore, based on microglial responses I and II, we conclude that future studies should focus on multiple therapeutic agents and strategies for optimal TBI therapy.

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TI Motor, Visual and Emotional Deficits in Mice after Closed-Head Mild

Traumatic Brain Injury Are Alleviated by the Novel CB2 Inverse Agonist

SMM-189

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

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DT Article

DE TBI; deficits; microglia; CB2 receptors; therapy

ID STRIATAL PROJECTION NEURON; GAMMA PKC-GAMMA; CONTRAST SENSITIVITY;

OPTIC-NERVE; MOUSE MODEL; CORTICOSPINAL TRACT; LOCOMOTOR BEHAVIOR;

IMMUNE-RESPONSES; AXONAL INJURY; BLAST INJURY

AB We have developed a focal blast model of closed-head mild traumatic brain injury (TBI) in mice. As true for individuals that have experienced mild TBI, mice subjected to 50-60 psi blast show motor, visual and emotional deficits, diffuse axonal injury and microglial activation, but no overt neuron loss. Because microglial activation can worsen brain damage after a concussive event and because microglia can be modulated by their cannabinoid type 2 receptors (CB2), we evaluated the effectiveness of the novel CB2 receptor inverse agonist SMM-189 in altering microglial activation and mitigating deficits after mild TBI. In vitro analysis indicated that SMM-189 converted human microglia from the pro-inflammatory M1 phenotype to the pro-healing M2 phenotype. Studies in mice showed that daily administration of SMM-189 for two weeks beginning shortly after blast greatly reduced the motor, visual, and emotional deficits otherwise evident after 50-60 psi blasts, and prevented brain injury that may contribute to these deficits. Our results suggest that treatment with the CB2 inverse agonist SMM-189 after a mild TBI event can reduce its adverse consequences by beneficially modulating microglial activation. These findings recommend further evaluation of CB2 inverse agonists as a novel therapeutic approach for treating mild TBI.

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TI Effects of pigment epithelium-derived factor on traumatic brain injury

SO RESTORATIVE NEUROLOGY AND NEUROSCIENCE

LA English

DT Article

DE PEDF; traumatic brain injury; cell death; microglia; SVZ

ID CONTROLLED CORTICAL IMPACT; CEREBELLAR GRANULE CELLS; FACTOR PEDF;

GROWTH-FACTOR; PROLIFERATION; BDNF; EXPRESSION; NEURONS; NEUROGENESIS;

INFLAMMATION

AB Purpose: Pigment epithelium-derived factor (PEDF) is a multifunctional protein with antiangiogenic, anti-inflammatory, neurotrophic and neurogenic properties. The effect of PEDF on traumatic brain injury (TBI) has not been explored. In this study, we aimed to show the in vivo effects of PEDF on lesion volume, cell death and cell proliferation after TBI. Methods: Rats were subjected to controlled cortical impact injury (CCII). PEDF mRNA brain levels were measured by RT-PCR. The lesion volume, cell proliferation, cell death and microglia activation were assessed in the brains of lesioned animals with intraventricular alzet infusion of PEDF or aCSF, and intraperitoneal injections of BrdU. Results: We detected a significant increase ofPEDFmRNAlevels after TBI. PEDFintraventricular infusion showed no significant effect on the contusion volume, whereas the number of dead cells, activated microglia, BrdU- positive cells around the lesion were significantly decreased. In contrast, PEDF application increased cell proliferation in the ipsilateral subventricular zone. No effect was found on cell proliferation in the dentate gyrus. Conclusion: The present work indicates that PEDF acts as a multifunctional agent after TBI influencing cell death, inflammation and cell proliferation.

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TI CB<sub>1</sub> and CB<sub>2</sub> Cannabinoid Receptor Antagonists

Prevent Minocycline-Induced Neuroprotection Following Traumatic Brain

Injury in Mice

SO CEREBRAL CORTEX

LA English

DT Article

DE cannabinoid system; diffuse axonal injury; microglial activation;

minocycline; neuroprotection; traumatic brain injury

ID CLOSED-HEAD INJURY; FOCAL CEREBRAL-ISCHEMIA; MICROGLIAL ACTIVATION;

PROTECTS NEURONS; KAPPA-B; EDEMA; EXPRESSION; CELLS; NEUROINFLAMMATION;

INVOLVEMENT

AB Traumatic brain injury (TBI) and its consequences represent one of the leading causes of death in young adults. This lesion mediates glial activation and the release of harmful molecules and causes brain edema, axonal injury, and functional impairment. Since glial activation plays a key role in the development of this damage, it seems that controlling it could be beneficial and could lead to neuroprotective effects. Recent studies show that minocycline suppresses microglial activation, reduces the lesion volume, and decreases TBI-induced locomotor hyperactivity up to 3 months. The endocannabinoid system (ECS) plays an important role in reparative mechanisms and inflammation under pathological situations by controlling some mechanisms that are shared with minocycline pathways. We hypothesized that the ECS could be involved in the neuroprotective effects of minocycline. To address this hypothesis, we used a murine TBI model in combination with selective CB1 and CB2 receptor antagonists (AM251 and AM630, respectively). The results provided the first evidence for the involvement of ECS in the neuroprotective action of minocycline on brain edema, neurological impairment, diffuse axonal injury, and microglial activation, since all these effects were prevented by the CB1 and CB2 receptor antagonists.

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TI Chronic Neurodegeneration After Traumatic Brain Injury: Alzheimer

Disease, Chronic Traumatic Encephalopathy, or Persistent

Neuroinflammation?

SO NEUROTHERAPEUTICS

LA English

DT Article

DE Traumatic brain injury; Neurodegeneration; Concussion; Repetitive mild

TBI; Alzheimer disease; Chronic traumatic encephalopathy; Microglial

activation; Chronic traumatic brain inflammation

ID AMYLOID-BETA-PROTEIN; INTRACEREBRAL INFLAMMATORY RESPONSE; LONG-TERM

ACCUMULATION; HEAD-INJURY; MICROGLIAL ACTIVATION; DIFFUSION-TENSOR;

AXONAL INJURY; RISK-FACTOR; LONGITUDINAL CHANGES; SYNAPTIC PLASTICITY

AB It has long been suggested that prior traumatic brain injury (TBI) increases the subsequent incidence of chronic neurodegenerative disorders, including Alzheimer disease, Parkinson disease, and amyotrophic lateral sclerosis. Among these, the association with Alzheimer disease has the strongest support. There is also a long-recognized association between repeated concussive insults and progressive cognitive decline or other neuropsychiatric abnormalities. The latter was first described in boxers as dementia pugilistica, and has received widespread recent attention in contact sports such as professional American football. The term chronic traumatic encephalopathy was coined to attempt to define a "specific" entity marked by neurobehavioral changes and the extensive deposition of phosphorylated tau protein. Nearly lost in the discussions of post-traumatic neurodegeneration after traumatic brain injury has been the role of sustained neuroinflammation, even though this association has been well established pathologically since the 1950s, and is strongly supported by subsequent preclinical and clinical studies. Manifested by extensive microglial and astroglial activation, such chronic traumatic brain inflammation may be the most important cause of post-traumatic neurodegeneration in terms of prevalence. Critically, emerging preclinical studies indicate that persistent neuroinflammation and associated neurodegeneration may be treatable long after the initiating insult(s).

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TI Granulocyte-Macrophage Colony Stimulating Factor Exerts Protective and

Immunomodulatory Effects in Cortical Trauma

SO JOURNAL OF NEUROIMMUNOLOGY

LA English

DT Article

DE Traumatic brain injury; Immunity; Microglia; Neuroinflammation; T cell

ID REGULATORY T-CELLS; EXPERIMENTAL MYASTHENIA-GRAVIS; BRAIN-INJURY;

GM-CSF; PARKINSONS-DISEASE; IN-VIVO; NEUROPROTECTIVE ACTIVITIES;

MICROGLIAL ACTIVATION; CEREBRAL-ISCHEMIA; MICE

AB Neurodegeneration after traumatic brain injury is facilitated by innate and adaptive immunity and can be harnessed to affect brain repair. In mice subjected to controlled cortical impact (CCI), we show that treatment with granulocyte macrophage colony stimulating factor (GM-CSF) affects regulatory T cell numbers in the cervical lymph nodes coincident with decreased lesion volumes and increased cortical tissue sparing. This paralleled increases in neurofilament and diminished reactive microglial staining. Transcriptomic analysis showed that GM-CSF induces robust immune neuroprotective responses seven days following CCI. Together, these results support the therapeutic potential of GM-CSF for TBI. (C) 2014 Elsevier B.V. All rights reserved.

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TI Methylene Blue Attenuates Traumatic Brain Injury-Associated

Neuroinflammation and Acute Depressive-Like Behavior in Mice

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE intervention; cytokines; fluid percussion injury; recovery; microglia

ID QUINOLINIC ACID; NITRIC-OXIDE; AGED MICE; COGNITIVE IMPAIRMENT;

OXIDATIVE STRESS; WORKING-MEMORY; HEAD-INJURY; INFLAMMATION; ACTIVATION;

EXPRESSION

AB Traumatic brain injury (TBI) is associated with cerebral edema, blood brain barrier breakdown, and neuroinflammation that contribute to the degree of injury severity and functional recovery. Unfortunately, there are no effective proactive treatments for limiting immediate or long-term consequences of TBI. Therefore, the objective of this study was to determine the efficacy of methylene blue (MB), an antioxidant agent, in reducing inflammation and behavioral complications associated with a diffuse brain injury. Here we show that immediate MB infusion (intravenous; 15-30 minutes after TBI) reduced cerebral edema, attenuated microglial activation and reduced neuroinflammation, and improved behavioral recovery after midline fluid percussion injury in mice. Specifically, TBI-associated edema and inflammatory gene expression in the hippocampus were significantly reduced by MB at 1 d post injury. Moreover, MB intervention attenuated TBI-induced inflammatory gene expression (interleukin [IL]-1 beta, tumor necrosis factor alpha) in enriched microglia/macrophages 1 d post injury. Cell culture experiments with lipopolysaccharide-activated BV2 microglia confirmed that MB treatment directly reduced IL-1 beta and increased IL-10 messenger ribonucleic acid in microglia. Last, functional recovery and depressive-like behavior were assessed up to one week after TBI. MB intervention did not prevent TBI-induced reductions in body weight or motor coordination 1-7 d post injury. Nonetheless, MB attenuated the development of acute depressive-like behavior at 7 d post injury. Taken together, immediate intervention with MB was effective in reducing neuroinflammation and improving behavioral recovery after diffuse brain injury. Thus, MB intervention may reduce life-threatening complications of TBI, including edema and neuroinflammation, and protect against the development of neuropsychiatric complications.

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TI Neuropathology of Traumatic Brain Injury: Comparison of Penetrating,

Nonpenetrating Direct Impact and Explosive Blast Etiologies

SO SEMINARS IN NEUROLOGY

LA English

DT Article

DE traumatic brain Injury; neuropathology; penetrating injury; closed head

injury; explosive blast; concussion; microglia; astrocytes; diffuse

axonal injury

ID AMYLOID PRECURSOR PROTEIN; HEAD-INJURY; AXONAL INJURY;

ALZHEIMERS-DISEASE; GUNSHOT WOUNDS; BETA-APP; MILD; DAMAGE; RISK;

SPECTROSCOPY

AB The neuropathology of traumatic brain injury (TBI) from various causes in humans is not as yet fully understood. The authors review and compare the known neuropathology in humans with severe, moderate, and mild TBI (mTBI) from nonpenetrating closed head injury (CHI) from blunt impacts and explosive blasts, as well as penetrating head injury (PHI). Penetrating head injury and CHI that are moderate to severe are more likely than mTBI to cause gross disruption of the cerebral vasculature. Axonal injury is classically exhibited as diffuse axonal injury (DAI) in severe to moderate CHI. Diffuse axonal injury is also prevalent in PHI. It is less so in mTBI. There may be a unique pattern of periventricular axonal injury in explosive blast mTBI. Neuronal injury is more prevalent in PHI and moderate to severe CHI than mTBI. Astrocyte and microglial activation and proliferation are found in all forms of animal TBI models and in severe to moderate TBI in humans. Their activation in mTBI in the human brain has not yet been studied.

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TI Neuroinflammation and brain atrophy in former NFL players: An <i>in

vivo</i> multimodal imaging pilot study

SO NEUROBIOLOGY OF DISEASE

LA English

DT Article

DE Mild traumatic brain injury; Translocator protein; Neuroinflammation;

Microglia; Molecular neuroimaging

ID PROFESSIONAL FOOTBALL PLAYERS; PROTEIN 18 KDA; PERIPHERAL

BENZODIAZEPINE-RECEPTOR; CHRONIC TRAUMATIC ENCEPHALOPATHY; RADIOLIGAND

BINDING; LEAGUE PLAYERS; RECURRENT CONCUSSION; PET RADIOLIGAND; HEAD

IMPACTS; INJURY

AB There are growing concerns about potential delayed, neuropsychiatric consequences (e.g, cognitive decline, mood or anxiety disorders) of sports-related traumatic brain injury (TBI). Autopsy studies of brains from a limited number of former athletes have described characteristic, pathologic changes of chronic traumatic encephalopathy (CTE) leading to questions about the relationship between these pathologic and the neuropsychiatric disturbances seen in former athletes. Research in this area will depend on in vivo methods that characterize molecular changes in the brain, linking CTE and other sports-related pathologies with delayed emergence of neuropsychiatric symptoms. In this pilot project we studied former National Football League (NFL) players using new neuroimaging techniques and clinical measures of cognitive functioning. We hypothesized that former NFL players would show molecular and structural changes in medial temporal and parietal lobe structures as well as specific cognitive deficits, namely those of verbal learning and memory. We observed a significant increase in binding of [C-11]DPA-713 to the translocator protein (TSPO), a marker of brain injury and repair, in several brain regions, such as the supramarginal gyrus and right amygdala, in 9 former NFL players compared to 9 age-matched, healthy controls. We also observed significant atrophy of the right hippocampus. Finally, we report that these same former players had varied performance on a test of verbal learning and memory, suggesting that these molecular and pathologic changes may play a role in cognitive decline. These results suggest that localized brain injury and repair, indicated by increased [C-11]DPA-713 binding to TSPO, may be linked to history of NFL play. [11C]DPA-713 PET is a promising new tool that can be used in future study design to examine further the relationship between TSPO expression in brain injury and repair, selective regional brain atrophy, and the potential link to deficits in verbal learning and memory after NFL play. (C) 2014 Elsevier Inc. All rights reserved.

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FU NIH [5R21MH082277, 5R01MH092443, R01EB012547, 5T32EB006351,

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TI Microglia in action: how aging and injury can change the brain's

guardians

SO FRONTIERS IN CELLULAR NEUROSCIENCE

LA English

DT Article

DE microglia; stroke; traumatic brain injury; inflammation; aging

ID ACTIVATED MICROGLIA; RESIDENT MICROGLIA; HEMATOPOIETIC STEM; COMMON

VARIANTS; CELL-DEATH; IN-VITRO; CNS; REVEALS; MECHANISM; FATE

AB Neuroinflarnmation, the inflammatory response in the central nervous system (CNS), is a major determinant of neuronal function and survival during aging and disease progression. Microglia, as the resident tissue-macrophages of the brain, provide constant support to surrounding neurons in healthy brain. Upon any stress signal (such as trauma, ischemia, inflammation) they are one of the first cells to react. Local and/or peripheral signals determine microglia stress response, which can vary within a continuum of states from beneficial to detrimental for neuronal survival, and can be shaped by aging and previous insults. In this review, we discuss the roles of microglia upon an ischemic or traumatic injury, and give our perspective how aging may contribute to microglia behavior in the injured brain. We speculate that a deeper understanding of specific microglia identities will pave the way to develop more potent therapeutics to treat the diseases of aging brain.

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TI Therapeutic Efficacy of Neuro AiD™ (MLC 601), a Traditional Chinese

Medicine, in Experimental Traumatic Brain Injury

SO JOURNAL OF NEUROIMMUNE PHARMACOLOGY

LA English

DT Article

DE Astragalosides; Traumatic brain injury; Microglia; Tumor necrosis

factor-alpha

ID MOTOR-ACTIVITY; ADULT-RAT; STROKE; MICROGLIA; EXPRESSION;

SYNAPTOGENESIS; ANGIOGENESIS; ETANERCEPT; INHIBITION; ACTIVATION

AB Traumatic brain injury (TBI) causes increased release of several mediators from injured and dead cells and elicits microglial activation. Activated microglia change their morphology, migrate to injury sites, and release tumor necrosis factor-alpha (TNF-alpha) and others. In this study we used a controlled fluid percussion injury model of TBI in the rat to determine whether early (4 h post-injury) or late (4 days post-injury) treatment with MLC 601, a Traditional Chinese Medicine, would affect microglial activation and improve recovery. MLC 601 was chosen for this study because its herbal component MLC 901 was beneficial in treating TBI in rats. Herein, rats with induced TBI were treated with MLC 601 (0.2-0.8 mg/kg) 1 h (early treatment) or 4 day post-injury (late treatment) and then injected once daily for consecutive 2 days. Acute neurological and motor deficits were assessed in all rats the day before and 4 days after early MLC 601 treatment. An immunofluorescence microscopy method was used to count the numbers of the cells colocalized with neuron- and apoptosis-specific markers, and the cells colocalized with microglia- and TNF-alpha-specific markers, in the contused brain regions 4 days post-injury. An immunohistochemistry method was used to evaluate both the number and the morphological transformation of microglia in the injured areas. It was found that early treatment with MLC 601 had better effects in reducing TBI-induced cerebral contusion than did the late therapy with MLC 601. Cerebral contusion caused by TBI was associated with neurological motor deficits, brain apoptosis, and activated microglia (e.g., microgliosis, amoeboid microglia, and microglial overexpression of TNF-alpha), which all were significantly attenuated by MLC 601 therapy. Our data suggest that MLC 601 is a promising agent for treatment of TBI in rats.

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TI HDAC inhibition prevents white matter injury by modulating

microglia/macrophage polarization through the GSK3β/PTEN/Akt axis

SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF

AMERICA

LA English

DT Article

DE traumatic brain injury; oligodendrocyte; microglial polarization;

myelination; inflammation

ID TRAUMATIC BRAIN-INJURY; MICROGLIA; ACTIVATION; NEUROPROTECTION;

DEGENERATION; INFLAMMATION; CHALLENGES; ISCHEMIA; DYNAMICS; DAMAGE

AB Severe traumatic brain injury (TBI) elicits destruction of both gray and white matter, which is exacerbated by secondary proinflammatory responses. Although white matter injury (WMI) is strongly correlated with poor neurological status, the maintenance of white matter integrity is poorly understood, and no current therapies protect both gray and white matter. One candidate approach that may fulfill this role is inhibition of class I/II histone deacetylases (HDACs). Here we demonstrate that the HDAC inhibitor Scriptaid protects white matter up to 35 d after TBI, as shown by reductions in abnormally dephosphorylated neurofilament protein, increases in myelin basic protein, anatomic preservation of myelinated axons, and improved nerve conduction. Furthermore, Scriptaid shifted microglia/macrophage polarization toward the protective M2 phenotype and mitigated inflammation. In primary cocultures of microglia and oligodendrocytes, Scriptaid increased expression of microglial glycogen synthase kinase 3 beta (GSK3 beta), which phosphorylated and inactivated phosphatase and tensin homologue (PTEN), thereby enhancing phosphatidylinositide 3-kinases (PI3K)/Akt signaling and polarizing microglia toward M2. The increase in GSK3 beta in microglia and their phenotypic switch to M2 was associated with increased preservation of neighboring oligodendrocytes. These findings are consistent with recent findings that microglial phenotypic switching modulates white matter repair and axonal remyelination and highlight a previously unexplored role for HDAC activity in this process. Furthermore, the functions of GSK3 beta may be more subtle than previously thought, in that GSK3 beta can modulate microglial functions via the PTEN/PI3K/Akt signaling pathway and preserve white matter homeostasis. Thus, inhibition of HDACs in microglia is a potential future therapy in TBI and other neurological conditions with white matter destruction.

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TI Microglia: dismantling and rebuilding circuits after acute neurological

injury

SO METABOLIC BRAIN DISEASE

LA English

DT Article

DE Microglia; Synaptic stripping; Neurological disease; Traumatic brain

injury; Epilepsy

ID TRAUMATIC BRAIN-INJURY; ALZHEIMERS-DISEASE; MULTIPLE ROLES; CELLS;

ACTIVATION; COMPLEMENT; ASTROCYTES; NEUROTOXICITY; FRACTALKINE; SYSTEM

AB The brain is comprised of neurons and its support system including astrocytes, glial cells and microglia, thereby forming neurovascular units. Neurons require support from glial cells to establish and maintain functional circuits, but microglia are often overlooked. Microglia function as the immune cell of the central nervous system, acting to monitor the microenvironment for changes in signaling, pathogens and injury. More recently, other functional roles for microglia within the healthy brain have been identified, including regulating synapse formation, elimination and function. This review aims to highlight and discuss these alternate microglial roles in the healthy and in contrast, diseased brain with a focus on two acute neurological diseases, traumatic brain injury and epilepsy. In these conditions, microglial roles in synaptic stripping and stabilization as part of neuronal:glial interactions may position them as mediators of the transition between injury-induced circuit dismantling and subsequent reorganization. Increased understanding of microglia roles could identify therapeutic targets to mitigate the consequences of neurological disease.

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TI The role of the microglia in acute CNS injury

SO METABOLIC BRAIN DISEASE

LA English

DT Review

DE Microglia; Inflammation; Brain; Spinal cord; Stroke; Trauma

ID TRAUMATIC BRAIN-INJURY; SPINAL-CORD-INJURY; FOCAL CEREBRAL-ISCHEMIA;

TUMOR-NECROSIS-FACTOR; NITRIC-OXIDE SYNTHASE; ACTIVATED PARENCHYMAL

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BLOOD-DERIVED MACROPHAGES; CENTRAL-NERVOUS-SYSTEM; GROWTH-FACTOR-BETA

AB Microglia are considered the brain's resident immune cell involved in immune defense, immunocompetence, and phagocytosis. They maintain tissue homeostasis within the brain and spinal cord under normal condition and serves as its initial host defense system. However, when the central nervous system (CNS) faces injury, microglia respond through signaling molecules expressed or released by neighboring cells. Microglial responses are dual in nature. They induce a nonspecific immune response that may exacerbate CNS injury, especially in the acute stages, but are also essential to CNS recovery and repair. The full range of microglial mechanisms have yet to be clarified, but there is accumulating knowledge about microglial activation in acute CNS injury. Microglial responses require hours to days to fully develop, and may present a therapeutic target for intervention with a much longer window of opportunity compare to other neurological treatments. The challenge will be to find ways to selectively suppress the deleterious effects of microglial activation without compromising its beneficial functions. This review aims to provide an overview of the recent progress relating on the deleterious and beneficial effect of microglia in the setting of acute CNS injury and the potential therapeutic intervention against microglial activation to CNS injury.

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TI Attenuation of traumatic brain injury-induced cognitive impairment in

mice by targeting increased cytokine levels with a small molecule

experimental therapeutic

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Cytokines; Glia; Interleukin; Neuroinflammation; Drug discovery;

Microglia; Astrocytes; Traumatic brain injury; Closed head injury;

Cognitive dysfunction

ID PROINFLAMMATORY CYTOKINE; ALZHEIMERS-DISEASE; UP-REGULATION; MOUSE

MODEL; MICROGLIAL ACTIVATION; SYNAPTIC DYSFUNCTION; BEHAVIORAL DEFICITS;

GLIAL ACTIVATION; NEUROINFLAMMATION; DRUG

AB Background: Evidence from clinical studies and preclinical animal models suggests that proinflammatory cytokine overproduction is a potential driving force for pathology progression in traumatic brain injury (TBI). This raises the possibility that selective targeting of the overactive cytokine response, a component of the neuroinflammation that contributes to neuronal dysfunction, may be a useful therapeutic approach. MW151 is a CNS-penetrant, small molecule experimental therapeutic that selectively restores injury-or disease-induced overproduction of proinflammatory cytokines towards homeostasis. We previously reported that MW151 administered post-injury (p.i.) is efficacious in a closed head injury (CHI) model of diffuse TBI in mice. Here we test dose dependence of MW151 to suppress the target mechanism (proinflammatory cytokine up-regulation), and explore the therapeutic window for MW151 efficacy.

Methods: We examined suppression of the acute cytokine surge when MW151 was administered at different times post-injury and the dose-dependence of cytokine suppression. We also tested a more prolonged treatment with MW151 over the first 7 days post-injury and measured the effects on cognitive impairment and glial activation.

Results: MW151 administered up to 6 h post-injury suppressed the acute cytokine surge, in a dose-dependent manner. Administration of MW151 over the first 7 days post-injury rescues the CHI-induced cognitive impairment and reduces glial activation in the focus area of the CHI.

Conclusions: Our results identify a clinically relevant time window post-CHI during which MW151 effectively restores cytokine production back towards normal, with a resultant attenuation of downstream cognitive impairment.

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TI Closed Head Injury in an Age-Related Alzheimer Mouse Model Leads to an

Altered Neuroinflammatory Response and Persistent Cognitive Impairment

SO JOURNAL OF NEUROSCIENCE

LA English

DT Article

DE amyloid plaque; astrocytes; cytokines; microglia; neuroinflammation;

traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; AMYLOID PRECURSOR PROTEIN; A-BETA DEPOSITION;

EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS; ATTENUATES SYNAPTIC

DYSFUNCTION; CYTOKINE UP-REGULATION; GENE-TARGETED MICE; PROINFLAMMATORY

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AB Epidemiological studies have associated increased risk of Alzheimer's disease (AD)-related clinical symptoms with a medical history of head injury. Currently, little is known about pathophysiology mechanisms linked to this association. Persistent neuroinflammation is one outcome observed in patients after a single head injury. Neuroinflammation is also present early in relevant brain regions during AD pathology progression. In addition, previous mechanistic studies in animal models link neuroinflammation as a contributor to neuropathology and cognitive impairment in traumatic brain injury (TBI) or AD-related models. Therefore, we explored the potential interplay of neuroinflammatory responses in TBI and AD by analysis of the temporal neuroinflammatory changes after TBI in an AD model, the APP/PS1 knock-in (KI) mouse. Discrete temporal aspects of astrocyte, cytokine, and chemokine responses in the injured KI mice were delayed compared with the injured wild-type mice, with a peak neuroinflammatory response in the injured KI mice occurring at 7 d after injury. The neuroinflammatory responses were more persistent in the injured KI mice, leading to a chronic neuroinflammation. At late time points after injury, KI mice exhibited a significant impairment in radial arm water maze performance compared with sham KI mice or injured wild-type mice. Intervention with a small-molecule experimental therapeutic (MW151) that selectively attenuates proinflammatory cytokine production yielded improved cognitive behavior outcomes, consistent with a link between neuroinflammatory responses and altered risk for AD-associated pathology changes with head injury.

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TI The pathophysiology of repetitive concussive traumatic brain injury in

experimental models; new developments and open questions

SO MOLECULAR AND CELLULAR NEUROSCIENCE

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DT Article

DE Concussion; Traumatic brain injury; Traumatic axonal injury;

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ID AMYLOID PROTEIN DEPOSITION; AXONAL INJURY; MOUSE MODEL;

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AB In recent years, there has been an increasing interest in the pathophysiology of repetitive concussive traumatic brain injury (rcTBI) in large part due to the association with dramatic cases of progressive neurological deterioration in professional athletes, military personnel, and others. However, our understanding of the pathophysiology of rcTBI is less advanced than for more severe brain injuries. Most prominently, the mechanisms underlying traumatic axonal injury, microglial activation, amyloid-beta accumulation, and progressive tau pathology are not yet known. In addition, the role of injury to dendritic spine cytoskeletal structures, vascular reactivity impairments, and microthrombi are intriguing and subjects of ongoing inquiry. Methods for quantitative analysis of axonal injury, dendritic injury, and synaptic loss need to be refined for the field to move forward in a rigorous fashion. We and others are attempting to develop translational approaches to assess these specific pathophysiological events in both animals and humans to facilitate clinically relevant pharmacodynamic assessments of candidate therapeutics. In this article, we review and discuss several of the recent experimental results from our lab and others. We include new initial data describing the difficulty in modeling progressive tau pathology in experimental rcTBI, and results demonstrating that sertraline can alleviate social interaction deficits and depressive-like behaviors following experimental rcTBI plus foot shock stress. Furthermore, we propose a discrete set of open, experimentally tractable questions that may serve as a framework for future investigations. In addition, we also raise several important questions that are less experimentally tractable at this time, in hopes that they may stimulate future methodological developments to address them. This article is part of a Special Issue entitled "Traumatic Brain Injury". (C) 2015 Elsevier Inc All rights reserved.

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TI A single dose of PPARγ agonist pioglitazone reduces cortical oxidative

damage and microglial reaction following lateral fluid percussion brain

injury in rats

SO PROGRESS IN NEURO-PSYCHOPHARMACOLOGY & BIOLOGICAL PSYCHIATRY

LA English

DT Article

DE Inflammation; Oxidative stress; Pioglitazone; Rat; Traumatic brain

injury

ID PROLIFERATOR-ACTIVATED RECEPTORS; TRANSIENT FOCAL ISCHEMIA;

NECROSIS-FACTOR-ALPHA; EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS;

INDUCED STATUS EPILEPTICUS; NF-KAPPA-B; GLUTATHIONE-PEROXIDASE; NEURONAL

INJURY; HEAD-INJURY; PROVIDES NEUROPROTECTION

AB Neuroprotective actions of the peroxisome proliferator-activated receptor-gamma (PPAR gamma) agonists have been observed in various animal models of the brain injuries. In this study we examined the effects of a single dose of pioglitazone on oxidative and inflammatory parameters as well as on neurodegeneration and the edema formation in the rat parietal cortex following traumatic brain injury (TBI) induced by the lateral fluid percussion injury (LFPI) method. Pioglitazone was administered in a dose of 1 mg/kg at 10 min after the brain trauma. The animals of the control group were sham-operated and injected by vehicle. The rats were decapitated 24 h after LFPI and their parietal cortices were analyzed by biochemical and histological methods. Cortical edema was evaluated in rats sacrificed 48 h following TBI. Brain trauma caused statistically significant oxidative damage of lipids and proteins, an increase of glutathione peroxidase (GSH-Px) activity, the cyclooxygenase-2 (COX-2) overexpression, reactive astrocytosis, the microglia activation, neurodegeneration, and edema, but it did not influence the superoxide dismutase activity and the expressions of interleukin-1 beta, interleukin-6 and tumor necrosis factor-alpha in the rat parietal cortex. Pioglitazone significantly decreased the cortical lipid and protein oxidative damage, increased the GSH-Px activity and reduced microglial reaction. Although a certain degree of the TBI-induced COX-2 overexpression, neurodegeneration and edema decrease was detected in pioglitazone treated rats, it was not significant. In the injured animals, cortical reactive astrocytosis was unchanged by the tested PPAR gamma agonist. These findings demonstrate that pioglitazone, administered only in a single dose, early following LFPI, reduced cortical oxidative damage, increased antioxidant defense and had limited anti-inflammatory effect, suggesting the need for further studies of this drug in the treatment of TBI. (C) 2015 Elsevier Inc. All rights reserved.

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TI Cerebrospinal Fluid Markers of Macrophage and Lymphocyte Activation

After Traumatic Brain Injury in Children

SO PEDIATRIC CRITICAL CARE MEDICINE

LA English

DT Article

DE cluster of differentiation 163; ferritin; head injury; interleukin-2

receptor; macrophage activation syndrome; microglia

ID CONTROLLED CORTICAL IMPACT; HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS; CD163(+)

MACROPHAGES/MICROGLIA; MULTIPLE-SCLEROSIS; IMMATURE RAT; FERRITIN;

EXPRESSION; RESPONSES; ACCUMULATION; INTERLEUKIN-10

AB Objectives: The magnitude and role of the cellular immune response following pediatric traumatic brain injury remains unknown. We tested the hypothesis that macrophage/microglia and T-cell activation occurs following pediatric traumatic brain injury by measuring cerebrospinal fluid levels of soluble cluster of differentiation 163 and ferritin and soluble interleukin-2 receptor , respectively, and determined whether these biomarkers were associated with relevant clinical variables and outcome.

Design: Retrospective analysis of samples from an established, single-center cerebrospinal fluid bank.

Setting: PICU in a tertiary children's hospital.

Patients: Sixty-six pediatric patients after severe traumatic brain injury (Glasgow Coma Scale score < 8) who were 1 month to 16 years old and 17 control patients who were 1 month to 14 years old.

Interventions: None.

Measurements and Main Results: Cerebrospinal fluid levels of soluble cluster of differentiation 163, ferritin, and soluble interleukin-2 receptor were determined by enzyme-linked immunosorbent assay at two time points (t(1) = 17 10 hr; t(2) = 72 +/- 15 hr) for each traumatic brain injury patient. Cerebrospinal fluid levels of soluble cluster of differentiation 163, ferritin, and soluble interleukin-2 receptor after traumatic brain injury were compared with controls and analyzed for associations with age, patient sex, initial Glasgow Coma Scale score, diagnosis of abusive head trauma, the presence of hemorrhage on CT scan, and Glasgow Outcome Scale score. Cerebrospinal fluid level of soluble cluster of differentiation 163 was increased in traumatic brain injury patients at t(2) versus t(1) and controls (median, 95.4 ng/mL [interquartile range, 21.8-134.0 ng/mL] vs 31.0 ng/mL [5.7-77.7 ng/mL] and 27.8 ng/mL [19.1-43.1 ng/mL], respectively; p < 0.05). Cerebrospinal fluid level of ferritin was increased in traumatic brain injury patients at t(2) and t(1) versus controls (8.3 ng/mL [<7.5-19.8 ng/mL] and 8.9 ng/mL [<7.5-26.7 ng/mL] vs <7.5 ng/mL below lower limit of detection, respectively; p < 0.05). Cerebrospinal fluid levels of soluble interleukin-2 receptor in traumatic brain injury patients at t(2) and t(1) were not different versus controls. Multivariate regression revealed associations between high ferritin and age 4 years or younger, lower Glasgow Coma Scale score, abusive head trauma, and unfavorable Glasgow Outcome Scale score.

Conclusions: Children with traumatic brain injury demonstrate evidence for macrophage activation after traumatic brain injury, and in terms of cerebrospinal fluid ferritin, this appears more prominent with young age, initial injury severity, abusive head trauma, and unfavorable outcome. Further study is needed to determine whether biomarkers of macrophage activation may be used to discriminate between aberrant and adaptive immune responses and whether inflammation represents a therapeutic target after traumatic brain injury.

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TI Resolvins AT-D1 and E1 differentially impact functional outcome,

post-traumatic sleep, and microglial activation following diffuse brain

injury in the mouse

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

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DE TBI; Inflammation; Resolvins; Protectins; Sleep; Mouse; Behavior;

Aspirin-triggered resolvin

ID RAT MODEL; MICE; CYTOKINES; WAKE; WAKEFULNESS; PATHOPHYSIOLOGY;

NEUROPROTECTION; INFLAMMATION; EXPRESSION; MEDIATORS

AB Traumatic brain injury (TBI) is induced by mechanical forces which initiate a cascade of secondary injury processes, including inflammation. Therapies which resolve the inflammatory response may promote neural repair without exacerbating the primary injury. Specific derivatives of omega-3 fatty acids loosely grouped as specialized pro-resolving lipid mediators (SPMs) and termed resolvins promote the active resolution of inflammation. In the current study, we investigate the effect of two resolvin molecules, RvE1 and AT-RvD1, on post-traumatic sleep and functional outcome following diffuse TBI through modulation of the inflammatory response.

Adult, male C57BL/6 mice were injured using a midline fluid percussion injury (mFPI) model (6-10 min righting reflex time for brain-injured mice). Experimental groups included mFPI administered RvE1 (100 ng daily), AT-RvD1 (100 ng daily), or vehicle (sterile saline) and counterbalanced with uninjured sham mice. Resolvins or saline were administered daily for seven consecutive days beginning 3 days prior to TBI to evaluate proof-of-principle to improve outcome. Immediately following diffuse TBI, post-traumatic sleep was recorded for 24 h post-injury. For days 1-7 post-injury, motor outcome was assessed by rotarod. Cognitive function was measured at 6 days post-injury using novel object recognition (NOR). At 7 days post-injury, microglial activation was quantified using immunohistochemistry for Iba-1.

In the diffuse brain-injured mouse, AT-RvD1 treatment, but not RvE1, mitigated motor and cognitive deficits. RvEl treatment significantly increased post-traumatic sleep in brain-injured mice compared to all other groups. RvEl treated mice displayed a higher proportion of ramified microglia and lower proportion of activated rod microglia in the cortex compared to saline or AT-RvD1 treated brain-injured mice. Thus, RvEl treatment modulated post-traumatic sleep and the inflammatory response to TB!, albeit independently of improvement in motor and cognitive outcome as seen in AT-RvD1-treated mice. This suggests AT-RvD1 may impart functional benefit through mechanisms other than resolution of inflammation alone. (C) 2015 Elsevier Inc. All rights reserved.

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TI Neuroprotective effects of nitidine against traumatic CNS injury via

inhibiting microglia activation

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE Nitidine; Microglia; Traumatic brain injury; Spinal cord injury;

Neuroprotection

ID SPINAL-CORD-INJURY; OLFACTORY ENSHEATHING CELLS; NF-KAPPA-B; REACTIVE

MICROGLIA; K+ CHANNEL; EXPRESSION; RECOVERY; PATHWAY; NEUROTOXICITY;

KCNN4/KCA3.1

AB Glial cell response to injury has been well documented in the pathogenesis after traumatic brain injury (TBI) and spinal cord injury (SCI). Although microglia, the resident macrophages in the central nervous system (CNS), are responsible for clearing debris and toxic substances, excessive activation of these cells will lead to exacerbated secondary damage by releasing a variety of inflammatory and cytotoxic mediators and ultimately influence the subsequent repair after CNS injury. In fact, inhibition of microgliosis represents a therapeutic strategy for CNS trauma. We here showed that nitidine, a benzophenanthridine alkaloid, restricted reactive microgliosis and promoted CNS repair after traumatic injury. Nitidine was shown to prevent cultured microglia from LPS-induced reactive activation by regulation of ERK and NF-kappa B signaling pathway. Furthermore, the nitidine-mediated inhibition of microgliosis was also shown in injured brain and spinal cord, which significantly increased neuronal survival and decreased neural tissue damage after injury. Importantly, behavioral analysis revealed that nitidine-treated mice with SCI had improved functional recovery as assessed by Basso Mouse Scale and swimming test. Together, these findings indicated that nitidine increased CNS tissue sparing and improved functional recovery by attenuating reactive microgliosis, suggestive of the potential therapeutic benefit for CNS injury. (C) 2015 Elsevier Inc. All rights reserved.

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TI A New Rabbit Model of Pediatric Traumatic Brain Injury

SO JOURNAL OF NEUROTRAUMA

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DT Article

DE microglia; pediatric traumatic brain injury; motor; rabbit; cognition

ID CONTROLLED CORTICAL IMPACT; CENTRAL-NERVOUS-SYSTEM; PROTEIN 18 KDA;

WHITE-MATTER; HEAD-INJURY; CEREBRAL-PALSY; POSTNATAL-DEVELOPMENT;

COGNITIVE DEFICITS; MOTOR DEFICITS; AXONAL INJURY

AB Traumatic brain injury (TBI) is a common cause of disability in childhood, resulting in numerous physical, behavioral, and cognitive sequelae, which can influence development through the lifespan. The mechanisms by which TBI influences normal development and maturation remain largely unknown. Pediatric rodent models of TBI often do not demonstrate the spectrum of motor and cognitive deficits seen in patients. To address this problem, we developed a New Zealand white rabbit model of pediatric TBI that better mimics the neurological injury seen after TBI in children. On postnatal Day 5-7 (P5-7), rabbits were injured by a controlled cortical impact (6-mm impactor tip; 5.5 m/sec, 2-mm depth, 50-msec duration). Rabbits from the same litter served as naive (no injury) and sham (craniotomy alone) controls. Functional abilities and activity levels were measured 1 and 5 d after injury. Maturation level was monitored daily. We performed cognitive tests during P14-24 and sacrificed the animals at 1, 3, 7, and 21 d after injury to evaluate lesion volume and microglia. TBI kits exhibited delayed achievement of normal developmental milestones. They also demonstrated significant cognitive deficits, with lower percentage of correct alternation rate in the T-maze (n=9-15/group; p<0.001) and less discrimination between novel and old objects (p<0.001). Lesion volume increased from 16% at Day 3 to 30% at Day 7 after injury, indicating ongoing secondary injury. Activated microglia were noted at the injury site and also in white matter regions of the ipsilateral and contralateral hemispheres. The neurologic and histologic changes in this model are comparable to those reported clinically. Thus, this rabbit model provides a novel platform for evaluating neuroprotective therapies in pediatric TBI.

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TI Microglial priming and enhanced reactivity to secondary insult in aging,

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AB Glia of the central nervous system (CNS) help to maintain homeostasis in the brain and support efficient neuronal function. Microglia are innate immune cells of the brain that mediate responses to pathogens and injury. They have key roles in phagocytic clearing, surveying the local microenvironment and propagating inflammatory signals. An interruption in homeostasis induces a cascade of conserved adaptive responses in glia. This response involves biochemical, physiological and morphological changes and is associated with the production of cytokines and secondary mediators that influence synaptic plasticity, cognition and behavior. This reorganization of host priorities represents a beneficial response that is normally adaptive but may become maladaptive when the profile of microglia is compromised. For instance, microglia can develop a primed or pro-inflammatory mRNA, protein and morphological profile with aging, traumatic brain injury and neurodegenerative disease. As a result, primed microglia exhibit an exaggerated inflammatory response to secondary and sub-threshold challenges. Consequences of exaggerated inflammatory responses by microglia include the development of cognitive deficits, impaired synaptic plasticity and accelerated neurodegeneration. Moreover, impairments in regulatory systems in these circumstances may make microglia more resistant to negative feedback and important functions of glia can become compromised and dysfunctional. Overall, the purpose of this review is to discuss key concepts of microglial priming and immune-reactivity in the context of aging, traumatic CNS injury and neurodegenerative disease.

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TI Time-dependent effects of CX3CR1 in a mouse model of mild traumatic

brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE CX3CR1; TBI; Microglia; Cognitive function; Cytokines

ID CONTROLLED CORTICAL IMPACT; FOCAL CEREBRAL-ISCHEMIA; SPINAL-CORD-INJURY;

FRACTALKINE-RECEPTOR; MICROGLIAL ACTIVATION; ALZHEIMERS-DISEASE;

ALTERNATIVE ACTIVATION; CHEMOKINE FRACTALKINE; AMYLOID DEPOSITION;

MACROPHAGE SUBSETS

AB Background: Neuroinflammation is an important secondary mechanism that is a key mediator of the long-term consequences of neuronal injury that occur in traumatic brain injury (TBI). Microglia are highly plastic cells with dual roles in neuronal injury and recovery. Recent studies suggest that the chemokine fractalkine (CX3CL1, FKN) mediates neural/microglial interactions via its sole receptor CX3CR1. CX3CL1/CX3CR1 signaling modulates microglia activation, and depending upon the type and time of injury, either protects or exacerbates neurological diseases.

Methods: In this study, mice deficient in CX3CR1 were subjected to mild controlled cortical impact injury (CCI), a model of TBI. We evaluated the effects of genetic deletion of CX3CR1 on histopathology, cell death/survival, microglia activation, and cognitive function for 30 days post-injury.

Results: During the acute post-injury period (24 h-15 days), motor deficits, cell death, and neuronal cell loss were more profound in injured wild-type than in CX3CR1(-/-) mice. In contrast, during the chronic period of 30 days post-TBI, injured CX3CR1(-/-) mice exhibited greater cognitive dysfunction and increased neuronal death than wild-type mice. The protective and deleterious effects of CX3CR1 were associated with changes in microglia phenotypes; during the acute phase CX3CR1(-/-) mice showed a predominant anti-inflammatory M2 microglial response, with increased expression of Ym1, CD206, and TGF beta. In contrast, increased M1 phenotypic microglia markers, Marco, and CD68 were predominant at 30 days post-TBI.

Conclusion: Collectively, these novel data demonstrate a time-dependent role for CX3CL1/CX3CR1 signaling after TBI and suggest that the acute and chronic responses to mild TBI are modulated in part by distinct microglia phenotypes.

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TI Macrophage activation and its role in repair and pathology after spinal

cord injury

SO BRAIN RESEARCH

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DT Article

DE Microglia; Neuroinflammation; Regeneration; Axon; TLR; Alternative

activation; Wound; Monocyte; Traumatic brain injury; TBI; SCI;

Macrophage; Inflammation; M2; M1; Dieback; Retraction; Astrocyte;

Healing; Wound; Phenotype; Neurotrauma; Reactive oxygen species;

Arginase; Mannose; OPC; Oligodendrocyte; IL-10; LPS; IL-12; STAT6;

STAT3; SLAM; MARCO; Proliferation; ECM; Ym1; Fizz-1; VEGF; IL-6; IL-4;

Immune complex; Regulatory; M2b; M2c

ID MEDIATED AXONAL DIEBACK; TRAUMATIC BRAIN-INJURY; PPAR-GAMMA AGONIST;

INFLAMMATORY RESPONSE; ALTERNATIVE ACTIVATION; FUNCTIONAL RECOVERY;

WALLERIAN DEGENERATION; OXIDATIVE STRESS; APOPTOTIC CELLS; CNS

AB The injured spinal cord does not heal properly. In contrast, tissue repair and functional recovery occur after skin or muscle injuries. The reason for this dichotomy in wound repair is unclear but inflammation, and specifically macrophage activation, likely plays a key role. Macrophages have the ability to promote the repair of injured tissue by regulating transitions through different phase of the healing response. In the current review we compare and contrast the healing and inflammatory responses between spinal cord injuries and tissues that undergo complete wound resolution. Through this comparison, we identify key macrophage phenotypes that are inaptly triggered or absent after spinal cord injury and discuss spinal cord stimuli that contribute to this maladaptive response. Sequential activation of classic, pro-inflammatory, M1 macrophages and alternatively activated, M2a, M2b, and M2c macrophages occurs during normal healing and facilitates transitions through the inflammatory, proliferative, and remodeling phases of repair. In contrast, in the injured spinal cord, pro-inflammatory macrophages potentiate a prolonged inflammatory phase and remodeling is not properly initiated. The desynchronized macrophage activation after spinal cord injury is reminiscent of the inflammation present in chronic, non-healing wounds. By refining the role macrophages play in spinal cord injury repair we bring to light important areas for future neuroinflammation and neurotrauma research. This article is part of a Special Issue entitled SI: Spinal cord injury. (C) 2015 The Authors. Published by Elsevier B.V.

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TI Microglia processes associate with diffusely injured axons following

mild traumatic brain injury in the micro pig

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Mild traumatic brain injury; Diffuse axonal injury; Neuroinflammation;

Microglia; Micro pig

ID SYNAPSE INTERACTIONS; OPTIC-NERVE; ACTIVATION; INFLAMMATION; MATTER;

WHITE; NEUROINFLAMMATION; MACROPHAGES; ATROPHY; PATHOPHYSIOLOGY

AB Background: Mild traumatic brain injury (mTBI) is an all too common occurrence that exacts significant personal and societal costs. The pathophysiology of mTBI is complex, with reports routinely correlating diffuse axonal injury (DAI) with prolonged morbidity. Progressive chronic neuroinflammation has also recently been correlated to morbidity, however, the potential association between neuroinflammatory microglia and DAI is not well understood. The majority of studies exploring neuroinflammatory responses to TBI have focused on more chronic phases of injury involving phagocytosis associated with Wallerian change. Little, however, is known regarding the neuroinflammatory response seen acutely following diffuse mTBI and its potential relationship to early DAI. Additionally, while inflammation is drastically different in rodents compared to humans, pigs and humans share very similar inflammatory profiles and responses.

Methods: In the current study, we employed a modified central fluid percussion model in micro pigs. Using this model of diffuse mTBI, paired with various immunohistological endpoints, we assessed the potential association between acute thalamic DAI and neuroinflammation 6 h following injury.

Results: Injured micro pigs displayed substantial axonal damage reflected in the presence of APP+ proximal axonal swellings, which were particularly prominent in the thalamus. In companion, the same thalamic sites displayed extensive neuroinflammation, which was observed using Iba-1 immunohistochemistry. The physical relationship between microglia and DAI, assessed via confocal 3D analysis, revealed a dramatic increase in the number of Iba-1+ microglial processes that contacted APP+ proximal axonal swellings compared to uninjured myelinated thalamic axons in sham animals.

Conclusions: In aggregate, these studies reveal acute microglial process convergence on proximal axonal swellings undergoing DAI, an interaction not previously recognized in the literature. These findings transform our understanding of acute neuroinflammation following mTBI and may suggest its potential as a diagnostic and/or a therapeutic target.

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TI Administration of DHA Reduces Endoplasmic Reticulum Stress-Associated

Inflammation and Alters Microglial or Macrophage Activation in Traumatic

Brain Injury

SO ASN NEURO

LA English

DT Article

DE cortical contusion injury; docosahexaenoic acid; microglial

polarization; neuroinflammation; nuclear factor

kappa-light-chain-enhancer of activated B cells; secondary injury

ID MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS; DOCOSAHEXAENOIC ACID; ER

STRESS; RESPONSES; IMPACT; EXPRESSION; MODEL

AB We investigated the effects of the administration of docosahexaenoic acid (DHA) post-traumatic brain injury (TBI) on reducing neuroinflammation. TBI was induced by cortical contusion injury in Sprague Dawley rats. Either DHA (16mg/kg in dimethyl sulfoxide) or vehicle dimethyl sulfoxide (1ml/kg) was administered intraperitonially at 5min after TBI, followed by a daily dose for 3 to 21 days. TBI triggered activation of microglia or macrophages, detected by an increase of Iba1 positively stained microglia or macrophages in peri-lesion cortical tissues at 3, 7, and 21 days post-TBI. The inflammatory response was further characterized by expression of the proinflammatory marker CD16/32 and the anti-inflammatory marker CD206 in Iba1(+) microglia or macrophages. DHA-treated brains showed significantly fewer CD16/32(+) microglia or macrophages, but an increased CD206(+) phagocytic microglial or macrophage population. Additionally, DHA treatment revealed a shift in microglial or macrophage morphology from the activated, amoeboid-like state into the more permissive, surveillant state. Furthermore, activated Iba1(+) microglial or macrophages were associated with neurons expressing the endoplasmic reticulum (ER) stress marker CHOP at 3 days post-TBI, and the administration of DHA post-TBI concurrently reduced ER stress and the associated activation of Iba1(+) microglial or macrophages. There was a decrease in nuclear translocation of activated nuclear factor kappa-light-chain-enhancer of activated B cells protein at 3 days in DHA-treated tissue and reduced neuronal degeneration in DHA-treated brains at 3, 7, and 21 days after TBI. In summary, our study demonstrated that TBI mediated inflammatory responses are associated with increased neuronal ER stress and subsequent activation of microglia or macrophages. DHA administration reduced neuronal ER stress and subsequent association with microglial or macrophage polarization after TBI, demonstrating its therapeutic potential to ameliorate TBI-induced cellular pathology.

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TI Age decreases macrophage IL-10 expression: Implications for functional

recovery and tissue repair in spinal cord injury

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Microglia; Contusion; Brain; Monocyte: IL-12p40; IL-12; Neuroprotection;

Inflammation; Macrophage polarization; Macrophage polarity; Traumatic;

Neurotrauma; Aged; Aging; Adult; Digigait; Grid walk; Locomotor; BMS

ID MONOCYTE-DERIVED MACROPHAGES; ALTERNATIVE ACTIVATION; LOCOMOTOR

RECOVERY; SYNAPTIC PROTEINS; OXIDATIVE STRESS; DOWN-REGULATION;

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AB Macrophages with different activation states are present after spinal cord injury (SCI). M1 macrophages purportedly promote secondary injury processes while M2 cells support axon growth. The average age at the time of SCI has increased in recent decades, however, little is known about how different physiological factors contribute to macrophage activation states after SCI. Here we investigate the effect of age on IL-10, a key indicator of M2 macrophage activation. Following mild-moderate SCI in 4 and 14 month old (MO) mice we detected significantly reduced IL-10 expression with age in the injured spinal cord. Specifically, CD86/IL-10 positive macrophages, also known as M2b or regulatory macrophages, were reduced in 14 vs. 4 MO SCI animals. This age-dependent shift in macrophage phenotype was associated with impaired functional recovery and enhanced tissue damage in 14-month-old SCI mice. In vitro, M2b macrophages release anti-inflammatory cytokines without causing neurotoxicity, suggesting that imbalances in the M2b response in 14-month-old mice may be contributing to secondary injury processes. Our data indicate that age is an important factor that regulates SCI inflammation and recovery even to mild-moderate injury. Further, alterations in macrophage activation states may contribute to recovery and we have identified the M2b phenotype as a potential target for therapeutic intervention. (C) 2015 Elsevier Inc. All rights reserved.

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TI Expression of Nogo receptor 1 in microglia during development and

following traumatic brain injury

SO BRAIN RESEARCH

LA English

DT Article

DE NgR1; Microglia; Expression; Development; Traumatic brain injury

ID OUTGROWTH INHIBITOR; FAMILY-MEMBER; MYELIN; ADHESION; NEUROGENESIS;

GLYCOPROTEIN; MIGRATION; CELLS; DEGENERATION; CORECEPTOR

AB As the receptor of myelin associated inhibitory factors Nogo receptor 1 (NgR1) plays an important role in central nervous system (CNS) injury and regeneration. It is found that NgR1 complex acts in neurons to transduce the signals intracelluarly including induction of growth cone collapse, inhibition of axonal regeneration and regulation of nerve inflammation. In recent studies, NgR1 has also been found to be expressed in the microglia. However, NgR1 expressed in microglia in the developing nervous systems and following CNS injury have not been widely investigated. In this study, we detected the expression and cellular localization of NgR1 in microglia during development and following traumatic brain injury (TBI) in mice. The results showed that NgR1 was mainly expressed in microglia during embryonic and postnatal periods. The expression levels peaked at P4 and decreased thereafter into adulthood, while increased significantly with aging representatively at 17 mo. On the other hand, there was no significant difference in the number of double positive NgR1(+)Iba1(+) cells between normal and TBI group. In summary, we first detected the expression of NgR1 in microglia during development and found that NgR1 protein expression increased significantly in microglia with aging. These findings will contribute to make a foundation for subsequent study about the role of NgR1 expressed in microglia on the CNS disorders. (C) 2015 Elsevier B.V. All rights reserved.

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TI S100B inhibition reduces behavioral and pathologic changes in

experimental traumatic brain injury

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE AGER; behavior (rodent); brain trauma; microglia; neurodegeneration;

neuroprotection; S100B

ID FLUID-PERCUSSION INJURY; GLYCATION END-PRODUCTS; NEURONAL SURVIVAL;

TRANSGENIC MICE; PROTEIN S100B; RAGE; RECEPTOR; ACTIVATION; S100-BETA;

EXPRESSION

AB Neuroinflammation following traumatic brain injury (TBI) is increasingly recognized to contribute to chronic tissue loss and neurologic dysfunction. Circulating levels of S100B increase after TBI and have been used as a biomarker. S100B is produced by activated astrocytes and can promote microglial activation; signaling by S100B through interaction with the multiligand advanced glycation end product-specific receptor (AGER) has been implicated in brain injury and microglial activation during chronic neurodegeneration. We examined the effects of S100B inhibition in a controlled cortical impact model, using S100B knockout mice or administration of neutralizing S100B antibody. Both interventions significantly reduced TBI-induced lesion volume, improved retention memory function, and attenuated microglial activation. The neutralizing antibody also significantly reduced sensorimotor deficits and improved neuronal survival in the cortex. However, S100B did not alter microglial activation in BV2 cells or primary microglial cultures stimulated by lipopolysaccharide or interferon gamma. Further, proximity ligation assays did not support direct interaction in the brain between S100B and AGER following TBI. Future studies are needed to elucidate specific pathways underlying S100B-mediated neuroinflammatory actions after TBI. Our results strongly implicate S100B in TBI-induced neuroinflammation, cell loss, and neurologic dysfunction, thereby indicating that it is a potential therapeutic target for TBI.

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AF Scott, Gregory

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TI Thalamic inflammation after brain trauma is associated with

thalamo-cortical white matter damage

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Microglia; Translocator protein; Positron emission tomography; Traumatic

brain injury; Traumatic axonal injury; PK11195; Thalamus

ID MICROGLIAL ACTIVATION; AXONAL INJURY; ISCHEMIC-STROKE; IN-VIVO;

NEUROINFLAMMATION; SINGLE

AB Background: Traumatic brain injury can trigger chronic neuroinflammation, which may predispose to neurodegeneration. Animal models and human pathological studies demonstrate persistent inflammation in the thalamus associated with axonal injury, but this relationship has never been shown in vivo.

Findings: Using [C-11]-PK11195 positron emission tomography, a marker of microglial activation, we previously demonstrated thalamic inflammation up to 17 years after traumatic brain injury. Here, we use diffusion MRI to estimate axonal injury and show that thalamic inflammation is correlated with thalamo-cortical tract damage.

Conclusions: These findings support a link between axonal damage and persistent inflammation after brain injury.

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TI Progesterone treatment reduces neuroinflammation, oxidative stress and

brain damage and improves long-term outcomes in a rat model of repeated

mild traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Concussion; Chronic traumatic encephalopathy; Animal model; DTI; MRI;

Treatment; Microglia; Macrophages; Astrogliosis; Lipid peroxidation

ID HIGH-DOSE PROGESTERONE; RECURRENT CONCUSSION; ANIMAL-MODEL; IN-VIVO;

INFLAMMATORY RESPONSE; COGNITIVE IMPAIRMENT; SYNAPTIC PLASTICITY;

LIPID-PEROXIDATION; CLINICAL-TRIAL; MOUSE MODEL

AB Background: Repeated mild traumatic brain injuries, such as concussions, may result in cumulative brain damage, neurodegeneration and other chronic neurological impairments. There are currently no clinically available treatment options known to prevent these consequences. However, growing evidence implicates neuroinflammation and oxidative stress in the pathogenesis of repetitive mild brain injuries; thus, these may represent potential therapeutic targets. Progesterone has been demonstrated to have potent anti-inflammatory and anti-oxidant properties after brain insult; therefore, here, we examined progesterone treatment in rats given repetitive mild brain injuries via the repeated mild fluid percussion injury model.

Methods: Male Long-Evans rats were assigned into four groups: sham injury + vehicle treatment, sham injury + progesterone treatment (8 mg/kg/day), repeated mild fluid percussion injuries + vehicle treatment, and repeated mild fluid percussion injuries + progesterone treatment. Rats were administered a total of three injuries, with each injury separated by 5 days. Treatment was initiated 1 h after the first injury, then administered daily for a total of 15 days. Rats underwent behavioural testing at 12-weeks post-treatment to assess cognition, motor function, anxiety and depression. Brains were then dissected for analysis of markers for neuroinflammation and oxidative stress. Ex vivo MRI was conducted in order to examine structural brain damage and white matter integrity.

Results: Repeated mild fluid percussion injuries + progesterone treatment rats showed significantly reduced cognitive and sensorimotor deficits compared to their vehicle-treated counterparts at 12-weeks post-treatment. Progesterone treatment significantly attenuated markers of neuroinflammation and oxidative stress in rats given repeated mild fluid percussion injuries, with concomitant reductions in grey and white matter damage as indicated by MRI.

Conclusions: These findings implicate neuroinflammation and oxidative stress in the pathophysiological aftermath of mild brain injuries and suggest that progesterone may be a viable treatment option to mitigate these effects and their detrimental consequences.

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NR 74

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PU BMC

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TI Long-Term Anesthetic-Dependent Hypoactivity after Repetitive Mild

Traumatic Brain Injuries in Adolescent Mice

SO DEVELOPMENTAL NEUROSCIENCE

LA English

DT Article

DE Mild traumatic brain injury; Impact; Concussion; Adolescent; Mouse;

Microglia; Behavior; Pathology; Anesthetic

ID SPORT-RELATED CONCUSSION; PROFESSIONAL SOCCER PLAYERS; SCHOOL FOOTBALL

PLAYERS; DEFAULT MODE NETWORK; STATES HIGH-SCHOOL; MOUSE-BRAIN;

PRECLINICAL RESEARCH; TRANSGENIC MICE; MALE RATS; VULNERABILITY

AB Recent evidence supports the hypothesis that repetitive mild traumatic brain injuries (rmTBIs) culminate in neurological impairments and chronic neurodegeneration, which have wide-ranging implications for patient management and return-to-play decisions for athletes. Adolescents show a high prevalence of sports-related head injuries and may be particularly vulnerable to rmTBIs due to ongoing brain maturation. However, it remains unclear whether rmTBIs, below the threshold for acute neuronal injury or symptomology, influence long-term outcomes. To address this issue, we first defined a very mild injury in adolescent mice (postnatal day 35) as evidenced by an increase in lba-1-labeled microglia in white matter in the acutely injured brain, in the absence of indices of cell death, axonal injury, and vasogenic edema. Using this level of injury severity and Avertin (2,2,2-tribromoethanol) as the anesthetic, we compared mice subjected to either a single mTBI or 2 rmTBIs, each separated by 48 h. Neurobehavioral assessments were conducted at 1 week and at 1 and 3 months postimpact. Mice subjected to rmTBIs showed transient anxiety and persistent and pronounced hypoactivity compared to sham control mice, alongside normal sensorimotor, cognitive, social, and emotional function. As isoflurane is more commonly used than Avertin in animal models of TBI, we next examined long-term outcomes after rmTBIs in mice that were anesthetized with this agent. However, there was no evidence of abnormal behaviors even with the addition of a third rnnTBI. To determine whether isoflurane may be neuroprotective, we compared the acute pathology after a single mTBI in mice anesthetized with either Avertin or isoflurane. Pathological findings were more pronounced in the group exposed to Avertin compared to the isoflurane group. These collective findings reveal distinct behavioral phenotypes (transient anxiety and prolonged hypoactivity) that emerge in response to rmTBIs. Our findings further suggest that selected anesthetics may confer early neuroprotection after rmTBIs, and as such mask long-term abnormal phenotypes that may otherwise emerge as a consequence of acute pathogenesis. (C) 2016 S. Karger AG, Basel

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TI Granulocyte-colony stimulating factor promotes brain repair following

traumatic brain injury by recruitment of microglia and increasing

neurotrophic factor expression

SO RESTORATIVE NEUROLOGY AND NEUROSCIENCE

LA English

DT Article

DE Granulocyte-colony stimulating factor; traumatic brain injury;

neuro-inflammation; neurogenesis; doublecortin; astrocytosis;

microgliosis; chimeric mice; green fluorescent protein

ID REVERSES COGNITIVE IMPAIRMENT; BONE-MARROW-CELLS; FACTOR G-CSF; CELLULAR

PROLIFERATION; FUNCTION RECOVERY; PURKINJE NEURONS; ISCHEMIC-STROKE;

NEUROGENESIS; ADULT; TRANSPLANTATION

AB Purpose: The overall objective was to elucidate cellular mechanisms by which G-CSF enhances recovery from traumatic brain injury in a hippocampal-dependent learning task.

Methods: Chimeric mice were prepared by transplanting bone marrow cells that express green fluorescent protein (GFP+) from a transgenic "green" mice into C57BL/6 mice. Two months later, the animals sustained mild controlled cortical impact (CCI) to the right frontal-parietal cortex, followed by G-CSF (100 [kg/kg) treatment for 3 consecutive days. The primary behavioral end-point was performance on the radial arm water maze (RAWM) assessed before and after CCI (days 7 and 14). Secondary endpoints included a), motor performance on a rotating cylinder (rotarod), b) measurement of microglial and astroglial response, c) hippocampal neurogenesis, and d) measures of neurotrophic factors (BDNF, GDNF) in brain homogenates.

Results: G-CSF treatment resulted in significantly better performance on the rotorod at one week, and in the RAWM after one and two weeks. The cellular changes found 2 wks after CCI in the G-CSF group included increased numbers of hippocampal newborn neurons as well as astrocytosis and microgliosis in striatum and frontal cortex on both sides of brain. GFP+ cells that co-labeled with Ibal (microglial marker) comprised a significant proportion of striatal microglia in G-CSF treated animals, indicating the capacity of G-CSF to increase microglial recruitment to the site of injury. Neurotrophic factors GDNF and BDNF, elaborated by activated microglia and astrocytes, were increased in G-CSF treated mice.

Conclusions: G-CSF serves as a neurotrophic factor that increases hippocampal neurogenesis (or enhances survival of new-born neurons), and activates astrocytes and microglia. In turn, these activated glia release a plethora of cytokines and neurotrophic factors that contribute, in a poorly understood cascade, to the brain's repair response. G-CSF also acts directly on bone marrow-derived cells to enhance recruitment of microglia to the site of CCI from circulating monocytes to the site of CCI.

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TI Differential detection of impact site versus rotational site injury by

magnetic resonance imaging and microglial morphology in an unrestrained

mild closed head injury model

SO JOURNAL OF NEUROCHEMISTRY

LA English

DT Article

DE confocal microscopy; traumatic brain injury; T2-weighted imaging;

neuroinflammation

ID TRAUMATIC BRAIN-INJURY; IN-VIVO; RAT; FRACTALKINE; ACTIVATION;

EXPRESSION; DISEASE; CELLS; MICE

AB Seventy-five percent of all traumatic brain injuries are mild and do not cause readily visible abnormalities on routine medical imaging making it difficult to predict which individuals will develop unwanted clinical sequelae. Microglia are brain-resident macrophages and early responders to brain insults. Their activation is associated with changes in morphology or expression of phenotypic markers including P2Y12 and major histocompatibility complex class II. Using a murine model of unrestrained mild closed head injury (mCHI), we used microglia as reporters of acute brain injury at sites of impact versus sites experiencing rotational stress 24h post-mCHI. Consistent with mild injury, a modest 20% reduction in P2Y12 expression was detected by quantitative real-time PCR (qPCR) analysis but only in the impacted region of the cortex. Furthermore, neither an influx of blood-derived immune cells nor changes in microglial expression of CD45, TREM1, TREM2, major histocompatibility complex class II or CD40 were detected. Using magnetic resonance imaging (MRI), small reductions in T2 weighted values were observed but only near the area of impact and without overt tissue damage (blood deposition, edema). Microglial morphology was quantified without cryosectioning artifacts using ScaleA(2) clarified brains from CX3CR1-green fluorescence protein (GFP) mice. The cortex rostral to the mCHI impact site receives greater rotational stress but neither MRI nor molecular markers of microglial activation showed significant changes from shams in this region. However, microglia in this rostral region did display signs of morphologic activation equivalent to that observed in severe CHI. Thus, mCHI-triggered rotational stress is sufficient to cause injuries undetectable by routine MRI that could result in altered microglial surveillance of brain homeostasis.

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TI Human Neural Stem Cell Transplantation-Mediated Alteration of

Microglial/Macrophage Phenotypes After Traumatic Brain Injury

SO CELL TRANSPLANTATION

LA English

DT Article

DE Neural stem cells (NSCs); Microglia; Traumatic brain injury (TBI);

Transplantation; Immunomodulation

ID INFLAMMATORY RESPONSE; MACROPHAGE ACTIVATION; STEM/PROGENITOR CELLS; M2

MICROGLIA; THERAPY; SYSTEM; NEUROINFLAMMATION; IMMUNOGENICITY;

POLARIZATION; REGENERATION

AB Neural stem cells (NSCs) promote recovery from brain trauma, but neuronal replacement is unlikely the sole underlying mechanism. We hypothesize that grafted NSCs enhance neural repair at least partially through modulating the host immune response after traumatic brain injury (TBI). C57BL/6 mice were intracerebrally injected with primed human NSCs (hNSCs) or vehicle 24 h after a severe controlled cortical impact injury. Six days after transplantation, brain tissues were collected for Western blot and immunohistochemical analyses. Observations included indicators of microglia/macrophage activation, M1 and M2 phenotypes, axonal injury detected by amyloid precursor protein (APP), lesion size, and the fate of grafted hNSCs. Animals receiving hNSC transplantation did not show significant decreases of brain lesion volumes compared to transplantation procedures with vehicle alone, but did show significantly reduced injury-dependent accumulation of APP. Furthermore, intracerebral transplantation of hNSCs reduced microglial activation as shown by a diminished intensity of Ibal immunostaining and a transition of microglia/macrophages toward the M2 anti-inflammatory phenotype. The latter was represented by an increase in the brain M2/M1 ratio and increases of M2 microglial proteins. These phenotypic switches were accompanied by the increased expression of anti-inflammatory interleukin-4 receptor alpha and decreased proinflammatory interferon-gamma receptor beta. Finally, grafted hNSCs mainly differentiated into neurons and were phagocytized by either M1 or M2 microglia/macrophages. Thus, intracerebral transplantation of primed hNSCs efficiently leads host microglia/macrophages toward an anti-inflammatory phenotype that presumably contributes to stem cell-mediated neuroprotective effects after severe TBI in mice.

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TI Microglia in the TBI brain: The good, the bad, and the dysregulated

SO EXPERIMENTAL NEUROLOGY

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DT Review

DE Traumatic brain injury; Neuroinflammation; Microglia; Macrophage;

Phenotype; M1-like; M2-like; Polarization; Neurodegeneration; Repair

ID CENTRAL-NERVOUS-SYSTEM; GROWTH-FACTOR-I; IMPROVES FUNCTIONAL RECOVERY;

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CELLS; SPINAL-CORD-INJURY; MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS;

HIPPOCAMPAL PROGENITOR CELLS; ALTERNATIVE M2 MACROPHAGES

AB As the major cellular component of the innate immune system in the central nervous system (CNS) and the first line of defense whenever injury or disease occurs, microglia play a critical role in neuroinflammation following a traumatic brain injury (TBI). In the injured brain microglia can produce neuroprotective factors, clear cellular debris and orchestrate neurorestorative processes that are beneficial for neurological recovery after TBI. However, microglia can also become dysregulated and can produce high levels of pro-inflammatory and cytotoxic mediators that hinder CNS repair and contribute to neuronal dysfunction and cell death. The dual role of microglial activation in promoting beneficial and detrimental effects on neurons may be accounted for by their polarization state and functional responses after injury. In this review article we discuss emerging research on microglial activation phenotypes in the context of acute brain injury, and the potential role of microglia in phenotype-specific neurorestorative processes such as neurogenesis, angiogenesis, oligodendrogenesis and regeneration. We also describe some of the known molecular mechanisms that regulate phenotype switching, and highlight new therapeutic approaches that alter microglial activation state balance to enhance long-term functional recovery after TBI. An improved understanding of the regulatory mechanisms that control microglial phenotypic shifts may advance our knowledge of post-injury recovery and repair, and provide opportunities for the development of novel therapeutic strategies for TBI. (C) 2015 Elsevier Inc. All rights reserved.

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TI Methylene blue exerts a neuroprotective effect against traumatic brain

injury by promoting autophagy and inhibiting microglial activation

SO MOLECULAR MEDICINE REPORTS

LA English

DT Article

DE methylene blue; traumatic brain injury; autophagy; microglia

ID IMPROVES FUNCTIONAL RECOVERY; EDEMA; MECHANISMS

AB Traumatic brain injury (TBI) leads to permanent neurological impairment, and methylene blue (MB) exerts central nervous system neuroprotective effects. However, only one previous study has investigated the effectiveness of MB in a controlled cortical impact injury model of TBI. In addition, the specific mechanisms underlying the effect of MB against TBI remain to be elucidated. Therefore, the present study investigated the neuroprotective effect of MB on TBI and the possible mechanisms involved. In a mouse model of TBI, the animals were randomly divided into sham, vehicle (normal saline) or MB groups. The treatment time-points were 24 and 72 h (acute phase of TBI), and 14 days (chronic phase of TBI) post-TBI. The brain water content (BWC), and levels of neuronal death, and autophagy were determined during the acute phase, and neurological deficit, injury volume and microglial activation were assessed at all time-points. The injured hemisphere BWC was significantly increased 24 h post-TBI, and this was attenuated following treatment with MB. There was a significantly higher number of surviving neurons in the MB group, compared with the Vehicle group at 24 and 72 h post-TBI. In the acute phase, the MB-treated animals exhibited significantly upregulated expression of Beclin 1 and increased LC3-II to LC3-I ratios, compared with the vehicle group, indicating an increased rate of autophagy. Neurological functional deficits, measured using the modified neurological severity score, were significantly lower in the acute phase in the MB-treated animals and cerebral lesion volumes in the MB-treated animals were significantly lower, compared with the other groups at all time-points. Microglia were activated 24 h after TBI, peaked at 72 h and persisted until 14 days after TBI. Although the number of Iba-1-positive cells in the vehicle and MB groups 24 h post-TBI were not significantly different, marked microglial inhibition was observed in the MB group 72 h and 14 days after -TBI. These results indicated that MB exerts a neuroprotective effect by increasing autophagy, decreasing brain edema and inhibiting microglial activation.

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TI The contribution of astrocytes and microglia to traumatic brain injury

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DT Review

ID CENTRAL-NERVOUS-SYSTEM; MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS;

FIBRILLARY ACIDIC PROTEIN; CONCISE GUIDE; REACTIVE ASTROCYTES; SCAR

FORMATION; BARRIER PERMEABILITY; WHITE-MATTER; HEAD-INJURY; RAT-BRAIN

AB Traumatic brain injury (TBI) represents a major cause of death and disability in developed countries. Brain injuries are highly heterogeneous and can also trigger other neurological complications, including epilepsy, depression and dementia. The initial injury often leads to the development of secondary sequelae; cellular hyperexcitability, vasogenic and cytotoxic oedema, hypoxia-ischaemia, oxidative stress and inflammation, all of which influence expansion of the primary lesion. It is widely known that inflammatory events in the brain following TBI contribute to the widespread cell death and chronic tissue degeneration. Neuroinflammation is a multifaceted response involving a number of cell types, both within the CNS and in the peripheral circulation. Astrocytes and microglia, cells of the CNS, are considered key players in initiating an inflammatory response after injury. These cells are capable of secreting various cytokines, chemokines and growth factors, and following injury to the CNS, undergo changes in morphology. Ultimately, these changes can influence the local microenvironment and thus determine the extent of damage and subsequent repair. This review will focus on the roles of microglia and astrocytes following TBI, highlighting some of the key processes, pathways and mediators involved in this response. Additionally, both the beneficial and the detrimental aspects of these cellular responses will be examined using evidence from animal models and human post-mortem TBI studies.

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TI MW151 Inhibited IL-1β Levels after Traumatic Brain Injury with No Effect

on Microglia Physiological Responses

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DT Article

ID ATTENUATES SYNAPTIC DYSFUNCTION; CYTOKINE UP-REGULATION; PROINFLAMMATORY

CYTOKINE; ALZHEIMERS-DISEASE; MOUSE MODEL; GLIAL ACTIVATION; P38-ALPHA

MAPK; INFLAMMATION; NEUROINFLAMMATION; PROGRESSION

AB A prevailing neuroinflammation hypothesis is that increased production of proinflammatory cytokines contributes to progressive neuropathology, secondary to the primary damage caused by a traumatic brain injury (TBI). In support of the hypothesis, post-injury interventions that inhibit the proinflammatory cytokine surge can attenuate the progressive pathology. However, other post-injury neuroinflammatory responses are key to endogenous recovery responses. Therefore, it is critical that pharmacological attenuation of detrimental or dysregulated neuroinflammatory processes avoid pan-suppression of inflammation. MW151 is a CNS-penetrant, small molecule experimental therapeutic that restores injuryor disease-induced overproduction of proinflammatory cytokines towards homeostasis without immunosuppression. Post-injury administration of MW151 in a closed head injury model of mild TBI suppressed acute cytokine up-regulation and downstream cognitive impairment. Here, we report results from a diffuse brain injury model in mice using midline fluid percussion. Low dose (0.5-5.0 mg/kg) administration of MW151 suppresses interleukin-1 beta (IL-1 beta) levels in the cortex while sparing reactive microglia and astrocyte responses. To probe molecular mechanisms, we used live cell imaging of the BV-2 microglia cell line to demonstrate that MW151 does not affect proliferation, migration, or phagocytosis of the cells. Our results provide insight into the roles of glial responses to brain injury and indicate the feasibility of using appropriate dosing for selective therapeutic modulation of injurious IL-1 beta increases while sparing other glial responses to injury.

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TI Neurovascular and Immuno-Imaging: From Mechanisms to Therapies.

Proceedings of the Inaugural Symposium

SO FRONTIERS IN NEUROSCIENCE

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DE neuroinflammation; multiple sclerosis; traumatic brain injury;

Alzheimer's disease; blood-brain barrier; microglia; myelin; two-photon

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ID BLOOD-BRAIN-BARRIER; CENTRAL-NERVOUS-SYSTEM; MULTIPLE-SCLEROSIS LESIONS;

FLUORESCENT-PROBES; BIOLOGICAL-SYSTEMS; HYDROGEN-SULFIDE; LIVING

SYSTEMS; DISEASE; ABNORMALITIES; TRANSITION

AB Breakthrough advances in intravital imaging have launched a new era for the study of dynamic interactions at the neurovascular interface in health and disease. The first Neurovascular and Immuno-Imaging Symposium was held at the Gladstone Institutes, University of California, San Francisco in March, 2015. This highly interactive symposium brought together a group of leading researchers who discussed how recent studies have unraveled fundamental biological mechanisms in diverse scientific fields such as neuroscience, immunology, and vascular biology, both under physiological and pathological conditions. These Proceedings highlight how advances in imaging technologies and their applications revolutionized our understanding of the communication between brain, immune, and vascular systems and identified novel targets for therapeutic intervention in neurological diseases.

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TI Role of Glia in Memory Deficits Following Traumatic Brain Injury:

Biomarkers of Glia Dysfunction

SO FRONTIERS IN INTEGRATIVE NEUROSCIENCE

LA English

DT Review

DE astrocytes; microglia; oligodendrocytes; traumtic brain injury (TBI);

biomarkers; MRS spectroscopy; memory impairment; gliosis

ID CORTICAL IMPACT INJURY; MITOCHONDRIAL DYSFUNCTION; AQUAPORIN-4

EXPRESSION; REACTIVE ASTROCYTES; N-ACETYLASPARTATE; DEPENDENT CHANGES;

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AB Historically, glial cells have been recognized as a structural component of the brain. However, it has become clear that glial cells are intimately involved in the complexities of neural networks and memory formations. Astrocytes, microglia, and oligodendrocytes have dynamic responsibilities which substantially impact neuronal function and activities. Moreover, the importance of glia following brain injury has come to the forefront in discussions to improve axonal regeneration and functional recovery. The numerous activities of glia following injury can either promote recovery or underlie the pathobiology of memory deficits. This review outlines the pathological states of glial cells which evolve from their positive supporting roles to those which disrupt synaptic function and neuroplasticity following injury. Evidence suggests that glial cells interact extensively with neurons both chemically and physically, reinforcing their role as pivotal for higher brain functions such as learning and memory. Collectively, this mini review surveys investigations of how glial dysfunction following brain injury can alter mechanisms of synaptic plasticity and how this may be related to an increased risk for persistent memory deficits. We also include recent findings, that demonstrate new molecular avenues for clinical biomarker discovery.

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TI Minocycline Transiently Reduces Microglia/Macrophage Activation but

Exacerbates Cognitive Deficits Following Repetitive Traumatic Brain

Injury in the Neonatal Rat

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DE Abusive head trauma; Cognition; Inflammation; Microglia;

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ID WHITE-MATTER INJURY; NONACCIDENTAL HEAD-INJURY; HYPOXIC-ISCHEMIC INJURY;

LONG-TERM POTENTIATION; CYTOCHROME-C RELEASE; MICROGLIAL ACTIVATION;

AXONAL INJURY; IMMATURE RAT; INTRACEREBRAL-HEMORRHAGE; RODENT MODEL

AB Elevated microglial/macrophage-associated biomarkers in the cerebrospinal fluid of infant victims of abusive head trauma (AHT) suggest that these cells play a role in the pathophysiology of the injury. In a model of AHT in 11-day-old rats, 3 impacts (24 hours apart) resulted in spatial learning and memory deficits and increased brain microglial/macrophage reactivity, traumatic axonal injury, neuronal degeneration, and cortical and white-matter atrophy. The antibiotic minocycline has been effective in decreasing injury-induced microglial/macrophage activation while simultaneously attenuating cellular and functional deficits in models of neonatal hypoxic ischemia, but the potential for this compound to rescue deficits after impact-based trauma to the immature brain remains unexplored. Acute minocycline administration in this model of AHT decreased microglial/macrophage reactivity in the corpus callosum of brain-injured animals at 3 days postinjury, but this effect was lost by 7 days postinjury. Additionally, minocycline treatment had no effect on traumatic axonal injury, neurodegeneration, tissue atrophy, or spatial learning deficits. Interestingly, minocycline-treated animals demonstrated exacerbated injury-induced spatial memory deficits. These results contrast with previous findings in other models of brain injury and suggest that minocycline is ineffective in reducing microglial/macrophage activation and ameliorating injury-induced deficits following repetitive neonatal traumatic brain injury.

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AF Savitz, Sean I.

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TI Concise Review: Cell Therapies for Stroke and Traumatic Brain Injury:

Targeting Microglia

SO STEM CELLS

LA English

DT Review

DE Stem cells; Stroke; Microglia; Brain injuries

ID ACTIVATED MICROGLIAL/MACROPHAGE RESPONSE; MARROW MONONUCLEAR-CELLS;

TRANSLOCATOR PROTEIN; 18 KDA; DIFFUSION-TENSOR; INFLAMMATION;

NEUROINFLAMMATION; NEURODEGENERATION; DYNAMICS; ATROPHY

AB We present a model hypothesis of how several types of cell therapies may target microglia as one of the principal cell types contributing to the inflammatory response after brain injury and discuss how imaging of brain inflammation could potentially be applied to develop biomarkers in patients with stroke and TBI enrolled into stem cell clinical trials.

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AU Fidan, E

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TI Repetitive Mild Traumatic Brain Injury in the Developing Brain: Effects

on Long-Term Functional Outcome and Neuropathology

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE axonal injury; development; head injury; microglia

ID CLOSED-HEAD INJURY; POSTTRAUMATIC-STRESS-DISORDER; FLUID PERCUSSION

INJURY; AXONAL INJURY; HIGH-SCHOOL; COGNITIVE IMPAIRMENT; NORMOBARIC

HYPEROXIA; NERVOUS-SYSTEM; RAT; RECOVERY

AB Although accumulating evidence suggests that repetitive mild TBI (rmTBI) may cause long-term cognitive dysfunction in adults, whether rmTBI causes similar deficits in the immature brain is unknown. Here we used an experimental model of rmTBI in the immature brain to answer this question. Post-natal day (PND) 18 rats were subjected to either one, two, or three mild TBIs (mTBI) or an equivalent number of sham insults 24 h apart. After one or two mTBIs or sham insults, histology was evaluated at 7 days. After three mTBIs or sham insults, motor (d1-5), cognitive (d11-92), and histological (d21-92) outcome was evaluated. At 7 days, silver degeneration staining revealed axonal argyrophilia in the external capsule and corpus callosum after a single mTBI, with a second impact increasing axonal injury. Iba-1 immunohistochemistry showed amoeboid shaped microglia within the amygdalae bilaterally after mTBI. After three mTBI, there were no differences in beam balance, Morris water maze, and elevated plus maze performance versus sham. The rmTBI rats, however, showed impairment in novel object recognition and fear conditioning. Axonal silver staining was observed only in the external capsule on d21. Iba-1 staining did not reveal activated microglia on d21 or d92. In conclusion, mTBI results in traumatic axonal injury and microglial activation in the immature brain with repeated impact exacerbating axonal injury. The rmTBI in the immature brain leads to long-term associative learning deficit in adulthood. Defining the mechanisms damage from rmTBI in the developing brain could be vital for identification of therapies for children.

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TI Lack of NG2 Exacerbates Neurological Outcome and Modulates Glial

Responses After Traumatic Brain Injury

SO GLIA

LA English

DT Article

DE astrocytes; microglia; macrophages; gliosis; CNS inflammation; Cxcl13

ID CHONDROITIN SULFATE PROTEOGLYCANS; OLIGODENDROCYTE PRECURSOR CELLS;

CORTICAL IMPACT INJURY; SPINAL-CORD-INJURY; ACTIVATED MICROGLIA;

LYMPHOID CHEMOKINE; NG2-POSITIVE CELLS; UP-REGULATION; PROLIFERATION;

MACROPHAGES

AB Traumatic brain injury (TBI) is a major cause of death and disability. The underlying pathophysiology is characterized by secondary processes including neuronal death and gliosis. To elucidate the role of the NG2 proteoglycan we investigated the response of NG2-knockout mice (NG2-KO) to TBI. Seven days after TBI behavioral analysis, brain damage volumetry and assessment of blood brain barrier integrity demonstrated an exacerbated response of NG2-KO compared to wild-type (WT) mice. Reactive astrocytes and expression of the reactive astrocyte and neurotoxicity marker Lcn2 (Lipocalin-2) were increased in the perilesional brain tissue of NG2-KO mice. In addition, microglia/macrophages with activated morphology were increased in number and mRNA expression of the M2 marker Arg1 (Arginase 1) was enhanced in NG2-KO mice. While TBI-induced expression of pro-inflammatory cytokine genes was unchanged between genotypes, PCR array screening revealed a marked TBI-induced up-regulation of the C-X-C motif chemokine 13 gene Cxcl13 in NG2-KO mice. CXCL13, known to attract immune cells to the inflamed brain, was expressed by activated perilesional microglia/macrophages seven days after TBI. Thirty days after TBI, NG2-KO mice still exhibited more pronounced neurological deficits than WT mice, up-regulation of Cxcl13, enhanced CD45+ leukocyte infiltration and a relative increase of activated Iba-1+/CD45+ microglia/macrophages. Our study demonstrates that lack of NG2 exacerbates the neurological outcome after TBI and associates with abnormal activation of astrocytes, microglia/macrophages and increased leukocyte recruitment to the injured brain. These findings suggest that NG2 may counteract neurological deficits and adverse glial responses in TBI.

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TI Suppressor of Cytokine Signaling-2 (SOCS2) Regulates the Microglial

Response and Improves Functional Outcome after Traumatic Brain Injury in

Mice

SO PLOS ONE

LA English

DT Article

ID CONTROLLED CORTICAL IMPACT; CD40 GENE-EXPRESSION; SUBVENTRICULAR ZONE;

NEURITE OUTGROWTH; ERYTHROPOIETIN; NEUROGENESIS; PROLIFERATION; CELLS;

DIFFERENTIATION; RECOVERY

AB Traumatic brain injury (TBI) is frequently characterized by neuronal, axonal and myelin loss, reactive gliosis and neuroinflammation, often associated with functional deficits. Endogenous repair mechanisms include production of new neurons from precursor cells, but usually the new neurons fail to integrate and survive more than a few weeks. This is in part mediated by the toxic and inflammatory environment present in the injured brain which activates precursor cells to proliferate and differentiate but limits survival of the newborn progeny. Therefore, an understanding of mechanisms that regulate production and survival of newborn neurons and the neuroinflammatory response after brain injury may lead to therapeutic options to improve outcomes. Suppressor of Cytokine Signaling 2 (SOCS2) promotes hippocampal neurogenesis and survival of newborn neurons in the adult brain and regulates anti-inflammatory responses in the periphery, suggesting it may be a useful candidate to improve outcomes of TBI. In this study the functional and cellular responses of SOCS2 over-expressing transgenic (SOCS2Tg) mice were compared to wildtype litter-mates following mild or moderately severe TBI. Unlike wildtype controls, SOCS2Tg mice showed functional improvement on a ladder test, with a smaller lesion volume at 7d post injury and increased numbers of proliferative CD11b(+) microglia/macrophages at 35d post-injury in the mild injury paradigm. At 7d post-moderately severe injury there was an increase in the area covered by cells expressing an anti-inflammatory M2 phenotype marker (CD206(+)) but no difference in cells with a pro-inflammatory M1 phenotype marker (CD16/32(+)). No effect of SOCS2 overexpression was observed in production or survival of newborn neurons, even in the presence of the neuroprotective agent erythropoietin (EPO). Therefore, SOCS2 may improve outcome of TBI in mice by regulating aspects of the neuroinflammatory response, promoting a more anti-inflammatory environment, although this was not sufficient to enhance survival of newborn cortical neurons.

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TI Age exacerbates the CCR2/5-mediated neuroinflammatory response to

traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Microglia; Macrophage; CCR2; Chemokine; Antagonist; Aging; Neurotrauma

ID MONOCYTE CHEMOATTRACTANT PROTEIN-1; NADPH OXIDASE; ALTERNATIVE

ACTIVATION; SPINAL-CORD; MICROGLIA; RECRUITMENT; NEUROTOXICITY;

FRACTALKINE; CHEMOKINES; RECOVERY

AB Background: Traumatic brain injury (TBI) is a major risk factor for the development of multiple neurodegenerative diseases, including Alzheimer's disease (AD) and numerous recent reports document the development of dementia after TBI. Age is a significant factor in both the risk of and the incidence of acquired brain injury. TBI-induced inflammatory response is associated with activation of brain resident microglia and accumulation of infiltrating monocytes, which plays a pivotal role in chronic neurodegeneration and loss of neurological function after TBI. Despite the extensive clinical evidence implicating neuroinflammation with the TBI-related sequelae, the specific role of these different myeloid cells and the influence of age on TBI-initiated innate immune response remain unknown and poorly studied.

Methods: We used gene profiling and pathway analysis to define the effect of age on inflammatory response at the time of injury. The recruitment of peripheral CCR2(+) macrophages was delineated using the CX3CR1(GFP/+)CCR2(RFP/+) reporter mouse. These responses were examined in the context of CCR2/5 antagonism using cenicriviroc.

Results: Unsupervised gene clustering and pathway analysis revealed that age predisposes exacerbated inflammatory response related to the recruitment and activation of peripheral monocytes to the injured brain. Using a unique reporter animal model able to discriminate resident versus peripherally derived myeloid cells, we demonstrate that in the aged brain, there is an increased accumulation of peripherally derived CCR2(+) macrophages after TBI compared to young animals. Exaggerated recruitment of this population of cells was associated with an augmented inflammatory response in the aged TBI animals. Targeting this cellular response with cenicriviroc, a dual CCR2/5 antagonist, significantly ameliorated injury-induced sequelae in the aged TBI animals.

Conclusions: Importantly, these findings demonstrate that peripheral monocytes play a non-redundant and contributing role to the etiology of trauma-induced inflammatory sequelae in the aged brain.

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TI Rho kinase inhibition following traumatic brain injury in mice promotes

functional improvement and acute neuron survival but has little effect

on neurogenesis, glial responses or neuroinflammation

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Rho GTPase; Astrocytic gliosis; Y27632; Fasudil; Microglia; Controlled

cortical impact; Horizontal ladder test; NeuN

ID CONTROLLED CORTICAL IMPACT; NEURITE OUTGROWTH; ROCK INHIBITOR; IN-VITRO;

DOPAMINERGIC-NEURONS; AXONAL REGENERATION; LESIONAL EXPRESSION; FAMILY

GTPASES; MOUSE MODEL; FASUDIL

AB Inhibition of the Rho/Rho kinase pathway has been shown to be beneficial in a variety of neural injuries and diseases. In this manuscript we investigate the role of Rho kinase inhibition in recovery from traumatic brain injury using a controlled cortical impact model in mice. Mice subjected to a moderately severe TBI were treated for 1 or 4 weeks with the Rho kinase inhibitor Y27632, and functional outcomes and neuronal and glial cell responses were analysed at 1, 7 and 35 days post-injury. We hypothesised that Y27632-treated mice would show functional improvement, with augmented recruitment of neuroblasts from the SVZ and enhanced survival of newborn neurons in the pericontusional cortex, with protection against neuronal degeneration, neuroinflammation and modulation of astrocyte reactivity and blood-brain-barrier permeability. While Rho kinase inhibition enhanced recovery of motor function after trauma, there were no substantial increases in the recruitment of DCX+ neuroblasts or the number of BrdU(+) or EdU(+) labelled newborn neurons in the pericontusional cortex of Y27632-treated mice. Inhibition of Rho kinase significantly reduced the number of degenerating cortical neurons at 1 day post-injury compared to saline controls but had no longer term effect on neuronal degeneration, with only modest effects on astrocytic reactivity and macrophage/microglial responses. Overall, this study showed that Rho kinase contributes to acute neurodegenerative processes in the injured cortex but does not play a significant role in SVZ neural precursor cell-derived adult neurogenesis, glial responses or blood-brain barrier permeability following a moderately severe brain injury. (C) 2016 Elsevier Inc. All rights reserved.

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TI Cognitive deficits develop 1 month after diffuse brain injury and are

exaggerated by microglia-associated reactivity to peripheral immune

challenge

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE Traumatic brain injury; Fluid percussion injury; Microglia; Astrocytes;

Cognitive decline; Lipopolysaccharide; Neuroinflammation

ID DEPRESSIVE-LIKE BEHAVIOR; LONG-TERM POTENTIATION; AGED MICE; HIPPOCAMPAL

NEUROGENESIS; EXPERIMENTAL-MODELS; SICKNESS BEHAVIOR; NEURONAL LOSS;

HEAD-INJURY; ACTIVATION; MEMORY

AB Traumatic brain injury (TBI) elicits immediate neuroinflammatory events that contribute to acute cognitive, motor, and affective disturbance. Despite resolution of these acute complications, significant neuropsychiatric and cognitive issues can develop and progress after TBI. We and others have provided novel evidence that these complications are potentiated by repeated injuries, immune challenges and stressors. A key component to this may be increased sensitization or priming of glia after TBI. Therefore, our objectives were to determine the degree to which cognitive deterioration occurred after diffuse TBI (moderate midline fluid percussion injury) and ascertain if glial reactivity induced by an acute immune challenge potentiated cognitive decline 30 days post injury (dpi). In post-recovery assessments, hippocampal-dependent learning and memory recall were normal 7 dpi, but anterograde learning was impaired by 30 dpi. Examination of mRNA and morphological profiles of glia 30 dpi indicated a low but persistent level of inflammation with elevated expression of GFAP and IL-1 beta in astrocytes and MHCII and IL-1 beta in microglia. Moreover, an acute immune challenge 30 dpi robustly interrupted memory consolidation specifically in TBI mice. These deficits were associated with exaggerated microglia-mediated inflammation with amplified (IL-1 beta, CCL2, TNF alpha) and prolonged (TNF alpha) cytokine/chemokine expression, and a marked reactive morphological profile of microglia in the CA3 of the hippocampus. Collectively, these data indicate that microglia remain sensitized 30 dpi after moderate TBI and a secondary inflammatory challenge elicits robust microglial reactivity that augments cognitive decline.

Statement of Significance: Traumatic brain injury (TBI) is a major risk factor in development of neuropsychiatric problems long after injury, negatively affecting quality of life. Mounting evidence indicates that inflammatory processes worsen with time after a brain injury and are likely mediated by glia. Here, we show that primed microglia and astrocytes developed in mice 1 month following moderate diffuse TBI, coinciding with cognitive deficits that were not initially evident after injury. Additionally, TBI-induced glial priming may adversely affect the ability of glia to appropriately respond to immune challenges, which occur regularly across the lifespan. Indeed, we show that an acute immune challenge augmented microglial reactivity and cognitive deficits. This idea may provide new avenues of clinical assessments and treatments following TBI. (C) 2016 Elsevier Inc. All rights reserved.

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TI Changes in Binding of [<SUP>123</SUP>I]CLINDE, a High-Affinity

Translocator Protein 18 kDa (TSPO) Selective Radioligand in a Rat Model

of Traumatic Brain Injury

SO NEUROMOLECULAR MEDICINE

LA English

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ID PERIPHERAL BENZODIAZEPINE-RECEPTORS; POSITRON-EMISSION-TOMOGRAPHY;

MICROGLIAL ACTIVATION; NERVOUS-SYSTEM; IN-VIVO; FOCAL ISCHEMIA;

INFLAMMATION; PET; NEUROINFLAMMATION; SITES

AB After traumatic brain injury (TBI), secondary injuries develop, including neuroinflammatory processes that contribute to long-lasting impairments. These secondary injuries represent potential targets for treatment and diagnostics. The translocator protein 18 kDa (TSPO) is expressed in activated microglia cells and upregulated in response to brain injury and therefore a potential biomarker of the neuroinflammatory processes. Second-generation radioligands of TSPO, such as [I-123]CLINDE, have a higher signal-to-noise ratio as the prototype ligand PK11195. [I-123]CLINDE has been employed in human studies using single-photon emission computed tomography to image the neuroinflammatory response after stroke. In this study, we used the same tracer in a rat model of TBI to determine changes in TSPO expression. Adult Sprague-Dawley rats were subjected to moderate controlled cortical impact injury and sacrificed at 6, 24, 72 h and 28 days post surgery. TSPO expression was assessed in brain sections employing [I-123]CLINDE in vitro autoradiography. From 24 h to 28 days post surgery, injured animals exhibited a marked and time-dependent increase in [I-123]CLINDE binding in the ipsilateral motor, somatosensory and parietal cortex, as well as in the hippocampus and thalamus. Interestingly, binding was also significantly elevated in the contralateral M1 motor cortex following TBI. Craniotomy without TBI caused a less marked increase in [I-123]CLINDE binding, restricted to the ipsilateral hemisphere. Radioligand binding was consistent with an increase in TSPO mRNA expression and CD11b immunoreactivity at the contusion site. This study demonstrates the applicability of [I-123]CLINDE for detailed regional and quantitative assessment of glial activity in experimental models of TBI.

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AB An impaired ability to regulate microglia activation by fractalkine (CX3CL1) leads to microglia chronic sub-activation. How this condition affects outcome after acute brain injury is still debated, with studies showing contrasting results depending on the timing and the brain pathology. Here, we investigated the early and delayed consequences of fractalkine receptor (CX3CR1) deletion on neurological outcome and on the phenotypical features of the myeloid cells present in the lesions of mice with traumatic brain injury (TBI). Wild type (WT) and CX3CR1(-/-) C57Bl/6 mice were subjected to sham or controlled cortical impact brain injury. Outcome was assessed at 4 days and 5 weeks after TBI by neuroscore, neuronal count, and terminal deoxynucleotidyl transferase dUTP nick end labeling (TUNEL) staining. Compared with WT mice, CX3CR1(-/-) TBI mice showed a significant reduction of sensorimotor deficits and lower cellular damage in the injured cortex 4 days post-TBI. Conversely, at 5 weeks, they showed a worsening of sensorimotor deficits and pericontusional cell death. Microglia (M) and macrophage () activation and polarization were assessed by quantitative immunohistochemistry for CD11b, CD68, Ym1, and inducible nitric oxide synthase (iNOS)markers of M/ activation, phagocytosis, M2, and M1 phenotypes, respectively. Morphological analysis revealed a decreased area and perimeter of CD11b(+) cells in CX3CR1(-/-) mice at 4 days post-TBI, whereas, at 5 weeks, both parameters were significantly higher, compared with WT mice. At 4 days, CX3CR1(-/-) mice showed significantly decreased CD68 and iNOS immunoreactivity, while at 5 weeks post-injury, they showed a selective increase of iNOS. Gene expression on CD11b(+) sorted cells revealed an increase of interleukin 10 and insulin-like growth factor 1 (IGF1) at 1 day and a decrease of IGF1 4 days and 5 weeks post-TBI in CX3CR1(-/-), compared with WT mice. These data show an early protection followed by a chronic exacerbation of TBI outcome in the absence of CX3CR1. Thus, longitudinal effects of myeloid cell manipulation at different stages of pathology should be investigated to understand how and when their modulation may offer therapeutic chances.

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TI Nuclear Factor Erythroid 2-Related Factor 2 (Nrf2) Mediates

Neuroprotection in Traumatic Brain Injury at Least in Part by

Inactivating Microglia

SO MEDICAL SCIENCE MONITOR

LA English

DT Article

DE Brain Injuries; Microglia; NF-E2-Related Factor 2

ID TRANSCRIPTION FACTOR NRF2; ACTIVATION; INFLAMMATION; MICE; PROTECTION;

PHENOTYPE; SYSTEM; RAT

AB Background: Microglial activation has been reported to be involved in traumatic brain injury (TBI). Nuclear factor erythroid 2-related factor 2 (Nrf2) plays a significant role in protecting against TBI-induced secondary brain injury. However, the exact mechanism is not clearly understood. The present study aimed to explore whether Nrf2 protects against TBI partly by regulating microglia function.

Material/Methods: Microglia cells were isolated from C57BL/6 mouse brains (postnatal day 1-3). The expression of Nrf2 was suppressed by transfection with Nrf2-specific small interfering RNA (siRNA), and overexpressed by transfections with pcDNA3.1-Nrf2. The expression of Nrf2 was confirmed by real-time PCR and Western blotting. After transfection, cell viability, phagocytic ability, and the expression of pro-inflammatory cytokines (tumor necrosis factor (TNF)-alpha and interleukin (IL)-6) were determined by 3-(4, 5-dimethylthiazol-2-yl)-2, 5-diphenyltetrazolium bromide (MTT) colorimetric assay, phagocytosis assay, and enzyme-linked immunosorbent assay (ELISA), respectively.

Results: mRNA and protein expression levels of Nrf2 were significantly reduced by transfection with Nrf2-specific siRNA (both P<0.05) but were elevated by transfection with pcDNA3.1-Nrf2 (both P<0.01). The cell viability, phagocytic ability, and the expression of TNF-alpha and IL-6 were all significantly reduced by overexpression of Nrf2 but were significantly increased by silencing of Nrf2 compared with the control group.

Conclusions: Our results suggest that Nrf2 protects against TBI, at least part by regulating microglia function.

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TI Decreased cysteine uptake by EAAC1 gene deletion exacerbates neuronal

oxidative stress and neuronal death after traumatic brain injury

SO AMINO ACIDS

LA English

DT Article

DE EAAC1; Cysteine; Zinc; TBI; Reactive oxygen species; Microglia

ID TRANSIENT CEREBRAL-ISCHEMIA; ALTERS ZINC HOMEOSTASIS; GLUTAMATE

TRANSPORTER; MICROGLIAL ACTIVATION; GLUTATHIONE; MICE; SUSCEPTIBILITY;

SUPEROXIDE; INCREASES; RELEASE

AB Excitatory amino acid carrier type 1 (EAAC1), a high-affinity glutamate transporter, can expend energy to move glutamate into neurons. However, under normal physiological conditions, EAAC1 does not have a great effect on glutamate clearance but rather participates in the neuronal uptake of cysteine. This process is critical to maintaining neuronal antioxidant function by providing cysteine for glutathione synthesis. Previous study showed that mice lacking EAAC1 show increased neuronal oxidative stress following transient cerebral ischemia. In the present study, we sought to characterize the role of EAAC1 in neuronal resistance after traumatic brain injury (TBI). Young adult C57BL/6 wild-type or EAAC1 (-/-) mice were subjected to a controlled cortical impact model for TBI. Neuronal death after TBI showed more than double the number of degenerating neurons in the hippocampus in EAAC1 (-/-) mice compared with wild-type mice. Superoxide production, zinc translocation and microglia activation similarly showed a marked increase in the EAAC1 (-/-) mice. Pretreatment with N-acetyl cysteine (NAC) reduced TBI-induced neuronal death, superoxide production and zinc translocation. These findings indicate that cysteine uptake by EAAC1 is important for neuronal antioxidant function and survival following TBI. This study also suggests that administration of NAC has therapeutic potential in preventing TBI-induced neuronal death.

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TI Traumatic brain injury increases levels of miR-21 in extracellular

vesicles: implications for neuroinflammation

SO FEBS OPEN BIO

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DT Article

DE controlled cortical impact; exosomes; microglia; microRNA;

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ID SPINAL-CORD-INJURY; MICRORNA EXPRESSION; IMMUNE-RESPONSES; INFLAMMATORY

RESPONSE; MESSENGER-RNAS; UP-REGULATION; CELL-DEATH; RECEPTOR 7; RATS;

NEURONS

AB Traumatic brain injury (TBI) is an important health concern and effective treatment strategies remain elusive. Understanding the complex multicellular response to TBI may provide new avenues for intervention. In the context of TBI, cell-cell communication is critical. One relatively unexplored form of cell-cell communication in TBI is extracellular vesicles (EVs). These membrane-bound vesicles can carry many different types of cargo between cells. Recently, miRNA in EVs have been shown to mediate neuroinflammation and neuronal injury. To explore the role of EV-associated miRNA in TBI, we isolated EVs from the brain of injured mice and controls, purified RNA from brain EVs, and performed miRNA sequencing. We found that the expression of miR-212 decreased, while miR-21, miR-146, miR-7a, and miR-7b were significantly increased with injury, with miR-21 showing the largest change between conditions. The expression of miR-21 in the brain was primarily localized to neurons near the lesion site. Interestingly, adjacent to these miR-21-expressing neurons were activated microglia. The concurrent increase in miR-21 in EVs with the elevation of miR-21 in neurons, suggests that miR-21 is secreted from neurons as potential EV cargo. Thus, this study reveals a new potential mechanism of cell-cell communication not previously described in TBI.

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TI Recombinant human interleukin-1 receptor antagonist promotes M1

microglia biased cytokines and chemokines following human traumatic

brain injury

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE Chemokines; brain trauma; microglia; inflammation; neurochemistry

ID ACUTE ISCHEMIC-STROKE; PHASE-II; MACROPHAGE ACTIVATION; HEAD-INJURY;

MICE; POLARIZATION; INFLAMMATION; MCP-1; NEUROINFLAMMATION;

NEUTRALIZATION

AB Interleukin-1 receptor antagonist (IL1ra) has demonstrated efficacy in a wide range of animal models of neuronal injury. We have previously published a randomised controlled study of IL1ra in human severe TBI, with concomitant microdialysis and plasma sampling of 42 cytokines and chemokines. In this study, we have used partial least squares discriminant analysis to model the effects of drug administration and time following injury on the cytokine milieu within the injured brain. We demonstrate that treatment with rhIL1ra causes a brain-specific modification of the cytokine and chemokine response to injury, particularly in samples from the first 48 h following injury. The magnitude of this response is dependent on the concentration of IL1ra achieved in the brain extracellular space. Chemokines related to recruitment of macrophages from the plasma compartment (MCP-1) and biasing towards a M1 microglial phenotype (GM-CSF, IL1) are increased in patient samples in the rhIL1ra-treated patients. In control patients, cytokines and chemokines biased to a M2 microglia phenotype (IL4, IL10, MDC) are relatively increased. This pattern of response suggests that a simple classification of IL1ra as an 'anti-inflammatory' cytokine may not be appropriate and highlights the importance of the microglial response to injury.

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TI Etifoxine improves sensorimotor deficits and reduces glial activation,

neuronal degeneration, and neuroinflammation in a rat model of traumatic

brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Traumatic brain injury; Etifoxine; Neuroinflammation; TSPO;

Neurosteroids; Functional recovery; Cytokines; Astrogliosis; Neuronal

degeneration; Microglia

ID PERIPHERAL BENZODIAZEPINE-RECEPTOR; CONTROLLED CORTICAL IMPACT;

TUMOR-NECROSIS-FACTOR; PROTEIN 18 KDA; CELL-DEATH; INFLAMMATORY

RESPONSE; ANXIOLYTIC ETIFOXINE; GABA(A) RECEPTORS; TEMPORAL PROFILE;

NERVOUS-SYSTEM

AB Background: Traumatic brain injury (TBI) results in important neurological impairments which occur through a cascade of deleterious physiological events over time. There are currently no effective treatments to prevent these consequences. TBI is followed not only by an inflammatory response but also by a profound reorganization of the GABAergic system and a dysregulation of translocator protein 18 kDa (TSPO). Etifoxine is an anxiolytic compound that belongs to the benzoxazine family. It potentiates GABAergic neurotransmission, either through a positive allosteric effect or indirectly, involving the activation of TSPO that leads to an increase in neurosteroids synthesis. In several models of peripheral nerve injury, etifoxine has been demonstrated to display potent regenerative and anti-inflammatory properties and to promote functional recovery. Prior study also showed etifoxine efficacy in reducing brain edema in rats. In light of these positive results, we used a rat model of TBI to explore etifoxine treatment effects in a central nervous system injury, from functional outcomes to the underlying mechanisms.

Methods: Male Sprague-Dawley rats received contusion (n = 18) or sham (n = 19) injuries centered laterally to bregma over the left sensorimotor cortex. They were treated with etifoxine (50 mg/kg, i.p.) or its vehicle 30 min following injury and every day during 7 days. Rats underwent behavioral testing to assess sensorimotor function. In another experiment, injured rats (n = 10) or sham rats (n = 10) received etifoxine (EFX) (50 mg/kg, i.p.) or its vehicle 30 min post-surgery. Brains were then dissected for analysis of neuroinflammation markers, glial activation, and neuronal degeneration.

Results: Brain-injured rats exhibited significant sensorimotor function deficits compared to sham-injured rats in the bilateral tactile adhesive removal test, the beam walking test, and the limb-use asymmetry test. After 2 days of etifoxine treatment, behavioral impairments were significantly reduced. Etifoxine treatment reduced pro-inflammatory cytokines levels without affecting anti-inflammatory cytokines levels in injured rats, reduced macrophages and glial activation, and reduced neuronal degeneration.

Conclusions: Our results showed that post-injury treatment with etifoxine improved functional recovery and reduced neuroinflammation in a rat model of TBI. These findings suggest that etifoxine may have a therapeutic potential in the treatment of TBI.

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TI Targeting Kv1.3 channels to reduce white matter pathology after

traumatic brain injury

SO EXPERIMENTAL NEUROLOGY

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DT Article

DE Microglia; Inflammation; White matter injury; Recovery of function;

Compound action potentials; Glial cell culture

ID MITOCHONDRIAL PERMEABILITY TRANSITION; CALCINEURIN-INDEPENDENT

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TACROLIMUS FK506

AB Axonal injury is present in essentially all clinically significant cases of traumatic brain injury (TBI). While no effective treatment has been identified to date, experimental TBI models have shown promising axonal protection using immunosuppressants FK506 and Cyclosporine-A, with treatment benefits attributed to calcineurin inhibition or protection of mitochondrial function. However, growing evidence suggests neuroprotective efficacy of these compounds may also involve direct modulation of ion channels, and in particular Kv1.3. The present study tested whether blockade of Kv1.3 channels, using Clofazimine (CFZ), would alleviate TEI-induced white matter pathology in rodents. Postinjury CFZ administration prevented suppression of compound action potential (CAP) amplitude in the corpus callosum of adult rats following midline fluid percussion TBI, with injury and treatment effects primarily expressed in unmyelinated CAPs. Kv1.3 protein levels in callosal tissue extracts were significantly reduced postinjury, but this loss was prevented by CFZ treatment. In parallel, CFZ also attenuated the injury-induced elevation in pro-inflammatory cytokine IL1-beta. The effects of CFZ on glial function were further studied using mixed microglia/astrocyte cell cultures derived from P3-5 mouse corpus callosum. Cultures of callosal glia challenged with lipopolysaccharide exhibited a dramatic increase in IL1-beta levels, accompanied by reactive morphological changes in microglia, both of which were attenuated by CFZ treatment. These results support a cell specific role for Kv1.3 signaling in white matter pathology after TBI, and suggest a treatment approach based on the blockade of these channels. This therapeutic strategy may be especially efficacious for normalizing neuro-glial interactions affecting unmyelinated axons after TBI. (C) 2016 Elsevier Inc All rights reserved.

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TI Protective Effects of Chinese Herbal Medicine <i>Rhizoma drynariae</i>

in Rats After Traumatic Brain Injury and Identification of Active

Compound

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article

DE Microglia; Rhizoma drynariae; Traumatic brain injury; UPLC

ID CHAIHU-SHUGAN-SAN; INTRACEREBRAL HEMORRHAGE; MERANZIN HYDRATE; CELL;

TRANSPLANTATION; PROGESTERONE; MICE; NRF2

AB Traumatic brain injury (TBI) is a leading cause of death and disability in the USA. Effective therapeutic strategies for TBI are needed, and increasing attention is turning toward traditional herbal medicine. Rhizoma drynariae is a traditional Chinese medicine that has immunomodulatory and anti-inflammatory effects. Here, using the controlled cortical impact model of TBI in rats, we examined whether oral administration of R. drynariae can reduce TBI-induced brain injury in rats. We also identified the likely active compound among its four major phytochemicals in decoction. We found that post-treatment with R. drynariae decreased brain lesion volume, improved neurologic and cognitive function, and reduced anxiety- and depression-like behaviors. These changes were accompanied by reduced blood levels of IL-6 and increased IL-10. R. drynariae treatment also reversed the TBI-induced decrease in blood monocyte numbers and percentage of blood CD3 and CD4 T lymphocytes while inhibiting microglial/macrophage activation. Furthermore, by using ultra performance liquid chromatography and comparing retention times with authentic standards, we identified eriodictyol as the putative active compound of R. drynariae extract in the blood of rats with TBI. These novel findings indicate that the traditional Chinese herbal medicine R. drynariae protects brain against TBI-induced brain injury, possibly via immune-promoting, anti-inflammatory, and neuroprotective effects. Eriodictyol could be its active compound.

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TI Microglial/Macrophage Polarization Dynamics following Traumatic Brain

Injury

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE M1-like; M2-like; microglia/macrophage; NOX2; polarization; traumatic

brain injury

ID NADPH OXIDASE; MICROGLIAL ACTIVATION; PROGRESSIVE NEURODEGENERATION;

NEUROINFLAMMATORY PHENOTYPE; LIMITS NEUROINFLAMMATION; MACROPHAGE

POLARIZATION; INFLAMMATORY RESPONSE; GENE-EXPRESSION; NEURONAL DEATH;

WHITE-MATTER

AB Activated microglia and macrophages exert dual beneficial and detrimental roles after central nervous system injury, which are thought to be due to their polarization along a continuum from a classical pro-inflammatory M1-like state to an alternative anti-inflammatory M2-like state. The goal of the present study was to analyze the temporal dynamics of microglia/macrophage polarization within the lesion micro-environment following traumatic brain injury (TBI) using a moderate-level controlled cortical impact (CCI) model in mice. We performed a detailed phenotypic analysis of M1-and M2-like polarized microglia/macrophages, as well as nicotinamide adenine dinucleotide phosphate oxidase (NOX2) expression, through 7 days post-injury using real-time polymerase chain reaction (qPCR), flow cytometry and image analyses. We demonstrated that microglia/macrophages express both M1-and M2-like phenotypic markers early after TBI, but the transient up-regulation of the M2-like phenotype was replaced by a predominant M1-or mixed transitional (Mtran) phenotype that expressed high levels of NOX2 at 7 days post-injury. The shift towards the M1-like and Mtran phenotype was associated with increased cortical and hippocampal neurodegeneration. In a follow up study, we administered a selective NOX2 inhibitor, gp91ds-tat, to CCI mice starting at 24 h post-injury to investigate the relationship between NOX2 and M1-like/Mtran phenotypes. Delayed gp91ds-tat treatment altered M1-/M2-like balance in favor of the anti-inflammatory M2-like phenotype, and significantly reduced oxidative damage in neurons at 7 days post-injury. Therefore, our data suggest that despite M1-like and M2-like polarized microglia/macrophages being activated after TBI, the early M2-like response becomes dysfunctional over time, resulting in development of pathological M1-like and Mtran phenotypes driven by increased NOX2 activity.

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TI Mild Traumatic Brain Injury Produces Neuron Loss That Can Be Rescued by

Modulating Microglial Activation Using a CB2 Receptor Inverse Agonist

SO FRONTIERS IN NEUROSCIENCE

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DT Article

DE traumatic brain injury; neuron loss; neuron rescue; cerebral cortex;

striatum; basolateral amygdala; cannabinoid type-2 receptor inverse

agonist

ID BASOLATERAL AMYGDALA; CELL-DEATH; CONTAINING INTERNEURONS;

GLUTAMATE-RECEPTOR; CORPUS-CALLOSUM; BASAL GANGLIA; AXONAL INJURY; MOUSE

MODEL; BLAST; FEAR

AB We have previously reported that mild TBI created by focal left-side cranial blast in mice produces widespread axonal injury, microglial activation, and a variety of functional deficits. We have also shown that these functional deficits are reduced by targeting microglia through their cannabinoid type-2 (CB2) receptors using 2-week daily administration of the CB2 inverse agonist SMM-189. CB2 inverse agonists stabilize the G-protein coupled CB2 receptor in an inactive conformation, leading to increased phosphorylation and nuclear translocation of the cAMP response element binding protein (CREB), and thus bias activated microglia from a pro-inflammatory M1 to a pro-healing M2 state. In the present study, we showed that SMM-189 boosts nuclear pCREB levels in microglia in several brain regions by 3 days after TBI, by using pCREB/CD68 double immunofluorescent labeling. Next, to better understand the basis of motor deficits and increased fearfulness after TBI, we used unbiased stereological methods to characterize neuronal loss in cortex, striatum, and basolateral amygdala (BLA) and assessed how neuronal loss was affected by SMM-189 treatment. Our stereological neuron counts revealed a 20% reduction in cortical and 30% reduction in striatal neurons bilaterally at 2-3 months post blast, with SMM-189 yielding about 50% rescue. Loss of BLA neurons was restricted to the blast side, with 33% of Thy1+ fear-suppressing pyramidal neurons and 47% of fear-suppressing parvalbuminergic (PARV) interneurons lost, and Thy1-negative fear-promoting pyramidal neurons not significantly affected. SMM-189 yielded 50-60% rescue of Thy1+ and PARV neuron loss in BLA. Thus, fearfulness after mild TBI may result from the loss of fear-suppressing neuron types in BLA, and SMM-189 may reduce fearfulness by their rescue. Overall, our findings indicate that SMM-189 rescues damaged neurons and thereby alleviates functional deficits resulting from TBI, apparently by selectively modulating microglia to the beneficial M2 state. CB2 inverse agonists thus represent a promising therapeutic approach for mitigating neuroinflammation and neurodegeneration.

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TI Traumatic Brain Injury Leads to Development of Parkinson's Disease

Related Pathology in Mice

SO FRONTIERS IN NEUROSCIENCE

LA English

DT Article

DE chronic traumatic brain injury; Parkinson's disease; e-synuclein;

nuclear factor-kappa B; microglia; astrocytes; neurotrophic factors;

non-motor symptoms

ID NF-KAPPA-B; ALPHA-SYNUCLEIN; SUBSTANTIA-NIGRA; DOPAMINERGIC-NEURONS;

TREATMENT STRATEGIES; ANXIETY; RATS; PATHOGENESIS; ASTROCYTES;

EXPRESSION

AB Traumatic brain injury (TBI) is a major health and socio-economic problem that affects all societies. This condition results from the application of external physical strength to the brain that leads to transitory or permanent structural and functional impairments. Moreover, TBI is a risk factor for neurodegeneration and can e.g., increase the risk for Parkinson's disease (PD), a late-onset neurodegenerative disorder with loss of dopaminergic neurons in substantia nigra. In this study, we wanted to explore the possible development of PD-related pathology within the context of an experimental model of TBI. Traumatic brain injury was induced in mice by controlled cortical impact. At different time points behavioral tests (open field, elevated plus maze tests, and Barnes maze) were performed: The animals were sacrificed 30 days after the impact and the brains were processed for Western blot and immunohistochemical analyses. Following TBI there was a significant decrease in expression of tyrosine hydroxylase and dopamine transporter in the substantia nigra as well as significant behavioral alterations. In addition, a strong increase in neuroinflammation was evident, as shown by increased levels of cyclooxygenase-2 and inducible nitric oxide synthase as well as I kappa B-alpha degradation and nuclear-KB translocation. Moreover, neurotrophic factors such as brain-derived neurotrophic factor, neurotrophin-3, nerve growth factor, and glial cell line-derived neurotrophic factor were decreased 30 days post-TBI. Interestingly, we observed a significant accumulation of alpha-synuclein in microglia compared to astrocytes. This study suggests that PD-related molecular events can be triggered upon TBI. The biological mechanisms linking brain trauma and neurodegenerative diseases need to be further investigated.

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TI Neuroinflammation in animal models of traumatic brain injury

SO JOURNAL OF NEUROSCIENCE METHODS

LA English

DT Article

DE Traumatic brain injury (TBI); Neuroinflammation; Glia cells; Astrocytes;

Microglia; Controlled cortical impact; Weight-drop impact; Lateralfluid

percussion; Measurements evaluating neuroinflammation

ID NECROSIS-FACTOR-ALPHA; CONTROLLED CORTICAL IMPACT; PEPTIDE-1 RECEPTOR

AGONIST; FLUID PERCUSSION INJURY; CLOSED-HEAD INJURY;

SPINAL-CORD-INJURY; SIGNALING PATHWAY; OXIDATIVE STRESS;

GENE-EXPRESSION; CEREBRAL-CORTEX

AB Traumatic brain injury (TBI) is a leading cause of mortality and morbidity worldwide. Neuroinflammation is prominent in the short and long-term consequences of neuronal injuries that occur after TBI. Neuroinflammation involves the activation of glia, including microglia and astrocytes, to release inflammatory mediators within the brain, and the subsequent recruitment of peripheral immune cells. Various animal models of TBI have been developed that have proved valuable to elucidate the pathophysiology of the disorder and to assess the safety and efficacy of novel therapies prior to clinical trials. These models provide an excellent platform to delineate key injury mechanisms that associate with types of injury (concussion, contusion, and penetration injuries) that occur clinically for the investigation of mild, moderate, and severe forms of TBI. Additionally, TBI modeling in genetically engineered mice, in particular, has aided the identification of key molecules and pathways for putative injury mechanisms, as targets for development of novel therapies for human TBI. This Review details the evidence showing that neuroinflammation, characterized by the activation of microglia and astrocytes and elevated production of inflammatory mediators, is a critical process occurring in various TBI animal models, provides a broad overview of commonly used animal models of TBI, and overviews representative techniques to quantify markers of the brain inflammatory process. A better understanding of neuroinflammation could open therapeutic avenues for abrogation of secondary cell death and behavioral symptoms that may mediate the progression of TBI. (C) 2016 Elsevier B.V. All rights reserved.

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TI Microglial neuroinflammation contributes to tau accumulation in chronic

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DT Article

DE CTE; Neuroinflammation; Microglia; Repetitive head impacts; Mild

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AB The chronic effects of repetitive head impacts (RHI) on the development of neuroinflammation and its relationship to chronic traumatic encephalopathy (CTE) are unknown. Here we set out to determine the relationship between RHI exposure, neuroinflammation, and the development of hyperphosphorylated tau (ptau) pathology and dementia risk in CTE. We studied a cohort of 66 deceased American football athletes from the Boston University-Veteran's Affairs-Concussion Legacy Foundation Brain Bank as well as 16 non-athlete controls. Subjects with a neurodegenerative disease other than CTE were excluded. Counts of total and activated microglia, astrocytes, and ptau pathology were performed in the dorsolateral frontal cortex (DLF). Binary logistic and simultaneous equation regression models were used to test associations between RHI exposure, microglia, ptau pathology, and dementia. Duration of RHI exposure and the development and severity of CTE were associated with reactive microglial morphology and increased numbers of CD68 immunoreactive microglia in the DLF. A simultaneous equation regression model demonstrated that RHI exposure had a significant direct effect on CD68 cell density (p < 0.0001) and ptau pathology (p < 0.0001) independent of age at death. The effect of RHI on ptau pathology was partially mediated through increased CD68 positive cell density. A binary logistic regression demonstrated that a diagnosis of dementia was significantly predicted by CD68 cell density (OR = 1.010, p = 0.011) independent of age (OR = 1.055, p = 0.007), but this effect disappeared when ptau pathology was included in the model. In conclusion, RHI is associated with chronic activation of microglia, which may partially mediate the effect of RHI on the development of ptau pathology and dementia in CTE. Inflammatory molecules may be important diagnostic or predictive biomarkers as well as promising therapeutic targets in CTE.

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TI NOX2 drives M1-like microglial/macrophage activation and

neurodegeneration following experimental traumatic brain injury

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE NADPH oxidase; NOX2; Traumatic brain injury; Microglia; Macrophage;

Neuroinflammation; M1-/M2-like; Neurodegeneration

ID SPINAL-CORD-INJURY; NADPH OXIDASE; MICROGLIAL ACTIVATION; OXIDATIVE

STRESS; WHITE-MATTER; ALTERNATIVE ACTIVATION; INFLAMMATORY RESPONSE;

THERAPEUTIC TARGETS; REDOX REGULATION; MOUSE MODEL

AB Following traumatic brain injury (TBI), activation of microglia and peripherally derived inflammatory macrophages occurs in association with tissue damage. This neuroinflammatory response may have beneficial or detrimental effects on neuronal survival, depending on the functional polarization of these cells along a continuum from M1-like to M2-like activation states. The mechanisms that regulate M1-like and M2-like activation after TBI are not well understood, but appear in part to reflect the redox state of the lesion microenvironment. NADPH oxidase (NOX2) is a critical enzyme system that generates reactive oxygen species in microglia/macrophages. After TBI, NOX2 is strongly up-regulated in Ml-like, but not in M2-like polarized cells. Therefore, we hypothesized that NOX2 drives M1-like neuroinflammation and contributes to neurodegeneration and loss of neurological function after TBI. In the present studies we inhibited NOX2 activity using NOX2-knockout mice or the selective peptide inhibitor gp91ds-tat. We show that NOX2 is highly up-regulated in infiltrating macrophages after injury, and that NOX2 deficiency reduces markers of M1-like activation, limits tissue loss and neurodegeneration, and improves motor recovery after moderate-level control cortical injury (CCI). NOX2 deficiency also promotes M2-like activation after CCI, through increased IL-4R alpha signaling in infiltrating macrophages, suggesting that NOX2 acts as a critical switch between M1- and M2-like activation states after TBI. Administration of gp91ds-tat to wild-type CCI mice starting at 24 h post-injury reduces deficits in cognitive function and increased M2-like activation in the hippocampus. Collectively, our data indicate that increased NOX2 activity after TBI drives Ml-like activation that contributes to inflammatory-mediated neurodegeneration, and that inhibiting this pathway provides neuroprotection, in part by altering M1-/M2-like balance towards the M2-like neuroinflammatory response. (C) 2016 Elsevier Inc. All rights reserved.

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TI Early electroacupuncture treatment ameliorates neuroinflammation in rats

with traumatic brain injury

SO BMC COMPLEMENTARY AND ALTERNATIVE MEDICINE

LA English

DT Article

DE Astrocyte; Electroacupuncture; Microglia; Neuronal apoptosis; Traumatic

brain injury; Tumor Necrosis factor-alpha a

ID TUMOR-NECROSIS-FACTOR; LATERAL FLUID-PERCUSSION; ACUPUNCTURE;

ETANERCEPT; ACTIVATION; EXPRESSION; APOPTOSIS; ALPHA; ST36

AB Background: Neuroinflammation is the leading cause of neurological sequelae after traumatic brain injury (TBI). The aim of the present study was to investigate whether the neuroprotective effects of electroacupuncture (EA) are mediated by anti-neuroinflammatory effects in a rat model of TBI.

Methods: Male Sprague-Dawley rats were randomly divided into three groups: sham-operated, TBI control, and EA-treated. The animals in the sham-operated group underwent a sham operation, those in the TBI control group were subjected to TBI, but not EA, and those in the EA group were treated with EA for 60 min immediately after TBI, daily for 3 consecutive days. EA was applied at the acupuncture points GV20, GV26, LI4, and KI1, using a densedispersed wave, at frequencies of 0.2 and 1 Hz, and an amplitude of 1 mA. Cell infarction volume (TTC stain), neuronal apoptosis (markers: TUNEL and Caspase-3), activation of microglia (marker: Iba1) and astrocytes (marker: GFAP), and tumor necrosis factor (TNF)-alpha expression in the microglia and astrocytes were evaluated by immunofluorescence. Functional outcomes were assessed using the inclined plane test. All tests were performed 72 h after TBI.

Results: We found that TBI-induced loss of grasp strength, infarction volume, neuronal apoptosis, microglial and astrocyte activation, and TNF-alpha expression in activated microglia and astrocytes were significantly attenuated by EA treatment.

Conclusions: Treatment of TBI in the acute stage with EA for 60 min daily for 3 days could ameliorate neuroinflammation. This may thus represent a mechanism by which functional recovery can occur after TBI.

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JI BMC Complement. Altern. Med.

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TI A positive correlation exists between neurotrauma and TGF-β1-containing

microglia in rats

SO EUROPEAN JOURNAL OF CLINICAL INVESTIGATION

LA English

DT Article

DE Astragaloside; neuro AiD (TM) (MLC601); transforming growth factor-beta

1; traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; TRADITIONAL CHINESE MEDICINE; TRANSFORMING

GROWTH-FACTOR-BETA-1; INCREASES NEUROGENESIS; NEUROAID EFFICACY; STROKE;

INHIBITION; PROTECTS; MODEL; NEUROPROTECTION

AB Background Transforming growth factor-beta 1 (TGF-beta 1) regulates many processes after traumatic brain injury (TBI). Both Neuro AiD (TM) (MLC601) and astragaloside (AST) attenuate microglia activation in rats with TBI. The purpose of this study was to investigate whether MLC601 or AST improves output of TBI by affecting microglial expression of TGF-beta 1.

Materials and methods Adult male Sprague-Dawley rats (120 in number) were used to investigate the contribution of TGF-beta 1-containing microglia in the MLC601-mediated or the AST-mediated neuroprotection in the brain trauma condition using lateral fluid percussion injury.

Results Pearson correlation analysis revealed that there was a positive correlation between brain injury (evidenced by both brain contused volume and neurological severity score) and the cortical numbers of TGF-beta 1-containing microglia for the rats (n = 12) 4 days post-TBI. MLC601 or AST significantly (P < 0.05) attenuated TBI-induced brain contused volume (119 +/- 14 mm(3) or 108 +/- 11 mm(3) vs. 160 +/- 21 mm(3)), neurological severity score (7.8 +/- 0.3 or 8.1 +/- 0.4 vs. 10.2 +/- 0.5) and numbers of TGF-beta 1-containing microglia (6% +/- 2% or 11% +/- 3% vs. 79% +/- 7%) for the rats 4 days post-TBI.

Conclusions There was a positive correlation between TBI and cortical numbers of TGF-beta 1-containing microglia which could be significantly attenuated by astragaloside or NeuroAiD (TM) (MLC601) in rats.

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TI Contribution of Mast Cells to Injury Mechanisms in a Mouse Model of

Pediatric Traumatic Brain Injury

SO JOURNAL OF NEUROSCIENCE RESEARCH

LA English

DT Article

DE neuroinflammation; histamine; apoptosis; myelin; neuron; microglia

ID WHITE-MATTER; SYSTEMIC INFLAMMATION; IMMATURE RAT; HISTAMINE; MICROGLIA;

NEUROPROTECTION; RECEPTORS; ACCUMULATION; ACTIVATION; PLASTICITY

AB The cognitive and behavioral deficits caused by traumatic brain injury (TBI) to the immature brain are more severe and persistent than injuries to the adult brain. Understanding this developmental sensitivity is critical because children under 4 years of age of sustain TBI more frequently than any other age group. One of the first events after TBI is the infiltration and degranulation of mast cells (MCs) in the brain, releasing a range of immunomodulatory substances; inhibition of these cells is neuroprotective in other types of neonatal brain injury. This study investigates for the first time the role of MCs in mediating injury in a P7 mouse model of pediatric contusion-induced TBI. We show that various neural cell types express histamine receptors and that histamine exacerbates excitotoxic cell death in primary cultured neurons. Cromoglycate, an inhibitor of MC degranulation, altered the inflammatory phenotype of microglia activated by TBI, reversing several changes but accentuating others, when administered before TBI. However, without regard to the time of cromoglycate administration, inhibiting MC degranulation did not affect cell loss, as evaluated by ventricular dilatation or cleaved caspase-3 labeling, or the density of activated microglia, neurons, or myelin. In double-heterozygous cKit mutant mice lacking MCs, this overall lack of effect was confirmed. These results suggest that the role of MCs in this model of pediatric TBI is restricted to subtle effects and that they are unlikely to be viable neurotherapeutic targets. (C) 2016 Wiley Periodicals, Inc.

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TI Emerging Roles for the Immune System in Traumatic Brain Injury

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DE traumatic brain injury; neuroinflammation; cytokine; inflammasome;

innate immunology; neuroprotection; microglia; neurodegeneration

ID TUMOR-NECROSIS-FACTOR; INTERLEUKIN-1 RECEPTOR ANTAGONIST; AMYLOID

PROTEIN DEPOSITION; CONTROLLED CORTICAL IMPACT; COLONY-STIMULATING

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ALZHEIMERS-DISEASE; CEREBROSPINAL-FLUID

AB Traumatic brain injury (TBI) affects an ever-growing population of all ages with long-term consequences on health and cognition. Many of the issues that TBI patients face are thought to be mediated by the immune system. Primary brain damage that occurs at the time of injury can be exacerbated and prolonged for months or even years by chronic inflammatory processes, which can ultimately lead to secondary cell death, neurodegeneration, and long-lasting neurological impairment. Researchers have turned to rodent models of TBI in order to understand how inflammatory cells and immunological signaling regulate the post-injury response and recovery mechanisms. In addition, the development of numerous methods to manipulate genes involved in inflammation has recently expanded the possibilities of investigating the immune response in TBI models. As results from these studies accumulate, scientists have started to link cells and signaling pathways to pro-and anti-inflammatory processes that may contribute beneficial or detrimental effects to the injured brain. Moreover, emerging data suggest that targeting aspects of the immune response may offer promising strategies to treat TBI. This review will cover insights gained from studies that approach TBI research from an immunological perspective and will summarize our current understanding of the involvement of specific immune cell types and cytokines in TBI pathogenesis.

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TI Implantation of Brain-Derived Extracellular Matrix Enhances Neurological

Recovery after Traumatic Brain Injury

SO CELL TRANSPLANTATION

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DE astrogliosis; microglia; neurobehavioral function; neurodegeneration;

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ID MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS; WHITE-MATTER;

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AB Scaffolds composed of extracellular matrix (ECM) are being investigated for their ability to facilitate brain tissue remodeling and repair following injury. The present study tested the hypothesis that the implantation of brain-derived ECM would attenuate experimental traumatic brain injury (TBI) and explored potential underlying mechanisms. TBI was induced in mice by a controlled cortical impact (CCI). ECM was isolated from normal porcine brain tissue by decellularization methods, prepared as a hydrogel, and injected into the ipsilesional corpus callosum and striatum 1 h after CCI. Lesion volume and neurological function were evaluated up to 35 d after TBI. Immunohistochemistry was performed to assess post-TBI white matter integrity, reactive astrogliosis, and microglial activation. We found that ECM treatment reduced lesion volume and improved neuro-behavioral function. ECM-treated mice showed less post-TBI neurodegeneration in the hippocampus and less white matter injury than control, vehicle-treated mice. Furthermore, ECM ameliorated TBI-induced gliosis and microglial pro-inflammatory responses, thereby providing a favorable microenvironment for tissue repair. Our study indicates that brain ECM hydrogel implantation improved the brain microenvironment that facilitates post-TBI tissue recovery. Brain ECM offers excellent biocompatibility and holds potential as a therapeutic agent for TBI, alone or in combination with other treatments.

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TI Early monitoring and quantitative evaluation of macrophage infiltration

after experimental traumatic brain injury: A magnetic resonance imaging

and flow cytometric analysis

SO MOLECULAR AND CELLULAR NEUROSCIENCE

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INFILTRATION; BARRIER PERMEABILITY; CONTRAST AGENT; MOUSE-BRAIN; MRI;

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AB The inflammatory response following traumatic brain injury (TBI) is regulated by phagocytic cells. These cells comprising resident microglia and infiltrating macrophages play a pivotal role in the interface between early detrimental and delayed beneficial effects of inflammation. The aim of the present study was to monitor the early effect of monocyte/phagocytic accumulation and further to explore its kinetics in TBI mice. Localized macrophage population was monitored using ultrasmall superparamagnetic iron oxide (USPIO) nanoparticle enhanced in vivo serial magnetic resonance imaging (MRI). Flow cytometry based gating study was performed to discriminate between resident microglia (Ly6G(-) CD11b+ CD45(low)) and infiltrating macrophages (Ly6G(-) CD11b+ CD45(high)) at the injury site. The T-2\* relaxation analysis revealed that maximum macrophage infiltration occurs between 66 and 72 h post injury (42-48 h post administration of USPIO) at the site of inflammation. This imaging data was well supported by iron oxide specific Prussian blue staining and macrophage specific F4/80 immunohistochemistry (IHC) analysis. Quantitative real-time PCR analysis found significant expression of monocyte chemoattractant protein-1 (MCP-1) at 72 h post injury. Also, we found that flow cytometric analysis demonstrated a 7-fold increase in infiltrating macrophages around 72 h post injuries as compared to control. The MR imaging in combination with flow cytometric analysis enabled the dynamic measurement of macrophage infiltration at the injury site. This study may help in setting an optimal time window to intervene and prevent damage due to inflammation and to increase the therapeutic efficacy. (C) 2016 Elsevier Inc All rights reserved.

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TI Gamma-Secretase Inhibitors Attenuate Neurotrauma and Neurogenic Acute

Lung Injury in Rats by Rescuing the Accumulation of Hypertrophic

Microglia

SO CELLULAR PHYSIOLOGY AND BIOCHEMISTRY

LA English

DT Article

DE Traumatic brain injury; gamma-secretase; Neurogenic acute lung injury;

Microglia

ID TRAUMATIC BRAIN-INJURY; MOUSE MODEL; BLOOD-FLOW; ACTIVATION; CHF5074;

CELLS; INFLAMMATION; MODULATOR; PATHOLOGY; PEPTIDE

AB Background/Aims: In response to traumatic brain injury (TBI), activated microglia exhibit changes in their morphology from the resting ramified phenotype toward the activated hypertrophic or amoeboid phenotype. Here, we provide the first description of the mechanism underlying the neuroprotective effects of gamma-secretase inhibitors on TBI outcomes in rats. Methods: The neuroprotective effects of gamma-secretase inhibitors such as LY411575 or CHF5074 on TBI-induced neurotoxicity were analysed using a neurological motor function evaluation, cerebral contusion assay, immunohistochemical staining for microglia phenotypes, lung injury score and Evans Blue dye extravasation assay of brain and lung oedema. Results: Hypertrophic or amoeboid microglia accumulated in the injured cortex, the blood-brain-barrier was disrupted and neurological deficits and acute lung injury were observed 4 days after TBI in adult rats. However, a subcutaneous injection of LY411575 (5 mg/kg) or CHF5074 (30 mg/kg) immediately after TBI and once daily for 3 consecutive days post-TBI significantly attenutaed the accumulation of hypertrophic microglia in the injured brain, neurological injury, and neurogenic acute lung injury. Conclusion: Gamma-secretase inhibitors attenuated neurotrauma and neurogenic acute lung injury in rats by reducing the accumulation of hypertrophic microglia in the vicinity of the lesion. (c) 2017 The Author(s) Published by S. Karger AG, Basel

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TI Mesenchymal stem cells maintain the microenvironment of central nervous

system by regulating the polarization of macrophages/microglia after

traumatic brain injury

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DT Review

DE Mesenchymal stem cells; macrophage; microglia polarization; traumatic

brain injury; cell-based therapy; neuroinflammation

ID M2 MICROGLIA POLARIZATION; STROMAL CELLS; ANTIINFLAMMATORY PHENOTYPE;

PROGESTERONE-RECEPTOR; MACROPHAGE ACTIVATION; STEM/STROMAL CELLS;

ISCHEMIC-STROKE; ADIPOSE-TISSUE; NEUROINFLAMMATION; THERAPY

AB Mesenchymal stem cells (MSCs), which are regarded as promising candidates for cell replacement therapies, are able to regulate immune responses after traumatic brain injury (TBI). Secondary immune response following the mechanical injury is the essential factor leading to the necrosis and apoptosis of neural cells during and after the cerebral edema has subsided and there is lack of efficient agent that can mitigate such neuroinflammation in the clinical application. By means of three molecular pathways (prostaglandin E2 (PGE2), tumor-necrosis-factor-inducible gene 6 protein (TSG-6), and progesterone receptor (PR) and glucocorticoid receptors (GR)), MSCs induce the activation of macrophages/microglia and drive them polarize into the M2 phenotypes, which inhibits the release of pro-inflammatory cytokines and promotes tissue repair and nerve regeneration. The regulation of MSCs and the polarization of macrophages/microglia are dynamically changing based on the inflammatory environment. Under the stimulation of platelet lysate (PL), MSCs also promote the release of pro-inflammatory cytokines. Meanwhile, the statue of macrophages/microglia exerts significant effects on the survival, proliferation, differentiation and activation of MSCs by changing the niche of cells. They form positive feedback loops in maintaining the homeostasis after TBI to relieving the secondary injury and promoting tissue repair. MSC therapies have obtained great achievements in several central nervous system disease clinical trials, which will accelerate the application of MSCs in TBI treatment.

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TI Aging with a traumatic brain injury: Could behavioral morbidities and

endocrine symptoms be influenced by microglial priming?

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Review

DE Traumatic brain injury; Aging; Inflammation; Cognition; Microglia;

Endocrine dysfunction

ID HEAD-INJURY; SYSTEMIC INFLAMMATION; HORMONE DEFICIENCY; FOLLOW-UP;

CHILDREN; HYPOPITUITARISM; ACTIVATION; PLASTICITY; AGE; CHILDHOOD

AB A myriad of factors influence the developmental and aging process and impact health and life span. Mounting evidence indicates that brain injury, even moderate injury, can lead to lifetime of physical and mental health symptoms. Therefore, the purpose of this mini-review is to discuss how recovery from traumatic brain injury (TBI) depends on age-at-injury and how aging with a TBI affects long-term recovery. TBI initiates pathophysiological processes that dismantle circuits in the brain. In response, reparative and restorative processes reorganize circuits to overcome the injury-induced damage. The extent of circuit dismantling and subsequent reorganization depends as much on the initial injury parameters as other contributing factors, such as genetics and age. Age-at-injury influences the way the brain is able to repair itself, as a result of developmental status, extent of cellular senescence, and injury-induced inflammation. Moreover, endocrine dysfunction can occur with TBI. Depending on the age of the individual at the time of injury, endocrine dysfunction may disrupt growth, puberty, influence social behaviors, and possibly alter the inflammatory response. In turn, activation of microglia, the brain's immune cells, after injury may continue to fuel endocrine dysfunction. With age, the immune system develops and microglia become primed to subsequent challenges. Sustained inflammation and microglial activation can continue for weeks to months post-injury. This prolonged inflammation can influence developmental processes, behavioral performance and age-related decline. Overall, brain injury may influence the aging process and expedite glial and neuronal alterations that impact mental health. (C) 2016 Elsevier Inc. All rights reserved.

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TI Binge ethanol in adulthood exacerbates negative outcomes following

juvenile traumatic brain injury

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE Traumatic brain injury; Alcohol; Axon degeneration; Microglia;

Neuroinflammation; Learning and memory; Neurogenesis

ID SEIZURE-INDUCED NEUROGENESIS; INDUCED NEUROINFLAMMATION; ALCOHOLIC

BRAINS; SUBSTANCE-ABUSE; MICROGLIA; EXPRESSION; CELLS; EPIDEMIOLOGY;

RECEPTORS; DRINKING

AB Traumatic brain injuries (TBI) are a major public health problem with enormous costs in terms of health care dollars, lost productivity, and reduced quality of life. Alcohol is bidirectionally linked to TBI as many TBI patients are intoxicated at the time of their injury and we recently reported that, in accordance with human epidemiological data, animals injured during juvenile development self-administered significantly more alcohol as adults than did sham injured mice. There are also clinical data that drinking after TBI significantly reduces the efficacy of rehabilitation and leads to poorer long-term outcomes. In order to determine whether juvenile traumatic brain injury also increased the vulnerability of the brain to the toxic effects of high dose alcohol, mice were injured at 21 days of age and then seven weeks later treated daily with binge-like levels of alcohol 5 g/kg (by oral gavage) for ten days. Binge-like alcohol produced a greater degree of neuronal damage and neuroinflammation in mice that sustained a TBI. Further, mice that sustained a juvenile TBI exhibited mild learning and memory impairments in adulthood following binge alcohol and express a significant increase in hippocampal ectopic localization of newborn neurons. Taken together, these data provide strong evidence that a mild brain injury occurring early in life renders the brain highly vulnerable to the consequences of binge-like alcohol consumption. (C) 2016 Elsevier Inc. All rights reserved.

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TI Simvastatin Therapy in the Acute Stage of Traumatic Brain Injury

Attenuates Brain Trauma-Induced Depression-Like Behavior in Rats by

Reducing Neuroinflammation in the Hippocampus

SO NEUROCRITICAL CARE

LA English

DT Article

DE Fluid percussion injury; Depression-Like behavior; Forced swim;

Hippocampus; Maximal angle; Microglia; Tumor necrosis factor-alpha;

Simvastatin

ID LATERAL FLUID-PERCUSSION; NECROSIS-FACTOR-ALPHA; MICROGLIAL ACTIVATION;

NEURONAL INJURY; NITRIC-OXIDE; LIPID-LEVELS; STATINS; MODEL; INCREASE;

DEGENERATION

AB The antidepressant-like effects of simvastatin on traumatic brain injury (TBI) remain unclear. The present study aimed to investigate the neuroprotective effects of simvastatin and determine whether simvastatin attenuates TBI-induced depression-like behavior and, more specifically, acts as an antineuroinflammatory.

Anesthetized male Sprague-Dawley rats were divided into five groups: sham-operated controls, TBI controls, and TBI treatment with simvastatin 4, 10, or 20 mg/kg. Simvastatin was intraperitoneally injected 0, 24, and 48 h after TBI. The motor function was measured using an inclined plane, and depression-like behavior was evaluated using forced swimming tests. Neuronal apoptosis (markers: NeuN, TUNEL, caspase-3), microglia (marker: OX42) and astrocyte (marker: GFAP) activation, and TNF-alpha expression in the microglia and astrocytes of the hippocampal CA3 area were investigated using immunofluorescence assay. All parameters were measured on the 4th, 8th, and 15th day, or only on the 15th day after TBI.

TBI-induced depression-like behavior, which increased duration of immobility, was significantly attenuated by 20 mg simvastatin therapy on day 15 after TBI. TBI-induced neuronal apoptosis, microglia and astrocyte activation, and TNF-alpha expression in the microglia and astrocytes of the CA3 area of the hippocampus were significantly reduced by simvastatin treatment, particularly when 20 mg/kg was administered for 3 days.

Intraperitoneal injection of simvastatin attenuated TBI in rats during the acute stage by reducing neuronal apoptosis, microglia, and TNF-alpha expression, thereby resulting in a reduction of depressive-like behavior. Our results suggest that simvastatin may be a promising treatment for TBI-induced depression-like behavior.

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TI Lactate Shuttles in Neuroenergetics - Homeostasis, Allostasis and Beyond

SO FRONTIERS IN NEUROSCIENCE

LA English

DT Article

DE neuroenergetics; lactate; astrocyte-neuron lactate shuttle (ANLS);

astrocyte-microglia lactate shuttle (AMLS); neuropathology; traumatic

brain injury (TBI); neurodegenerative disease; infectious

neuroinflammatory disease

ID TRAUMATIC BRAIN-INJURY; CEREBRAL-BLOOD-FLOW; HUMAN VISUAL-CORTEX;

CEREBROSPINAL-FLUID; ENERGY-METABOLISM; IN-VIVO; AXONAL DEGENERATION;

SYNAPTIC ACTIVITY; OXIDATIVE STRESS; LACTIC-ACID

AB Understanding brain energy metabolism neuroenergetics is becoming increasingly important as it can be identified repeatedly as the source of neurological perturbations. Within the scientific community we are seeing a shift in paradigms from the traditional neurocentric view to that of a more dynamic, integrated one where astrocytes are no longer considered as being just supportive, and activated microglia have a profound influence. Lactate is emerging as the "good guy," contrasting its classical "bad guy" position in the now superseded medical literature. This review begins with the evolution of the concept of "lactate shuttles"; goes on to the recent shift in ideas regarding normal neuroenergetics (homeostasis) specifically, the astrocytc-neuron lactate shuttle; and progresses to covering the metabolic implications whereby homeostasis is lost a state of allostasis, and the function of microglia. The role of lactate, as a substrate and shuttle, is reviewed in light of allostatic stress, and beyond in an acute state of allostatic stress in terms of physical brain trauma, and reflected upon with respect to persistent stress as allostatic overload-neurodegenerative diseases. Finally, the recently proposed astrocyte-microglia lactate shuttle is discussed in terms of chronic neuroinflammatory infectious diseases, using tuberculous meningitis as an example. The novelty extended by this review is that the directionality of lactate, as shuttles in the brain, in neuropathophysiological states is emerging as crucial in neuroenergetics.

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TI Frequent mild head injury promotes trigeminal sensitivity concomitant

with microglial proliferation, astrocytosis, and increased neuropeptide

levels in the trigeminal pain system

SO JOURNAL OF HEADACHE AND PAIN

LA English

DT Article

DE Post-traumatic headache; Migraine; Concussion; Traumatic brain injury;

Trigeminal; Microglia; Astrocytosis; Calcitonin gene-related peptide

ID TRAUMATIC BRAIN-INJURY; GENE-RELATED PEPTIDE; POSTTRAUMATIC HEADACHE;

MILITARY PERSONNEL; NERVE-STIMULATION; MODEL; CONCUSSION; ALLODYNIA;

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AB Background: Frequent mild head injuries or concussion along with the presence of headache may contribute to the persistence of concussion symptoms.

Methods: In this study, the acute effects of recovery between mild head injuries and the frequency of injuries on a headache behavior, trigeminal allodynia, was assessed using von Frey testing up to one week after injury, while histopathological changes in the trigeminal pain pathway were evaluated using western blot, ELISA and immunohistochemistry.

Results: A decreased recovery time combined with an increased mild closed head injury (CHI) frequency results in reduced trigeminal allodynia thresholds compared to controls. The repetitive CHI group with the highest injury frequency showed the greatest reduction in trigeminal thresholds along with greatest increased levels of calcitonin gene-related peptide (CGRP) in the trigeminal nucleus caudalis. Repetitive CHI resulted in astrogliosis in the central trigeminal system, increased GFAP protein levels in the sensory barrel cortex, and an increased number of microglia cells in the trigeminal nucleus caudalis.

Conclusions: Headache behavior in rats is dependent on the injury frequency and recovery interval between mild head injuries. A worsening of headache behavior after repetitive mild head injuries was concomitant with increases in CGRP levels, the presence of astrocytosis, and microglia proliferation in the central trigeminal pathway. Signaling between neurons and proliferating microglia in the trigeminal pain system may contribute to the initiation of acute headache after concussion or other traumatic brain injuries.

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TI [<SUP>18</SUP>F]FDG-PET Combined with MRI Elucidates the Pathophysiology

of Traumatic Brain Injury in Rats

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE astrocytes; brain injury; FDG-PET; inflammation; microglia

ID CORTICAL IMPACT INJURY; POSITRON-EMISSION-TOMOGRAPHY; MICROGLIA

ACTIVATION; NEURONAL DEATH; UNITED-STATES; GLUCOSE; PATHOLOGY; RECEPTOR;

TIME

AB Non-invasive measurements of brain metabolism using F-18-fluorodeoxyglucose (FDG) with positron emission tomography (PET) may provide important information about injury severity following traumatic brain injury (TBI). There is growing interest in the potential of combining functional PET imaging with anatomical and functional magnetic resonance imaging (MRI). This study aimed to investigate the effectiveness of combining clinically available FDG-PET with T2 and diffusion MR imaging, with a particular focus on inflammation and the influence of glial alterations after injury. Adult male Sprague Dawley rats underwent a moderate controlled cortical impact (CCI) injury followed by FDG-PET, MRI, and histological evaluation. FDG uptake showed significant alterations in the corpus callosum, hippocampus, and amygdala after TBI, demonstrating that a relatively focal CCI injury can result in global alterations. Analysis of MRI T2 intensity and apparent diffusion coefficient (ADC) also showed significant alterations in these regions to include cytotoxic and vasogenic edema. Histology showed increased glial activation in the corpus callosum and hippocampus that was associated with increased FDG uptake at sub-acute time-points. Glial activation was not detected in the amygdala but neuronal damage was evident, as the amygdala was the only region to show a reduction in both FDG uptake and ADC at sub-acute time-points. Overall, FDG-PET detected glial activation but was confounded by the presence of cell damage, whereas MRI consistently detected cell damage but was confounded by glial activation. These results demonstrate that FDG-PET and MRI can be used together to improve our understanding of the complex alterations in the brain after TBI.

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TI Salsalate treatment fallowing traumatic brain injury reduces

inflammation and promotes a neuroprotective and neurogenic

transcriptional response with concomitant functional recovery

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE TBI; Myeloid cells; Microglia; NF-kappaB; Salicylate; Neuroprotection;

Neuroregeneration; Oxytocin; Motor function; NSAID

ID NF-KAPPA-B; ACID RECEPTOR-BETA; NEURONAL DEGENERATION; REACTIVE

ASTROCYTES; INSULIN-RESISTANCE; NEURODEGENERATION; SALICYLATE;

EXPRESSION; OXYTOCIN; GENES

AB Neuroinflammation plays a critical role in the pathogenesis of traumatic brain injury (TBI). TBI induces rapid activation of astrocytes and microglia, infiltration of peripheral leukocytes, and secretion of inflammatory cytokines. In the context of modest or severe TBI, such inflammation contributes to tissue destruction and permanent brain damage. However, it is clear that the inflammatory response is also necessary to promote post-injury healing. To date, anti-inflammatory therapies, including the broad class of non-steroidal anti-inflammatory drugs (NSAIDs), have met with little success in treatment of TBI, perhaps because these drugs have inhibited both the tissue-damaging and repair-promoting aspects of the inflammatory response, or because inhibition of inflammation alone is insufficient to yield therapeutic benefit. Salsalate is an unacetylated salicylate with long history of use in limiting inflammation. This drug is known to block activation of NF-KB, and recent data suggest that salsalate has a number of additional biological activities, which may also contribute to its efficacy in treatment of human disease. Here, we show that salsalate potently blocks pro-inflammatory gene expression and nitrite secretion by microglia in vitro. Using the controlled cortical impact (CCI) model in mice, we find that salsalate has a broad antiinflammatory effect on in vivo TBI-induced gene expression, when administered post-injury. Interestingly, salsalate also elevates expression of genes associated with neuroprotection and neurogenesis, including the neuropeptides, oxytocin and thyrotropin releasing hormone. Histological analysis reveals salsalate-dependent decreases in numbers and activation-associated morphological changes in microglia/macrophages, proximal to the injury site. Flow cytometry data show that salsalate changes the kinetics of CCI-induced accumulation of various populations of CD11b-positive myeloid cells in the injured brain. Behavioral assays demonstrate that salsalate treatment promotes significant recovery of function following CCI. These pre-clinical data suggest that salsalate may show promise as a TBI therapy with a multifactorial mechanism of action to enhance functional recovery.

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TI Microglial-derived microparticles mediate neuroinflammation after

traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Microparticles; Microglia; Neuroinflammation; Traumatic brain injury;

Interleukin-1 beta; miR-155

ID NANOPARTICLE TRACKING ANALYSIS; INDUCED NEUTROPHIL ACTIVATION; IMPROVES

FUNCTIONAL RECOVERY; IN-VIVO; EXTRACELLULAR VESICLES; VASCULAR INJURY;

PLATELET MICROPARTICLES; INFLAMMATORY RESPONSE; ALZHEIMERS-DISEASE;

OXIDATIVE STRESS

AB Background: Local and systemic inflammatory responses are initiated early after traumatic brain injury (TBI), and may play a key role in the secondary injury processes resulting in neuronal loss and neurological deficits. However, the mechanisms responsible for the rapid expansion of neuroinflammation and its long-term progression have yet to be elucidated. Here, we investigate the role of microparticles (MP), a member of the extracellular vesicle family, in the exchange of pro-inflammatory molecules between brain immune cells, as well as their transfer to the systemic circulation, as key pathways of inflammation propagation following brain trauma.

Methods: Adult male C57BL/6 mice were subjected to controlled cortical impact TBI for 24 h, and enriched MP were isolated in the blood, while neuroinflammation was assessed in the TBI cortex. MP were characterized by flow cytometry, and MP content was assayed using gene and protein markers for pro-inflammatory mediators. Enriched MP co-cultured with BV2 or primary microglial cells were used for immune propagation assays. Enriched MP from BV2 microglia or CD11b-positive microglia from the TBI brain were stereotactically injected into the cortex of uninjured mice to evaluate MP-related seeding of neuroinflammation in vivo.

Results: As the neuroinflammatory response is developing in the brain after TBI, microglial-derived MP are released into the circulation. Circulating enriched MP from the TBI animals can activate microglia in vitro. Lipopolysaccharide stimulation increases MP release from microglia in vitro and enhances their content of pro-inflammatory mediators, interleukin-1 beta and microRNA-155. Enriched MP from activated microglia in vitro or CD11b-isolated microglia/macrophage from the TBI brain ex vivo are sufficient to initiate neuroinflammation following their injection into the cortex of naive (uninjured) animals.

Conclusions: These data provide further insights into the mechanisms underlying the development and dissemination of neuroinflammation after TBI. MP loaded with pro-inflammatory molecules initially released by microglia following trauma can activate additional microglia that may contribute to progressive neuroinflammatory response in the injured brain, as well as stimulate systemic immune responses. Due to their ability to independently initiate inflammatory responses, MP derived from activated microglia may provide a potential target for other neurological disorders in which neuroinflammation may be a contributing factor.

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TI Hyperbaric Oxygen Effects on Depression-Like Behavior and

Neuroinflammation in Traumatic Brain Injury Rats

SO WORLD NEUROSURGERY

LA English

DT Article

DE Depression-like behavior; Fluid percussion injury; Forced swim;

Hippocampus; Microglia; Tumor necrosis factor-alpha

ID LATERAL FLUID-PERCUSSION; NECROSIS-FACTOR-ALPHA; FORCED-SWIMMING TEST;

MICROGLIAL ACTIVATION; MODEL; HIPPOCAMPUS; SEROTONIN; THERAPY;

ANGIOGENESIS; STIMULATION

AB OBJECTIVE: The aim of this study was to determine whether hyperbaric oxygen (HBO) therapy causes attenuation of traumatic brain injury (TBI)-induced depressionlike behavior and its associated anti-neuroinflammatory effects after fluid percussion injury.

METHODS: Anesthetized male Sprague-Dawley rats were divided into 3 groups: sham operation plus normobaric air (NBA) (21% oxygen at 1 absolute atmosphere [ATA]), TBI plus NBA, and TBI plus HBO (100% oxygen at 2.0 ATA). HBO was applied immediately for 60 min/d after TBI for 3 days. Depression-like behavior was tested by a forced swimming test, motor function was tested by an inclined plane test, and infarction volume was tested by triphenyltetrazolium chloride (TTC) staining on days 4, 8, and 15. Neuronal apoptosis (terminal deoxynucleotidyl transferase dUTP nick-end labeling assay), microglial (marker OX42) activation, and tumor necrosis factor (TNF)-alpha expression in microglia in the hippocampus CA3 were measured by immunofluorescence methods.

RESULTS: Compared with the TBI controls, without significant changes in TTC staining or in the motor function test, TBI-induced depression-like behavior was significantly attenuated by HBO therapy by day 15 after TBI. Simultaneously, TBI-induced neuronal apoptosis, microglial (marker OX42) activation, and TNF-alpha expression in the microglia in the hippocampus CA3 were significantly reduced by HBO.

CONCLUSIONS: Our results suggest that HBO treatment may ameliorate TBI-induced depression-like behavior in rats by attenuating neuroinflammation, representing one possible mechanism by which depression-like behavior recovery might occur. We also recommend HBO as a potential treatment for TBI-induced depression-like behavior if early intervention is possible.

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TI Role of Microglia Autophagy in Microglia Activation After Traumatic

Brain Injury

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LA English

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DE Apoptosis; Autophagy; Microglia; Traumatic brain injury

ID CONTROLLED CORTICAL IMPACT; NECROSIS-FACTOR-ALPHA; MODERATE HYPOTHERMIA;

CELL-DEATH; ALZHEIMERS-DISEASE; CEREBRAL-ISCHEMIA; EXPRESSION;

RAPAMYCIN; MICE; INTERLEUKIN-1

AB OBJECTIVE: We evaluated the role of microglia autophagy in microglia activation after traumatic brain injury (TBI) in rats.

METHODS: TBI was induced by a fluid percussion TBI device. All rats were killed 24 hours after TBI. The ipsilateral hippocampus in all rats was analyzed with hematoxylin-eosin staining. Immunohistochemistry and Western blotting of ionized calcium-binding adapter molecule 1 was used to determine changes in microglia activation. Double staining of microtubule-associated protein light chain 3, Beclin-1, and ionized calcium-binding adapter molecule 1 was used to assess changes of microglia autophagy. Enzyme-linked immunosorbent assay of tumor necrosis factor-alpha and interleukin-1 beta was used to evaluate changes in inflammatory responses. Terminal deoxyribonucleotidyl transferase-mediated deoxyuridine 5'-triphosphate nick-end labeling staining was used to determine cell death in the ipsilateral hippocampus.

RESULTS: At 24 hours after TBI, microglial cells became activated, and the autophagy inhibitor 3-methyladenine (3-MA) further promoted microglia activation. Protein light chain 3- and Beclin-1-positive microglial cells were increased after TBI, whereas 3-MA decreased the number of positive microglial cells, increasing the expression of tumor necrosis factor-alpha and interleukin-1b; terminal deoxyribonucleotidyl transferase-mediated deoxyuridine 5'-triphosphate nick-end labeling staining demonstrated that 3-MA could increase the number of terminal deoxyribonucleotidyl transferase-mediated deoxyuridine 5'-triphosphate nick-end labeling-positive cells (16.83 +/- 0.83 vs. 11 +/- 0.82, P < 0.001).

CONCLUSIONS: Our data demonstrated that TBI induced microglia activation and microglia autophagy. Inhibition of microglia autophagy with 3-MA increased microglia activation and neural apoptosis. These findings indicate that targeting microglia autophagy may be a therapeutic strategy for TBI.

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TI Rod-shaped microglia morphology is associated with aging in 2 human

autopsy series

SO NEUROBIOLOGY OF AGING

LA English

DT Article

DE Aging; Microglia activation; Neurodegeneration; Neuroinflammation;

Neuropathology; Hippocampus; Alzheimer's disease; Traumatic brain injury

ID FRACTALKINE RECEPTOR; ALZHEIMERS-DISEASE; BRAIN-INJURY; IMPAIRMENT;

PATHOLOGY; DEMENTIA; NEURONS; RISK; MICE

AB A subtype of microglia is defined by the morphological appearance of the cells as rod shaped. Little is known about this intriguing cell type, as there are only a few case reports describing rod-shaped microglia in the neuropathological literature. Rod-shaped microglia were shown recently to account for a substantial proportion of the microglia cells in the hippocampus of both demented and cognitively intact aged individuals. We hypothesized that aging could be a defining feature in the occurrence of rodshaped microglia. To test this hypothesis, 2 independent series of autopsy cases (total n 168 cases), which covered the adult lifespan from 20 to 100\_ years old, were included in the study. The presence or absence of rod-shaped microglia was scored on IBA1 immunohistochemically stained slides for the hippocampus and cortex. We found that age was one of the strongest determinants for the presence of rod-shaped microglia in the hippocampus and the cortex. We found no association with the presence of rod-shaped microglia and a self-reported history of a TBI. Alzheimer's diseaseerelated pathology was found to influence the presence of rod-shaped microglia, but only in the parietal cortex and not in the hippocampus or temporal cortex. Future studies are warranted to determine the functional relevance of rod-shaped microglia in supporting the health of neurons in the aged brain, and the signaling processes that regulate the formation of rod-shaped microglia. (C) 2017 Elsevier Inc. All rights reserved.

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TI Rapid neuroinflammatory response localized to injured neurons after

diffuse traumatic brain injury in swine

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Neuroinflammation; Microglia reactivity; Diffuse traumatic brain injury;

Permeabilized neurons; Concussion

ID AXONAL INJURY; MICROGLIAL RESPONSE; HEAD-INJURY; IN-VIVO; DAMAGE;

PERMEABILITY; IMPACT; MICE; DEGENERATION; INFLAMMATION

AB Despite increasing appreciation of the critical role that neuroinflammatory pathways play in brain injury and neurodegeneration, little is known about acute microglial reactivity following diffuse traumatic brain injury (TBI) - the most common clinical presentation that includes all concussions. Therefore, we investigated acute microglial reactivity using a porcine model of closed-head rotational velocity/acceleration-induced TBI that closely mimics the biomechanical etiology of inertial TBI in humans. We observed rapid microglial reactivity within 15 min of both mild and severe TBI. Strikingly, microglial activation was restrained to regions proximal to individual injured neurons - as denoted by trauma-induced plasma membrane disruption - which served as epicenters of acute reactivity. Single-cell quantitative analysis showed that in areas free of traumatically permeabilized neurons, microglial density and morphology were similar between sham or following mild or severe TBI. However, microglia density increased and morphology shifted to become more reactive in proximity to injured neurons. Microglial reactivity around injured neurons was exacerbated following repetitive TBI, suggesting further amplification of acute neuroinflammatory responses. These results indicate that neuronal trauma rapidly activates microglia in a highly localized manner, and suggest that activated microglia may rapidly influence neuronal stability and/or pathophysiology after diffuse TBI. (C) 2017 Elsevier Inc. All rights reserved.

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TI Repetitive Model of Mild Traumatic Brain Injury Produces Cortical

Abnormalities Detectable by Magnetic Resonance Diffusion Imaging,

Histopathology, and Behavior

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE concussion; diffusion kurtosis imaging; diffusion tensor imaging;

microglia; Thy1-YFP

ID WHITE-MATTER; AXONAL INJURY; DIFFERENTIAL DETECTION; CHRONIC

DEMYELINATION; COGNITIVE IMPAIRMENT; CORPUS-CALLOSUM; GLUCOSE-UPTAKE;

MOUSE MODEL; HEAD-INJURY; TRACK-TBI

AB Noninvasive detection of mild traumatic brain injury (mTBI) is important for evaluating acute through chronic effects of head injuries, particularly after repetitive impacts. To better detect abnormalities from mTBI, we performed longitudinal studies (baseline, 3, 6, and 42 days) using magnetic resonance diffusion tensor imaging (DTI) and diffusion kurtosis imaging (DKI) in adult mice after repetitive mTBI (r-mTBI; daily x 5) or sham procedure. This r-mTBI produced righting reflex delay and was first characterized in the corpus callosum to demonstrate low levels of axon damage, astrogliosis, and microglial activation, without microhemorrhages. High-resolution DTI-DKI was then combined with post-imaging pathological validation along with behavioral assessments targeted for the impact regions. In the corpus callosum, only DTI fractional anisotropy at 42 days showed significant change post-injury. Conversely, cortical regions under the impact site (M1-M2, anterior cingulate) had reduced axial diffusivity (AD) at all time points with a corresponding increase in axial kurtosis (K-a) at 6 days. Post-imaging neuropathology showed microglial activation in both the corpus callosum and cortex at 42 days after r-mTBI. Increased cortical microglial activation correlated with decreased cortical AD after r-mTBI (r=-0.853; n = 5). Using Thy1-YFP-16 mice to fluorescently label neuronal cell bodies and processes revealed low levels of axon damage in the cortex after r-mTBI. Finally, r-mTBI produced social deficits consistent with the function of this anterior cingulate region of cortex. Overall, vulnerability of cortical regions is demonstrated after mild repetitive injury, with underlying differences of DTI and DKI, microglial activation, and behavioral deficits.

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TI Microstructural and Microglial Changes After Repetitive Mild Traumatic

Brain Injury in Mice

SO JOURNAL OF NEUROSCIENCE RESEARCH

LA English

DT Article

DE DTI; microhemorrhage; inflammation; SWI; axial diffusion;

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ID TUMOR-NECROSIS-FACTOR; DIFFUSE AXONAL INJURY; WHITE-MATTER; CEREBRAL

MICROBLEEDS; MOUSE MODEL; SPINAL-CORD; TENSOR; RECEPTOR; TRACTOGRAPHY;

VALIDATION

AB Traumatic brain injury (TBI) is a major public health issue, with recently increased awareness of the potential long-term sequelae of repetitive injury. Although TBI is common, objective diagnostic tools with sound neurobiological predictors of outcome are lacking. Indeed, such tools could help to identify those at risk for more severe outcomes after repetitive injury and improve understanding of biological underpinnings to provide important mechanistic insights. We tested the hypothesis that acute and subacute pathological injury, including the microgliosis that results from repeated mild closed head injury (rmCHI), is reflected in susceptibility-weighted magnetic resonance imaging and diffusion-tensor imaging microstructural abnormalities. Using a combination of high-resolution magnetic resonance imaging, stereology, and quantitative PCR, we studied the pathophysiology of male mice that sustained seven consecutive mild traumatic brain injuries over 9 days in acute (24 hr) and subacute (1 week) time periods. rmCHI induced focal cortical microhemorrhages and impaired axial diffusivity at 1 week postinjury. These microstructural abnormalities were associated with a significant increase in microglia. Notably, microgliosis was accompanied by a change in inflammatory microenvironment defined by robust spatiotemporal alterations in tumor necrosis factor-a receptor mRNA. Together these data contribute novel insight into the fundamental biological processes associated with repeated mild brain injury concomitant with subacute imaging abnormalities in a clinically relevant animal model of repeated mild TBI. These findings suggest new diagnostic techniques that can be used as biomarkers to guide the use of future protective or reparative interventions. (C) 2016 Wiley Periodicals, Inc.

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TI Establishing the ferret as a gyrencephalic animal model of traumatic

brain injury: Optimization of controlled cortical impact procedures

SO JOURNAL OF NEUROSCIENCE METHODS

LA English

DT Article

DE Surgery; Behavior; Immunohistochemistry; Landmarks; Microglia;

Astrocytes; MRI; Craniotomy; TBI; CCI; Temporalis; Antibiotics;

Reproducibility; White matter; Cognitive; Motor; Impairment; Acute;

Chronic; Inflammation

ID MOUSE MODEL; MRI; NEURODEGENERATION; ABNORMALITIES; INFLAMMATION;

IMPAIRMENT; ESTROGEN; BEHAVIOR; DEFICIT; FEMALE

AB Background: Although rodent TBI studies provide valuable information regarding the effects of injury and recovery, an animal model with neuroanatomical characteristics closer to humans may provide a more meaningful basis for clinical translation. The ferret has a high white/gray matter ratio, gyrencephalic neocortex, and ventral hippocampal location. Furthermore, ferrets are amenable to behavioral training, have a body size compatible with pre-clinical MRI, and are cost-effective.

New methods: We optimized the surgical procedure for controlled cortical impact (CCI) using 9 adult male ferrets. We used subject-specific brain/skull morphometric data from anatomical MRIs to overcome across-subject variability for lesion placement. We also reflected the temporalis muscle, closed the craniotomy, and used antibiotics. We then gathered MRI, behavioral, and immunohistochemical data from 6 additional animals using the optimized surgical protocol: 1 control, 3 mild, and 1 severely injured animals (surviving one week) and 1 moderately injured animal surviving sixteen weeks.

Results: The optimized surgical protocol resulted in consistent injury placement. Astrocytic reactivity increased with injury severity showing progressively greater numbers of astrocytes within the white matter. The density and morphological changes of microglia amplified with injury severity or time after injury. Motor and cognitive impairments scaled with injury severity.

Comparison with existing method(s): The optimized surgical methods differ from those used in the rodent, and are integral to success using a ferret model.

Conclusions: We optimized ferret CCI surgery for consistent injury placement. The ferret is an excellent animal model to investigate pathophysiological and behavioral changes associated with TBI. Published by Elsevier B.V.

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TI Microglial Activation in Traumatic Brain Injury

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DE traumatic brain injury; CCI; microglia; neuroinflammation; TSPO;

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ID CENTRAL-NERVOUS-SYSTEM; CLOSED-HEAD INJURY; PROTEIN 18 KDA; IMPROVES

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PROTEIN; CONTROLLED CORTICAL IMPACT; PERIPHERAL BENZODIAZEPINE; IN-VIVO;

AXONAL INJURY

AB Microglia have a variety of functions in the brain, including synaptic pruning, CNS repair and mediating the immune response against peripheral infection. Microglia rapidly become activated in response to CNS damage. Depending on the nature of the stimulus, microglia can take a number of activation states, which correspond to altered microglia morphology, gene expression and function. It has been reported that early microglia activation following traumatic brain injury (TBI) may contribute to the restoration of homeostasis in the brain. On the other hand, if they remain chronically activated, such cells display a classically activated phenotype, releasing pro-inflammatory molecules, resulting in further tissue damage and contributing potentially to neurodegeneration. However, new evidence suggests that this classification is over-simplistic and the balance of activation states can vary at different points. In this article, we review the role of microglia in TBI, analyzing their distribution, morphology and functional phenotype over time in animal models and in humans. Animal studies have allowed genetic and pharmacological manipulations of microglia activation, in order to define their role. In addition, we describe investigations on the in vivo imaging of microglia using translocator protein (TSPO) PET and autoradiography, showing that microglial activation can occur in regions far remote from sites of focal injuries, in humans and animal models of TBI. Finally, we outline some novel potential therapeutic approaches that prime microglia/macrophages toward the beneficial restorative microglial phenotype after TBI.

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TI IL-2/Anti-IL-2 Complex Attenuates Inflammation and BBB Disruption in

Mice Subjected to Traumatic Brain Injury

SO FRONTIERS IN NEUROLOGY

LA English

DT Article

DE traumatic brain injury; IL-2/anti-IL-2; regulatory T cells; microglia;

inflammation; blood-brain barrier

ID REGULATORY T-CELLS; CONTROLLED CORTICAL IMPACT; INTRACEREBRAL

HEMORRHAGE; CEREBRAL-ISCHEMIA; IMMUNE-SYSTEM; SPINAL-CORD; TGF-BETA;

MICROGLIA; STROKE; ACTIVATION

AB Traumatic brain injury (TBI) induces the excessive inflammation and disruption of blood-brain barrier, both of which are partially mediated by the activation of microglia and release of inflammatory cytokines. Previous reports showed that administration of regulatory T cells (Tregs) could suppress inflammation and promote neurological function recovery, and that the IL-2/anti-IL-2 complex (IL-2C) could increase the number of Tregs. Thus, we hypothesized that IL-2C-mediated expansion of Tregs would be beneficial in mice subjected to TBI. In this study, mice received an intraperitoneal injection of IL-2C for three consecutive days. We observed that IL-2C dose-dependently increased Tregs without affecting the populations of CD4, CD8, or natural killer cells. IL-2C could improve the neurological recovery and reduce brain edema, tissue loss, neutrophils infiltration, and tight junction proteins degradation. Furthermore, this complex could also reduce the expression of CD16/32, IL-1 beta, or TNF-alpha, and elevate the expression of CD206, arginase 1, or TGF-beta. These results suggest that IL-2C could be a potential therapeutic method to alleviate excessive inflammation and maintain blood vessel stability after TBI.

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TI Role of microglia in a mouse model of paediatric traumatic brain injury

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE Phenotype; Cytokine; Chemokine; Apoptosis; Neuron; Immature; Cerebral;

Macrophage; Inflammation; Minocycline

ID WHITE-MATTER INJURY; CELL-DEATH; MICROGLIA/MACROPHAGE ACTIVATION;

CEREBRAL-ISCHEMIA; RAT-BRAIN; MINOCYCLINE; EXPRESSION; PROTECTS; AGE;

NEUROPROTECTION

AB The cognitive and behavioural deficits caused by traumatic brain injury (TBI) to the immature brain are more severe and persistent than TBI in the mature brain. Understanding this developmental sensitivity is critical as children under four years of age sustain TBI more frequently than any other age group. Microglia (MG), resident immune cells of the brain that mediate neuroinflammation, are activated following TBI in the immature brain. However, the type and temporal profile of this activation and the consequences of altering it are still largely unknown.

In a mouse model of closed head weight drop paediatric brain trauma, we characterized i) the temporal course of total cortical neuroinflammation and the phenotype of ex vivo isolated CD11B-positive microglia/macrophage (MG/M Phi) using a battery of 32 markers, and ii) neuropathological outcome 1 and 5 days post-injury. We also assessed the effects of targeting MG/M Phi activation directly, using minocycline a prototypical microglial activation antagonist, on these processes and outcome.

TBI induced a moderate increase in both pro- and anti-inflammatory cytokines/chemokines in the ipsilateral hemisphere. Isolated cortical MG/M Phi expressed increased levels of markers of endogenous reparatory/regenerative and immunomodulatory phenotypes compared with shams. Blocking MG/M Phi activation with minocycline at the time of injury and 1 and 2 days post-injury had only transient protective effects, reducing ventricular dilatation and cell death 1 day post-injury but having no effect on injury severity at 5 days.

This study demonstrates that, unlike in adults, the role of MG/M Phi in injury mechanisms following TBI in the immature brain may not be negative. An improved understanding of MG/M Phi function in paediatric TBI could support translational efforts to design therapeutic interventions. (C) 2016 The Author(s). Published by Elsevier Inc.

C1 [Chhor, Vibol; Moretti, Raffaella; Le Charpentier, Tifenn; Sigaut, Stephanie; Lebon, Sophie; Schwendimann, Leslie; Ore, Marie-Virginie; Zuiani, Chiara; Milan, Valentina; Josserand, Julien; Pansiot, Julien; Degos, Vincent; Titomanlio, Luigi; Gressens, Pierre; Fleiss, Bobbi] Unversite Paris Diderot, PROTECT, INSERM, Sorbonne Paris Cite, Paris, France.

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TI Omega-3 polyunsaturated fatty acid supplementation attenuates

microglial-induced inflammation by inhibiting the HMGB1/TLR4/NF-κB

pathway following experimental traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Traumatic brain injury; Omega-3 polyunsaturated fatty acid; Microglia;

Neuroinflammation; HMGB1/TLR4/NF-kappa B pathway

ID EXPERIMENTAL SUBARACHNOID HEMORRHAGE; GROUP BOX 1; DOCOSAHEXAENOIC ACID;

SIGNALING PATHWAY; ACTIVATION; OMEGA-3-FATTY-ACIDS; EXPRESSION;

NEUROPROTECTION; METASTASIS; INCREASE

AB Background: Microglial activation and the subsequent inflammatory response in the central nervous system play important roles in secondary damage after traumatic brain injury (TBI). High-mobility group box 1 (HMGB1) protein, an important mediator in late inflammatory responses, interacts with transmembrane receptor for advanced glycation end products (RAGE) and toll-like receptors (TLRs) to activate downstream signaling pathways, such as the nuclear factor (NF)-kappa B signaling pathway, leading to a cascade amplification of inflammatory responses, which are related to neuronal damage after TBI. Omega-3 polyunsaturated fatty acid (omega-3 PUFA) is a commonly used clinical immunonutrient, which has antioxidative and anti-inflammatory effects. However, the effects of omega-3 PUFA on HMGB1 expression and HMGB1-mediated activation of the TLR4/NF-kappa B signaling pathway are not clear.

Methods: The Feeney DM TBI model was adopted to induce brain injury in rats. Modified neurological severity scores, brain water content, and Nissl staining were employed to determine the neuroprotective effects of omega-3 PUFA supplementation. Assessment of microglial activation in lesioned sites and protein markers for proinflammatory, such as tumor necrosis factor (TNF)-alpha, interleukin (IL)-1 beta, IL-6, interferon (IFN)-gamma, and HMGB1 were used to evaluate neuroinflammatory responses and anti-inflammation effects of omega-3 PUFA supplementation. Immunofluorescent staining and western blot analysis were used to detect HMGB1 nuclear translocation, secretion, and HMGB1-mediated activation of the TLR4/NF-kappa B signaling pathway to evaluate the effects of omega-3 PUFA supplementation and gain further insight into the mechanisms underlying the development of the neuroinflammatory response after TBI.

Results: It was found that omega-3 PUFA supplementation inhibited TBI-induced microglial activation and expression of inflammatory factors (TNF-alpha, IL-1 beta, IL-6, and IFN-gamma), reduced brain edema, decreased neuronal apoptosis, and improved neurological functions after TBI. We further demonstrated that omega-3 PUFA supplementation inhibited HMGB1 nuclear translocation and secretion and decreased expression of HMGB1 in neurons and microglia in the lesioned areas. Moreover, omega-3 PUFA supplementation inhibited microglial activation and the subsequent inflammatory response by regulating HMGB1 and the TLR4/NF-kappa B signaling pathway.

Conclusions: The results of this study suggest that microglial activation and the subsequent neuroinflammatory response as well as the related HMGB1/TLR4/NF-kappa B signaling pathway play essential roles in secondary injury after TBI. Furthermore, omega-3 PUFA supplementation inhibited TBI-induced microglial activation and the subsequent inflammatory response by regulating HMGB1 nuclear translocation and secretion and also HMGB1-mediated activation of the TLR4/NF-kappa B signaling pathway, leading to neuroprotective effects.

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TI Posttraumatic therapeutic hypothermia alters microglial and macrophage

polarization toward a beneficial phenotype

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE Cytokines; inflammation; macrophages; microglial; traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; SPINAL-CORD-INJURY; BARRIER PERMEABILITY;

MODERATE HYPOTHERMIA; WHITE-MATTER; RAT; NEUROINFLAMMATION; ACTIVATION;

NEURODEGENERATION; NEUROPROTECTION

AB Posttraumatic inflammatory processes contribute to pathological and reparative processes observed after traumatic brain injury (TBI). Recent findings have emphasized that these divergent effects result from subsets of proinflammatory (M1) or anti-inflammatory (M2) microglia and macrophages. Therapeutic hypothermia has been tested in preclinical and clinical models of TBI to limit secondary injury mechanisms including proinflammatory processes. This study evaluated the effects of posttraumatic hypothermia (PTH) on phenotype patterns of microglia/ macrophages. Sprague-Dawley rats underwent moderate fluid percussion brain injury with normothermia (37 degrees C) or hypothermia (33 degrees C). Cortical and hippocampal regions were analyzed using flow cytometry and reverse transcription-polymerase chain reaction (RT-PCR) at several periods after injury. Compared to normothermia, PTH attenuated infiltrating cortical macrophages positive for CD11b(+) and CD45 high. At 24 h, the ratio of iNOS(+) (M1) to arginase(+) (M2) cells after hypothermia showed a decrease compared to normothermia. RT-PCR of M1-associated genes including iNOS and IL-1 beta was significantly reduced with hypothermia while M2-associated genes including arginase and CD163 were significantly increased compared to normothermic conditions. The injury-induced increased expression of the chemokine Ccl2 was also reduced with PTH. These studies provide a link between temperature-sensitive alterations in macrophage/microglia activation and polarization toward a M2 phenotype that could be permissive for cell survival and repair.

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JI J. Cereb. Blood Flow Metab.

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TI Anti-inflammatory and immunomodulatory mechanisms of atorvastatin in a

murine model of traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Traumatic brain injury; Atorvastatin; Immunomodulation;

Anti-inflammation; Leukocyte; Microglia/macrophage subtype

ID NATURAL-KILLER-CELLS; T-CELLS; INTRACEREBRAL HEMORRHAGE;

MICROGLIA/MACROPHAGE POLARIZATION; MACROPHAGE INFILTRATION; INFLAMMATORY

REACTION; NEUROPROTECTION; STATINS; TARGET; SYNAPTOGENESIS

AB Background: Neuroinflammation is an important secondary injury mechanism that has dual beneficial and detrimental roles in the pathophysiology of traumatic brain injury (TBI). Compelling data indicate that statins, a group of lipid-lowering drugs, also have extensive immunomodulatory and anti-inflammatory properties. Among statins, atorvastatin has been demonstrated as a neuroprotective agent in experimental TBI; however, there is a lack of evidence regarding its effects on neuroinflammation during the acute phase of TBI. The current study aimed to evaluate the effects of atorvastatin therapy on modulating the immune reaction, and to explore the possible involvement of peripheral leukocyte invasion and microglia/macrophage polarization in the acute period post-TBI.

Methods: C57BL/6 mice were subjected to TBI using a controlled cortical impact (CCI) device. Either atorvastatin or vehicle saline was administered orally starting 1 h post-TBI for three consecutive days. Short-term neurological deficits were evaluated using the modified neurological severity score (mNSS) and Rota-rod. Brain-invading leukocyte subpopulations were analyzed by flow cytometry and immunohistochemistry. Pro-and anti-inflammatory cytokines and chemokines were examined using enzyme-linked immunosorbent assay (ELISA). Markers of classically activated (M1) and alternatively activated (M2) microglia/macrophages were then determined by quantitative real-time PCR (qRT-PCR) and flow cytometry. Neuronal apoptosis was identified by double staining of terminal deoxynucleotidyl transferase-dUTP nick end labeling (TUNEL) staining and immunofluorescence labeling for neuronal nuclei (NeuN).

Results: Acute treatment with atorvastatin at doses of 1 mg/kg/day significantly reduced neuronal apoptosis and improved behavioral deficits. Invasions of T cells, neutrophils and natural killer (NK) cells were attenuated profoundly after atorvastatin therapy, as was the production of pro-inflammatory cytokines (IFN-gamma and IL-6) and chemokines (RANTES and IP-10). Notably, atorvastatin treatment significantly increased the proportion of regulatory T cells (Tregs) in both the peripheral spleen and brain, and at the same time, increased their main effector cytokines IL-10 and TGF-beta 1. We also found that atorvastatin significantly attenuated total microglia/macrophage activation but augmented the M2/M1 ratio by both inhibiting M1 polarization and enhancing M2 polarization.

Conclusions: Our data demonstrated that acute atorvastatin administration could modulate post-TBI neuroinflammation effectively, via a mechanism that involves altering peripheral leukocyte invasion and the alternative polarization of )microglia/macrophages.

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TI Sexual dimorphism in the inflammatory response to traumatic brain injury

SO GLIA

LA English

DT Article

DE cytokine; inflammation; microglia; sex-differences; trauma

ID MICROGLIAL ACTIVATION; ISCHEMIC-INJURY; FEMALE RATS; ESTROGEN; GENDER;

NEURODEGENERATION; NEUROPROTECTION; EXPRESSION; NEUROINFLAMMATION;

PROGESTERONE

AB The activation of resident microglial cells, alongside the infiltration of peripheral macrophages, are key neuroinflammatory responses to traumatic brain injury (TBI) that are directly associated with neuronal death. Sexual disparities in response to TBI have been previously reported; however it is unclear whether a sex difference exists in neuroinflammatory progression after TBI. We exposed male and female mice to moderate-to-severe controlled cortical impact injury and studied glial cell activation in the acute and chronic stages of TBI using immunofluorescence and in situ hybridization analysis. We found that the sex response was completely divergent up to 7 days postinjury. TBI caused a rapid and pronounced cortical microglia/macrophage activation in male mice with a prominent activated phenotype that produced both pro- (IL-1 beta and TNF alpha) and anti-inflammatory (Arg1 and TGF beta) cytokines with a single-phase, sustained peak from 1 to 7 days. In contrast, TBI caused a less robust microglia/macrophage phenotype in females with biphasic pro-inflammatory response peaks at 4 h and 7 days, and a delayed anti-inflammatory mRNA peak at 30 days. We further report that female mice were protected against acute cell loss after TBI, with male mice demonstrating enhanced astrogliosis, neuronal death, and increased lesion volume through 7 days post-TBI. Collectively, these findings indicate that TBI leads to a more aggressive neuroinflammatory profile in male compared with female mice during the acute and subacute phases postinjury. Understanding how sex affects the course of neuroinflammation following brain injury is a vital step toward developing personalized and effective treatments for TBI.

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TI Intranasal insulin treatment of an experimental model of moderate

traumatic brain injury

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE Glucose uptake; intranasal insulin; microglia; positron emission

tomography; traumatic brain injury

ID MORRIS WATER MAZE; CONTROLLED CORTICAL IMPACT; MEMORY DYSFUNCTION;

GLUCOSE-UPTAKE; COMBINED MICRODIALYSIS; MICROGLIAL ACTIVATION;

EMISSION-TOMOGRAPHY; SPINAL-CORD; (18)FDG PET; RAT-BRAIN

AB Traumatic brain injury (TBI) results in learning and memory dysfunction. Cognitive deficits result from cellular and metabolic dysfunction after injury, including decreased cerebral glucose uptake and inflammation. This study assessed the ability of intranasal insulin to increase cerebral glucose uptake after injury, reduce lesion volume, improve memory and learning function and reduce inflammation. Adult male rats received a controlled cortical impact (CCI) injury followed by intranasal insulin or saline treatment daily for 14 days. PET imaging of [F-18]-FDG uptake was performed at baseline and at 48 h and 10 days post-injury and MRI on days three and nine post injury. Motor function was tested with the beam walking test. Memory function was assessed with Morris water maze. Intranasal insulin after CCI significantly improved several outcomes compared to saline. Insulin-treated animals performed better on beam walk and demonstrated significantly improved memory. A significant increase in [F-18]-FDG uptake was observed in the hippocampus. Intranasal insulin also resulted in a significant decrease in hippocampus lesion volume and significantly less microglial immunolabeling in the hippocampus. These data show that intranasal insulin improves memory, increases cerebral glucose uptake and decreases neuroinflammation and hippocampal lesion volume, and may therefore be a viable therapy for TBI.

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TI THE ROLE OF MICROGLIA IN THE ETIOLOGY AND EVOLUTION OF CHRONIC TRAUMATIC

ENCEPHALOPATHY

SO SHOCK

LA English

DT Review

DE Chronic traumatic encephalopathy; microglia; neurofibrillary tangles;

phosphorylated tau; traumatic brain injury

ID CENTRAL-NERVOUS-SYSTEM; EXACERBATES TAU PATHOLOGY; ALZHEIMERS-DISEASE

MODEL; LONG-TERM CONSEQUENCES; BRAIN-INJURY; MOUSE MODEL; IN-VIVO;

COGNITIVE DEFICITS; PHOSPHORYLATED TAU; PRECURSOR CELLS

AB Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative disease that presents as a late sequela from traumatic brain injury (TBI). TBI is a growing and under-recognized public health concern with a high degree of morbidity and large associated global costs. While the immune response to TBI is complex, its contribution to the development of CTE remains largely unknown. In this review, we summarize the current understanding of the link between CTE and the resident innate immune system of the brain-microglia. We discuss the neuropathology underlying CTE including the creation and aggregation of phosphorylated tau protein into neurofibrillary tangles and the formation of amyloid beta deposits. We also present how microglia, the resident innate immune cells of the brain, drive the continuous low-level inflammation associated with the insidious onset of CTE. In this review, we conclude that the latency period between the index brain injury and the long-term development of CTE presents an opportunity for therapeutic intervention. Encouraging advances with microtubule stabilizers, cis p-tau antibodies, and the ability to therapeutically alter the inflammatory state of microglia have shown positive results in both animal and human trials. Looking forward, recent advancements in next-generation sequencing technology for the study of genomic, transcriptomic, and epigenetic information will provide an opportunity for significant advancement in our understanding of prorepair and pro-injury gene signatures allowing for targeted intervention in this highly morbid injury process.

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TI TLR4 signal ablation attenuated neurological deficits by regulating

microglial M1/M2 phenotype after traumatic brain injury in mice

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DT Article

ID TOLL-LIKE RECEPTOR-4; INNATE IMMUNE-SYSTEM; SPINAL-CORD-INJURY;

NERVOUS-SYSTEM; MACROPHAGE ACTIVATION; CELLS; POLARIZATION; DAMAGE;

NEUROPROTECTION; RECOGNITION

AB Traumatic brain injury (TBI) initiates inflammatory responses that result in an enduring cascade of secondary neuronal loss and behavioural impairment. Toll-like receptor 4 (TLR4), predominantly expressed by microglia, recognizes damage-associated molecular patterns (DAMPs) and regulates inflammatory processes. Interestingly, the switch of microglial M1/M2 phenotypes after TBI is highly important regarding damage and restoration of neurological function. Therefore, we investigated the role and mechanisms of the TLR4 signalling pathway in regulating microglial M1/M2 phenotypes. Using a controlled cortical impact (CCI) model, we found that TLR4 knockout (KO) mice exhibited decreased infarct volumes and improved outcomes in behavioural tests. In addition, mice lacking TLR4 had higher expression of M2 phenotype biomarkers but lower expression of M1 phenotype biomarkers. Compared with microglia derived from wild-type (WT) mice, increased expression of M2 phenotype biomarkers and decreased expression of M1 phenotype biomarkers were also noted in primary cultures of microglia from TLR4 KO mice. In TLR4 KO mice, the expression levels of downstream signalling molecules of TLR4, such as active Rac-1 and phospho-AKT, were higher, while MyD88 and phospho-NF-kappa B p65 expression levels were lower than in WT mice. Our results demonstrate that the absence of TLR4 induces microglial polarization toward the M2 phenotype and promotes microglial migration and, in turn, alleviates the development of neuroinflammation, which indicates potential neuroprotective effects in the TBI mouse model. Furthermore, up-regulation of IL-4 expression in TLR4 KO mice could contribute to anti-inflammatory functions and promote microglial polarization toward the M2 phenotype, which might be mediated by active Rac-1 expression. Taken together, TLR4 deficiency contributes to regulating microglia to switch to the M2 phenotype, which ameliorates neurological impairment after TBI.

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AF Ziebell, Jenna M.

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TI NOGO PRESENCE IS INVERSELY ASSOCIATED WITH SHIFTS IN CORTICAL MICROGLIAL

MORPHOLOGY FOLLOWING EXPERIMENTAL DIFFUSE BRAIN INJURY

SO NEUROSCIENCE

LA English

DT Article

DE microglia; Nogo; traumatic brain injury; myelin; oligodendrocytes

ID AXONAL REGENERATION; IN-VITRO; RECEPTOR; CNS; DEGENERATION; MACROPHAGES;

ACTIVATION; MICE; INFLAMMATION; INHIBITION

AB Diffuse traumatic brain injury (TBI) initiates secondary pathology, including inflammation and reduced myelination. Considering these injury-related pathologies, the many states of activated microglia as demonstrated by differing morphologies would form, migrate, and function in and through fields of growth-inhibitory myelin byproduct, specifically Nogo. Here we evaluate the relationship between inflammation and reduced myelin antigenicity in the wake of diffuse TBI and present the hypothesis that the Nogo-66 receptor antagonist peptide NEP(1-40) would reverse the injury-induced shift in distribution of microglia morphologies by limiting myelin-based inhibition. Adult male rats were subjected to midline fluid percussion sham or brain injury. At 2 h, 6 h, 1 d, 2 d, 7 d, and 21 d post injury, immunohistochemical staining was analyzed in sensory cortex (S1BF) for myelin antigens (myelin basic protein; MBP and CNPase), microglia morphology (ionized calcium-binding adapter protein; lba1), Nogo receptor and Nogo. Pronounced reduction in myelin antigenicity was evident transiently at 1 d post-injury, as evidenced by decreased MBP and CNPase staining, as well as loss of white matter organization, compared to sham and later injury time points. Concomitant with reduced myelin antigenicity, injury shifted microglia morphology from the predominantly ramified morphology observed in sham-injured cortex to hyper-ramified, activated, fully activated, or rod. Changes in microglial morphology were evident as early as 2 h post-injury, and remained at least until day 21. Additional cohorts of uninjured and brain-injured animals received vehicle or drug (NEP(1-40), i.p., 15 min and 19 h post-injury) and brains were collected at 2 h, 6 h, 1 d, 2 d, or 7 d post-injury. NEP(1-40) administration further shifted distributions of microglia away from an injury-induced activated morphology toward greater proportions of rod and macrophage-like morphologies compared to vehicle-treated. By 7 d post-injury, no differences in the distributions of microglia were noted between vehicle and NEP(1-40). This study begins to link secondary pathologies of white matter damage and inflammation after diffuse TBI. In the injured brain, secondary pathologies co-occur and likely interact, with consequences for neuronal circuit disruption leading to neurological symptoms. (C) 2017 IBRO. Published by Elsevier Ltd. All rights reserved.

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TI Exosomes secreted by stem cells from human exfoliated deciduous teeth

contribute to functional recovery after traumatic brain injury by

shifting microglia M1/M2 polarization in rats

SO STEM CELL RESEARCH & THERAPY

LA English

DT Article

DE Traumatic brain injury; Stem cells from human exfoliated deciduous

teeth; Exosomes; Microglia; Neuroinflammation

ID TISSUES; TRANSPLANTATION; REGENERATION; SCAFFOLDS; VESICLES; THERAPY

AB Background: Traumatic brain injury (TBI) is one of the major causes of mortality and disability for all ages worldwide. Mesenchymal stem cells (MSCs)-originated exosomes have provided therapeutic effects. However, as an indispensable component of MSCs, whether odontogenic stem cell-generated exosomes could benefit TBI is still unclear. Thus we aimed to explore the potential of stem cells from human exfoliated deciduous teeth-originated exosomes (SHED-Ex) for the management of TBI.

Methods: First, a transwell system was used to co-culture activated BV-2 microglia cells with SHED. The secretion levels of neuroinflammatory factors and nitrite were evaluated by enzyme-linked immunosorbent assay (ELISA) and Griess assay. Furthermore, purified SHED-Ex were co-cultured with activated BV-2. ELISA, Griess assay, flow cytometry, immunofluorescence, and qRT-PCR were performed to test the levels of inflammatory factors as well as the microglia phenotype. Finally, SHED and SHED-Ex were locally injected into TBI rat models. Basso, Beattie, and Bresnahan (BBB) scores were chosen to evaluate the motor functional recovery. Histopathology and immunofluorescence were performed to measure the lesion volume and neuroinflammation.

Results: As a result, SHED-Ex could reduce neuroinflammation by shifting microglia polarization. The administration of SHED-Ex improves rat motor functional recovery and reduces cortical lesion compared with the control group 2 weeks post-injury (P < 0.05).

Conclusions: The current study demonstrates for the first time that SHED-Ex contribute a therapeutic benefit to TBI in rats, at least in part by shifting microglia polarization to reduce neuroinflammation. The use of odontogenic stem cells, and indeed their exosomes, may be expanded for the treatment of TBI or other neurological disorders.

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TI White matter damage after traumatic brain injury: A role for damage

associated molecular patterns

SO BIOCHIMICA ET BIOPHYSICA ACTA-MOLECULAR BASIS OF DISEASE

LA English

DT Review

DE Microglia; Macrophage; White matter injury; Oligodendrocyte; HMGB1;

S100; Toll like receptor; T-cell; Lymphocyte; Leukocyte

ID GLYCATION END-PRODUCTS; GROUP BOX 1; CENTRAL-NERVOUS-SYSTEM; MYELIN

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CALCIUM-BINDING PROTEIN; NEURON-SPECIFIC ENOLASE; CLOSED-HEAD-INJURY;

REGULATORY T-CELLS

AB Traumatic brain injury (TBI) is a leading cause of mortality and long-term morbidity worldwide. Despite decades of pre-clinical investigation, therapeutic strategies focused on acute neuroprotection failed to improve TBI outcomes. This lack of translational success has necessitated a reassessment of the optimal targets for intervention, including a heightened focus on secondary injury mechanisms. Chronic immune activation correlates with progressive neurodegeneration for decades after TBI; however, significant challenges remain in functionally and mechanistically defining immune activation after TBI. In this review, we explore the burgeoning evidence implicating the acute release of damage associated molecular patterns (DAMPs), such as adenosine 5'-triphosphate (ATP), high mobility group box protein 1 (HMGB1), S100 proteins, and hyaluronic acid in the initiation of progressive neurological injury, including white matter loss after TBI. The role that pattern recognition receptors, including toll-like receptor and purinergic receptors, play in progressive neurological injury after TBI is detailed. Finally, we provide support for the notion that resident and infiltrating macrophages are critical cellular targets linking acute DAMP release with adaptive immune responses and chronic injury after TBI. The therapeutic potential of targeting DAMPs and barriers to clinical translational, in the context of TBI patient management, are discussed.

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AF Bai, Ruojing

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Lei, Ping

TI Flow Cytometric Characterization of T Cell Subsets and Microglia After

Repetitive Mild Traumatic Brain Injury in Rats

SO NEUROCHEMICAL RESEARCH

LA English

DT Article

DE Repetitive mild traumatic brain injury; T cell subsets; Microglia

ID CENTRAL-NERVOUS-SYSTEM; SPINAL-CORD-INJURY; STROKE; DAMAGE; HMGB1;

ENCEPHALOPATHY; MACROPHAGES; ISCHEMIA; S100B

AB Although, there is growing awareness in the progressive neurodegeneration of chronic traumatic encephalopathy, changes of immune reactions remain equivocal at best. Thus, in a clinically relevant rat repetitive mild traumatic brain injury (rmTBI) model, some immunologic cells (T cell subsets, microglia) in the injured brain and peripheral blood were analyzed by flow cytometry and immunofluorescence. In the injured brain, CD3(+) T cells showed a bimodal increase during 42 days post-injury (dpi). CD3(+)CD4(+) T cells firstly increased and then decreased, while CD3(+)CD8(+) T cells had reversed tendency. CD86(+)/CD11b(+) M1-like microglia increased at 42 dpi and CD206(+)/CD11b(+) M2-like microglia peaked at 7 dpi. In addition, peripheral immune suppression was implicated in the chronic phase after rmTBI. Taken together, the study provided useful information on long-term dynamic changes of some immune cells after rmTBI in rats.

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TI Acute ethanol administration results in a protective cytokine and

neuroinflammatory profile in traumatic brain injury

SO INTERNATIONAL IMMUNOPHARMACOLOGY

LA English

DT Article

DE Traumatic brain injury; Ethanol; Cytokines; Microglia

ID BLOOD-ALCOHOL CONCENTRATION; LUNG EPITHELIAL-CELLS; SYSTEMIC

INTERLEUKIN-6; UNITED-STATES; HEAD-INJURY; MICE; INTOXICATION;

EXPRESSION; MORTALITY; RATS

AB Ethanol intoxication is a common comorbidity in traumatic brain injury. To date, the effect of ethanol on TBI pathogenic cascades and resulting outcomes remains debated. A closed blunt weight-drop murine TBI model has been implemented to investigate behavioral (by sensorimotor and neurological tests), and neuro-immunological (by tissue cytokine arrays and immuno-histology) effects of ethanol intoxication on TBI. The effect of the occurrence of traumatic intracerebral hemorrhage was also studied. The results indicate that ethanol pretreatment results in a faster and better recovery after TBI with reduced infiltration of leukocytes and reduced microglia activation. These outcomes correspond to reduced parenchymal levels of GM-CSF, IL-6 and IL-3 and to the transient upregulation of IL-13 and VEGF, indicating an early shift in the cytokine profile towards reduced inflammation. A significant difference in the cytokine profile was still observed 24 h post injury in the ethanol pretreated mice, as shown by the delayed peak in IL-6 and by the suppression of GM-CSF, IFN-gamma, and IL-3. Seven days post-injury, ethanol-pretreated mice displayed a significant decrease both in CD45 + cells infiltration and in microglial activation. On the other hand, in the case of traumatic intracerebral hemorrhage, the cytokine profile was dominated by KC, CCL5, M-CSF and several interleukins and ethanol pretreatment did not produce any modification. We can thus conclude that ethanol intoxication suppresses the acute neuro-inflammatory response to TBI, an effect which is correlated with a faster and complete neurological recovery, whereas, the presence of traumatic intracerebral hemorrhage overrides the effects of ethanol.

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TI Hyperbaric oxygen alleviates the activation of NLRP-3-inflammasomes in

traumatic brain injury

SO MOLECULAR MEDICINE REPORTS

LA English

DT Article

DE traumatic brain injury hyperbaric oxygen therapy; NLRP-3 inflammasome;

microglia

ID NLRP3 INFLAMMASOME; THERAPY; NEUROPROTECTION; MECHANISMS; EXPRESSION;

NEURONS; SYSTEM; FLUID; IL-18

AB Growing evidence has demonstrated that the nucleotide-binding oligomerization domain-like receptor family pyrin domain containing 3 (NLRP-3) inflammasome-mediated inflammatory pathways have been involved in the secondary injury of traumatic brain injury (TBI). In the present study, the authors investigated the effects of hyperbaric oxygen (HBO) therapy on the NLRP-3 inflammasome pathway following TBI. Following the evaluation of motor deficits and brain edema, the therapeutic effects of HBO on interleukin (IL)-1 beta and IL-18 expression were assessed, as well as NLRP-3 inflammasome activation following TBI. HBO may improve motor score and reduce brain edema, accompanied with the reduction of IL-1 beta and IL-18 during the 7-day observation period. Furthermore, HBO suppressed mRNA and protein expression of NLRP-3-inflammasome components, especially reducing NLRP-3 expression in microglia. Thus, these results suggested that HBO alleviates the inflammatory response in experimental TBI via modulating microglial NLRP-3-inflammasome signaling.

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TI DRα1-MOG-35-55 treatment reduces lesion volumes and improves

neurological deficits after traumatic brain injury

SO METABOLIC BRAIN DISEASE

LA English

DT Article

DE Traumatic brain injury; DRa1-MOG-alpha 35-55 therapy; Neurological

deficits; CD74; Infiltrating macrophages/microglia

ID EXPERIMENTAL STROKE; CD74 EXPRESSION; ISCHEMIC-STROKE; CELL;

INFLAMMATION; CONSTRUCTS; ACTIVATION

AB Traumatic brain injury (TBI) results in severe neurological impairments without effective treatments. Inflammation appears to be an important contributor to key pathogenic events such as secondary brain injury following TBI and therefore serves as a promising target for novel therapies. We have recently demonstrated the ability of a molecular construct comprised of the human leukocyte antigen (HLA)-DR alpha 1 domain linked covalently to mouse (m)MOG-35-55 peptide (DR alpha 1-MOG-35-55 construct) to reduce CNS inflammation and tissue injury in animal models of multiple sclerosis and ischemic stroke. The aim of the current study was to determine if DR alpha 1-MOG-35-55 treatment of a fluid percussion injury (FPI) mouse model of TBI could reduce the lesion size and improve disease outcome measures. Neurodeficits, lesion size, and immune responses were determined to evaluate the therapeutic potential and mechanisms of neuroprotection induced by DR alpha 1-MOG-35-55 treatment. The results demonstrated that daily injections of DR alpha 1-MOG-35-55 given after FPI significantly reduced numbers of infiltrating CD74(+) and CD86(+) macrophages and increased numbers of CD206(+) microglia in the brain concomitant with smaller lesion sizes and improvement in neurodeficits. Conversely, DR alpha 1-MOG-35-55 treatment of TBI increased numbers of circulating CD11b(+) monocytes and their expression of CD74 but had no detectable effect on cell numbers or marker expression in the spleen. These results demonstrate that DR alpha 1-MOG-35-55 therapy can reduce CNS inflammation and significantly improve histological and clinical outcomes after TBI. Future studies will further examine the potential of DR alpha 1-MOG-35-55 for treatment of TBI.

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TI Effect of anesthetics on microglial activation and nanoparticle uptake:

Implications for drug delivery in traumatic brain injury

SO JOURNAL OF CONTROLLED RELEASE

LA English

DT Article; Proceedings Paper

CT 14th International Nanomedicine and Drug Delivery Symposium (NanoDDS)

CY SEP 16-18, 2016

CL Johns Hopkins Univ, Baltimore, MD

HO Johns Hopkins Univ

ID PENTOBARBITAL COMA; DENDRIMERS; CLEARANCE; NANOMEDICINE; THERAPY;

PATHWAY; ADULT

AB Traumatic brain injury (TBI) is a serious public health problem, often with devastating consequences for patients and their families. Affordable and timely therapies can have a substantial impact on outcomes in severe TBI. Despite the common use of sedatives and anesthetics in the acute phase of TBI management, their effect on glial cells is not well understood. We investigated the effect of a commonly used sedative, pentobarbital, on glial cells and their uptake of nanoparticles. First, we studied how pentobarbital affects BV2 mouse microglial cells in culture. The cell morphology was imaged by confocal microscopy and analyzed. Our results suggest that microglia change to a more swollen, 'activated' shape with pentobarbital (cell area increased by approximately 20%, p < 0.001). Such glial activation may have negative implications for the ability of the injured brain to clear edema. Second, we investigated how pentobarbital treatment affected nanoparticle uptake. BV-2 mouse microglial cells in the presence and absence of pentobarbital were treated with fluorescently-labeled, hydroxyl-functionalized poly(amidoamine) dendrimer nanoparticles (Dendrimer-Cy5). We demonstrated that the presence of pentobarbital increased the dendrimer nanoparticle uptake significantly (similar to 2-fold both 2 and 6 h following treatment). This semi-quantitative fluorescence assessment was broadly consistent among confocal image analysis, flow cytometry, and fluorescence quantification of cell-extracted dendrimer-Cy5. Although anesthetics appear to activate microglia, the increased uptake of dendrimer nanoparticles in their presence can be exploited to deliver drug-loaded nanoparticles directly to microglia after TBI. These drugs could restore glial and glymphatic function, enabling efficient drainage of waste and fluid from the brain and effectively improving recovery after TBI. A key future direction is to validate these findings in TBI models.

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PT J

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TI The bidirectional gut-brain-microbiota axis as a potential nexus between

traumatic brain injury, inflammation, and disease

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE Gut-brain axis; Traumatic Brain Injury; Concussion; Gut; Microbiota;

Chronic Traumatic Encephalopathy; Neurodegenerative disease;

Neuroinflammation; Microglia; Intestinal dysfunction

ID CAUSES CHRONIC NEUROINFLAMMATION; VAGUS NERVE-STIMULATION; C-REACTIVE

PROTEIN; BACTERIAL TRANSLOCATION; MICROGLIAL ACTIVATION; SYSTEMIC

INFLAMMATION; DOCOSAHEXAENOIC ACID; ALZHEIMERS-DISEASE; SICKNESS

BEHAVIOR; IMMUNE ACTIVATION

AB As head injuries and their sequelae have become an increasingly salient matter of public health, experts in the field have made great progress elucidating the biological processes occurring within the brain at the moment of injury and throughout the recovery thereafter. Given the extraordinary rate at which our collective knowledge of neurotrauma has grown, new insights may be revealed by examining the existing literature across disciplines with a new perspective. This article will aim to expand the scope of this rapidly evolving field of research beyond the confines of the central nervous system (CNS). Specifically, we will examine the extent to which the bidirectional influence of the gut-brain axis modulates the complex biological processes occurring at the time of traumatic brain injury (TBI) and over the days, months, and years that follow. In addition to local enteric signals originating in the gut, it is well accepted that gastrointestinal (GI) physiology is highly regulated by innervation from the CNS. Conversely, emerging data suggests that the function and health of the CNS is modulated by the interaction between 1) neurotransmitters, immune signaling, hormones, and neuropeptides produced in the gut, 2) the composition of the gut microbiota, and 3) integrity of the intestinal wall serving as a barrier to the external environment. Specific to TBI, existing pre-clinical data indicates that head injuries can cause structural and functional damage to the GI tract, but research directly investigating the neuronal consequences of this intestinal damage is lacking. Despite this void, the proposed mechanisms emanating from a damaged gut are closely implicated in the inflammatory processes known to promote neuropathology in the brain following TBI, which suggests the gut-brain axis may be a therapeutic target to reduce the risk of Chronic Traumatic Encephalopathy and other neurodegenerative diseases following TBI. To better appreciate how various peripheral influences are implicated in the health of the CNS following TBI, this paper will also review the secondary biological injury mechanisms and the dynamic pathophysiological response to neurotrauma. Together, this review article will attempt to connect the dots to reveal novel insights into the bidirectional influence of the gut-brain axis and propose a conceptual model relevant to the recovery from TBI and subsequent risk for future neurological conditions. (C) 2017 Elsevier Inc. All rights reserved.

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TI Interleukin-33 Promotes Recruitment of Microglia/Macrophages in Response

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DE alarmin; glia; microglia; traumatic brain injury; neuroinflammation

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AB Traumatic brain injury (TBI) is a devastating condition, often leading to life-long consequences for patients. Even though modern neurointensive care has improved functional and cognitive outcomes, efficient pharmacological therapies are still lacking. Targeting peripherally derived, or resident inflammatory, cells that are rapid responders to brain injury is promising, but complex, given that the contribution of inflammation to exacerbation versus improved recovery varies with time post-injury. The injury-induced inflammatory response is triggered by release of alarmins, and in the present study we asked whether interleukin-33 (IL-33), an injury-associated nuclear alarmin, is involved in TBI. Here, we used samples from human TBI microdialysate, tissue sections from human TBI, and mouse models of central nervous system injury and found that expression of IL-33 in the brain was elevated from nondetectable levels, reaching a maximum after 72 h in both human samples and mouse models. Astrocytes and oligodendrocytes were the main producers of IL-33. Post-TBI, brains of mice deficient in the IL-33 receptor, ST2, contained fewer microglia/macrophages in the injured region than wild-type mice and had an altered cytokine/chemokine profile in response to injury. These observations indicate that IL-33 plays a role in neuroinflammation with microglia/macrophages being cellular targets for this interleukin post-TBI.

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TI Deletion or inhibition of soluble epoxide hydrolase protects against

brain damage and reduces microglia-mediated neuroinflammation in

traumatic brain injury

SO ONCOTARGET

LA English

DT Article

DE soluble epoxide hydrolase; traumatic brain injury; microglia;

inflammation; AUDA

ID NITRIC-OXIDE SYNTHASE; N-TERMINAL DOMAIN; P38 MAPK PATHWAYS;

SUBARACHNOID HEMORRHAGE; INFLAMMATORY RESPONSES; THERAPEUTIC TARGET;

CEREBRAL-ISCHEMIA; CORTICAL IMPACT; PPAR-GAMMA; C-FOS

AB Traumatic brain injury (TBI) induces a series of inflammatory processes that contribute to neuronal damage. The present study investigated the involvement of soluble epoxide hydrolase (sEH) in neuroinflammation and brain damage in mouse TBI and in microglial cultures. The effects of genetic deletion of sEH and treatment with an sEH inhibitor, 12-(3-adamantan-1-yl-ureido)-dodecanoic acid (AUDA), on brain damage and inflammatory responses were evaluated in mice subjected to controlled cortical impact. The anti-inflammatory mechanism of sEH inhibition/deletion was investigated in vitro. TBI-induced an increase in sEH protein level in the injured cortex from 1 h to 4 days and sEH was expressed in microglia. Genetic deletion of sEH significantly attenuated functional deficits and brain damage up to 28 days post-TBI. Deletion of sEH also reduced neuronal death, apoptosis, brain edema, and BBB permeability at 1 and 4 day(s). These changes were associated with markedly reduced microglial/macrophage activation, neutrophil infiltration, matrix metalloproteinase-9 activity, inflammatory mediator expression at 1 and 4 day(s), and epoxyeicosatrienoic acid (EET) degradation at 1 and 4 day(s). Administration of AUDA attenuated brain edema, apoptosis, inflammatory mediator upregulation and EET degradation at 4 days. In primary microglial cultures, AUDA attenuated both LPS-or IFN-Upsilon-stimulated nitric oxide (NO) production and reduced LPS-or IFN-Upsilon-induced p38 MAPK and NF-kappa B signaling. Deletion of sEH also reduced IFN-Upsilon-induced NO production. Moreover, AUDA attenuated N2A neuronal death induced by BV2 microglial-conditioned media. Our results suggest that inhibition of sEH may be a potential therapy for TBI by modulating the cytotoxic functions of microglia.

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TI Major depressive disorder and anxiety disorders from the glial

perspective: Etiological mechanisms, intervention and monitoring

SO NEUROSCIENCE AND BIOBEHAVIORAL REVIEWS

LA English

DT Review

DE Glia; Major depressive disorder; Anxiety disorders; Early trauma; Brain

imaging; Astrocyte; Oligodendrocyte; Microglia; Radial glia; Psychiatric

disorders

ID DORSOLATERAL PREFRONTAL CORTEX; FIBRILLARY ACIDIC PROTEIN; SEIZURES

INDUCE PROLIFERATION; WHITE-MATTER ABNORMALITIES; ANTERIOR CINGULATE

CORTEX; MEDICATION-FREE PATIENTS; RECEPTOR MESSENGER-RNA;

CENTRAL-NERVOUS-SYSTEM; LATE-LIFE DEPRESSION; DSM-IV DISORDERS

AB Despite intense ongoing research efforts, the etiology of psychiatric disorders remains incompletely understood. Among biological factors playing a role in Major Depressive Disorder (MDD) and Anxiety Disorders (ANX), emerging evidence points to the relevance of different types of glia cells and efficient neuron-glia interactions. Here, we review recent findings highlighting the involvement of central nervous system (CNS) glia in MDD and ANX etiology and treatment response. Additionally, several relatively underexplored topics will be discussed: (1) glial response to non-pharmacological therapies, (2) impact of early life adversity on glia, (3) influence of lifestyle factors on glia in the context of MDD and ANX, and (4) monitoring glial functions in patients. It can be concluded that despite the sequence of events is still unclear, alterations in glial cell types are common and somewhat overlapping in ANX, MDD and corresponding animal models. Furthermore, glia are responsive to a variety of treatment and lifestyle options. Looking forward, new research developments can lead to novel types of therapeutic or symptom-relieving approaches targeting glia.

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TI Administration of Protocatechuic Acid Reduces Traumatic Brain

Injury-Induced Neuronal Death

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE traumatic brain injury; protocatechuic acid; neuron death; oxidative

injury; microglial activation

ID OXIDATIVE STRESS; SUPEROXIDE-PRODUCTION; BARRIER DISRUPTION;

LIPID-PEROXIDATION; IN-VITRO; DAMAGE; GLUTATHIONE; INHIBITION;

IMPAIRMENT; RADICALS

AB Protocatechuic acid (PCA) was first purified from green tea and has shown numerous biological activities, including anti-apoptotic, anti-inflammatory, and anti-atherosclerotic effects. The effect of PCA on traumatic brain injury (TBI)-induced neuronal death has not previously been evaluated. TBI is defined as damage to the brain resulting from external mechanical force, such as rapid acceleration or deceleration, impact, blast waves, or penetration by a projectile. TBI causes neuronal death in the hippocampus and cerebral cortex. The present study aimed to evaluate the therapeutic potential of PCA on TBI-induced neuronal death. Here, TBI was induced by a controlled cortical impact model using rats. PCA (30 mg/kg) was injected into the intraperitoneal (ip) space immediately after TBI. Neuronal death was evaluated with Fluoro Jade-B (FJB) staining at 24 h after TBI. Oxidative injury was detected by 4-hydroxy-2-nonenal (4HNE), glutathione (GSH) concentration was analyzed by glutathione adduct with N-ethylmaleimide (GS-NEM) staining at 24 h after TBI, and microglial activation in the hippocampus was detected by CD11b immunohistochemistry at one week after TBI. We found that the proportion of degenerating neurons, oxidative injury, GSH depletion, and microglia activation in the hippocampus and cortex were all reduced by PCA treatment following TBI. Therefore, our study suggests that PCA may have therapeutic potential in preventing TBI-induced neuronal death.

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TI Inhibition of P2X7 receptors improves outcomes after traumatic brain

injury in rats

SO PURINERGIC SIGNALLING

LA English

DT Article

DE Traumatic brain injury; P2X7R; Microglial cells; Microvesicles;

Neuroinflammation

ID MICROGLIAL ACTIVATION; EXTRACELLULAR ATP; WATER-MAZE; ASTROGLIOSIS;

INVOLVEMENT; RELEASE; EPIDEMIOLOGY; ANTAGONIST; ASTROCYTES; EXPRESSION

AB Traumatic brain injury (TBI) is the leading cause of death and disability for people under the age of 45 years worldwide. Neuropathology after TBI is the result of both the immediate impact injury and secondary injury mechanisms. Secondary injury is the result of cascade events, including glutamate excitotoxicity, calcium overloading, free radical generation, and neuroinflammation, ultimately leading to brain cell death. In this study, the P2X7 receptor (P2X7R) was detected predominately in microglia of the cerebral cortex and was up-regulated on microglial cells after TBI. The microglia transformed into amoeba-like and discharged many microvesicle (MV)-like particles in the injured and adjacent regions. A P2X7R antagonist (A804598) and an immune inhibitor (FTY720) reduced significantly the number of MV-like particles in the injured/adjacent regions and in cerebrospinal fluid, reduced the number of neurons undergoing apoptotic cell death, and increased the survival of neurons in the cerebral cortex injured and adjacent regions. Blockade of the P2X7R and FTY720 reduced interleukin-1 beta expression, P38 phosphorylation, and glial activation in the cerebral cortex and improved neurobehavioral outcomes after TBI. These data indicate that MV-like particles discharged by microglia after TBI may be involved in the development of local inflammation and secondary nerve cell injury.

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NR 57

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Z9 36

U1 2

U2 17

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J9 PURINERG SIGNAL

JI Purinergic Signal.

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TI Remote ischemic conditioning preserves cognition and motor coordination

in a mouse model of traumatic brain injury

SO JOURNAL OF TRAUMA AND ACUTE CARE SURGERY

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DT Article; Proceedings Paper

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DE Traumatic brain injury; biomarkers; blood-brain barrier; inflammation;

microglia

ID NEURON-SPECIFIC ENOLASE; NECROSIS-FACTOR-ALPHA; INFLAMMATORY RESPONSE;

STROKE; S100B; MICE; IMPAIRMENT; EXPRESSION; INCREASES; SURGERY

AB INTRODUCTION Management of traumatic brain injury (TBI) is focused on minimizing or preventing secondary brain injury. Remote ischemic conditioning (RIC) is an established treatment modality that has been shown to improve patient outcomes in different clinical settings by influencing inflammatory insults. In a clinical trial, RIC showed amelioration of SB100 and neuron-specific enolase. The aim of our study was to further elucidate the mechanisms and outcome when applying RIC in a mouse model of traumatic brain injury.

METHODS We subjected 100 male C57BL mice to a closed-skull cortical-controlled impact injury. Two hours after the TBI, the animals were allocated to either the RIC group (n = 50) or the sham group (n = 50). By clamping the exposed femoral artery, we induced RIC by six 4-minute cycles of ischemia and reperfusion. Circulating levels of S100-B, neuron-specific enolase, and glial fibrillary acidic protein were measured at multiple time points. Animals were additionally observed daily for cognition and motor coordination via novel object recognition and rotarod. Brain sections were stained and evaluated for neuronal injury at post-TBI Day 5.

RESULTS The RIC animals had a significantly higher recognition index than did sham at 24, 48, and 72 hours after intervention. Rotarod latency was higher in the RIC animals compared to the sham animals at all-time points, and statistically significant at 120 hours after intervention. The RIC group demonstrated preserved cognitive function and motor coordination compared to the sham. On hematoxylin and eosin and immunohistochemical staining of brain sections, there was less area of neuronal degeneration and astrocytosis, respectively, in the RIC group compared to the sham group. There was no significant difference in systemic neuronal markers between the RIC and sham animals.

CONCLUSION Remote ischemic conditioning 2 hours after injury preserved cognitive functions and motor coordination in a mouse model of TBI. Remote ischemic conditioning can preserve viability of neurons and astrocytes after TBI and has potential as a clinically noninvasive and relatively easy method to improve outcome after TBI.

LEVEL OF EVIDENCE Therapeutic studies, randomized controlled trial, level I.

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TI Regulatory role of NADPH oxidase 2 in the polarization dynamics and

neurotoxicity of microglia/macrophages after traumatic brain injury

SO FREE RADICAL BIOLOGY AND MEDICINE

LA English

DT Article

DE NADPH oxidase; NOX2; Microglia; Traumatic brain injury; Inflammation;

Oxidative stress; NF-kappa B

ID NF-KAPPA-B; NEURAL STEM-CELLS; NLRP3 INFLAMMASOME; GANGLIOSIDE GD3;

ACTIVATION; EXPRESSION; NEURODEGENERATION; NEURONS; CLONING; DAMAGE

AB Traumatic brain injury (TBI) is a leading cause of death and disability. Secondary injuries that develop after the initial trauma contribute to long-lasting neurophysiological deficits. Polarization of microglia/macrophages toward a pro-inflammatory (M1) phenotype may increase the progression of secondary injury following TBI; however, the regulatory and functional mechanisms underlying these changes remain poorly defined. In the present study, we showed elevated expression of NADPH oxidase 2 (NOX2) and activation of nuclear factorkappa B (NF-kappa B) predominantly in microglia/macrophages at 4-and 7-days after controlled cortical impact in mice. Delayed inhibition of NOX2, beginning one day after TBI, reduced reactive oxygen species production of myeloid cells and protected neurons from oxidative damage. Moreover, delayed NOX inhibition or global genetic NOX2 knockout suppressed the M1 "pro-inflammatory" profile of microglia/macrophages and simultaneously increased the M2 "anti-inflammatory" profile in the injured brain. These changes were associated with marked down-regulation of the classical NF-kappa B pathway in microglia/macrophages and reduced production of pro-inflammatory cytokines, tumor necrosis factor-alpha and interleukin-1 beta, after TBI. Finally, we demonstrated that wildtype microglia/macrophages isolated from the ipsilateral cortex at 7 days post-TBI were neurotoxic to co-cultured primary neurons, whereas this neurotoxicity was largely attenuated in microglia/macrophages from NOX2-KO mice. Taken together, our study shows a direct link between NOX2 and the NF-kappa B pathway in microglia/ macrophages after TBI, and it provides a novel mechanism by which NOX2 activation leads to the enhanced inflammatory response and neuronal damage after brain injury. Our data also supports the therapeutic potential of targeting NOX2, which may provide efficacy with an extended therapeutic window after TBI.

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TI A Three-Day Consecutive Fingolimod Administration Improves Neurological

Functions and Modulates Multiple Immune Responses of CCI Mice

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article

DE Traumatic brain injury (TBI); Fingolimod; Immuno-inflammatory response;

Regulatory T cell (Treg); Macrophage/microglia subtype; Cytokine array

ID TRAUMATIC BRAIN-INJURY; MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS;

CENTRAL-NERVOUS-SYSTEM; REGULATORY T-CELLS; MACROPHAGE SUBSETS;

WHITE-MATTER; FTY720; RECRUITMENT; NEUROTOXICITY; DEGENERATION

AB Excessive inflammation after traumatic brain injury (TBI) is a major cause of secondary TBI. Though several inflammatory biomarkers have been postulated as the risk factors of TBI, there has not been any comprehensive description of them. Fingolimod, a new kind of immunomodulatory agent which can diminish various kinds of inflammatory responses, has shown additional therapeutic effects in the treatment of intracranial cerebral hematoma (ICH), ischemia, spinal cord injury (SCI), and many other CNS disorders. However, its therapeutic application has not been confirmed in TBI. Thus, we hypothesized that a 3-day consecutive fingolimod administration could broadly modulate the inflammatory reactions and improve the outcomes of TBI. The TBI models of C57/BL6 mice were established with the controlled cortical impact injury (CCI) system. A 3-day consecutive fingolimod therapy (given at 1, 24, and 48 h post injury) was performed at a dose of 1 mg/kg. The flow cytometry, immunoflourence, cytokine array, and ELISA were all applied to evaluate the immune cells and inflammatory markers in the injured brains. Immunohistochemical staining with anti-APP antibody was performed to assess the axonal damage. The neurological functions of these TBI models were assessed by mNSS/Rota-rod and Morris water maze (MWM). The brain water content and integrity of the blood-brain barrier (BBB) were also observed. On the 3rd day after TBI, the accumulation of inflammatory cytokines and chemokines reached the peak and administration of fingolimod reduced as many as 20 kinds of cytokines and chemokines. Fingolimod decreased infiltrated T lymphocytes and NK cells but increased the percentage of regulatory T (Treg) cells, and the concentration of IL-10 on the 3rd day after TBI. Fingolimod also notably attenuated the general activated microglia but augmented the M2/M1 ratio accompanied by decreased axonal damage. The neurological functions were improved after the fingolimod treatment accompanied with alleviation of the brain edema and BBB damage. This study suggests that the 3-day consecutive fingolimod administration extensively modulates multiple immuno-inflammatory responses and improves the neurological deficits after TBI, and therefore, it may be a new approach to the treatment of secondary TBI.

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TI Post-Injury Administration of Galantamine Reduces Traumatic Brain Injury

Pathology and Improves Outcome

SO JOURNAL OF NEUROTRAUMA

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DT Article; Early Access

DE blood-brain barrier permeability; cholinergic neurotransmission;

GABAergic neurodegeneration; memory impairments; microglial polarization

ID CHOLINERGIC RECEPTOR-BINDING; ADULT HIPPOCAMPAL NEUROGENESIS; CORTICAL

IMPACT INJURY; DENTATE GYRUS; ACETYLCHOLINESTERASE INHIBITORS; COGNITIVE

IMPAIRMENT; ALLOSTERIC MODULATOR; PATTERN SEPARATION; ALPHA-7 NACHRS;

RAT-BRAIN

AB Acetylcholine is an excitatory neurotransmitter in the central nervous system that plays a key role in cognitive function, including learning and memory. Previous studies have shown that experimental traumatic brain injury (TBI) reduces cholinergic neurotransmission, decreases evoked release of acetylcholine, and alters cholinergic receptor levels. Galantamine (U.S. Food and Drug Administration approved for the treatment of vascular dementia and Alzheimer's disease) has been shown to inhibit acetylcholinesterase activity and allosterically potentiate nicotinic receptor signaling. We investigated whether acute administration of galantamine can reduce TBI pathology and improve cognitive function tested days after the termination of the drug treatment. Post-injury administration of galantamine was found to decrease TBI-triggered blood-brain barrier (BBB) permeability (tested 24h post-injury), attenuate the loss of both GABAergic and newborn neurons in the ipsilateral hippocampus, and improve hippocampal function (tested 10 days after termination of the drug treatment). Specifically, significant improvements in the Morris water maze, novel object recognition, and context-specific fear memory tasks were observed in injured animals treated with galantamine. Although messenger RNAs for both M1 (Nos2, TLR4, and IL-12 beta ) and M2 (Arg1, CCL17, and Mcr1) microglial phenotypes were elevated post-TBI, galantamine treatment did not alter microglial polarization tested 24h and 6 days post-injury. Taken together, these findings support the further investigation of galantamine as a treatment for TBI.

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TI Minocycline Attenuates High Mobility Group Box 1 Translocation,

Microglial Activation, and Thalamic Neurodegeneration after Traumatic

Brain Injury in Post-Natal Day 17 Rats

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE HMGB1; microglia; minocycline; neuroinflammation; traumatic brain injury

ID CEREBROSPINAL-FLUID; CYTOCHROME-C; NEUROPROTECTION; CHILDREN; STATES;

MICE; DYSAUTONOMIA; IMPAIRMENT; APOPTOSIS; HMGB1

AB In response to cell injury, the danger signal high mobility group box-1 (HMGB) is released, activating macrophages by binding pattern recognition receptors. We investigated the role of the anti-inflammatory drug minocycline in attenuating HMGB1 translocation, microglial activation, and neuronal injury in a rat model of pediatric traumatic brain injury (TBI). Post-natal day 17 Sprague-Dawley rats underwent moderate-severe controlled cortical impact (CCI). Animals were randomized to treatment with minocycline (90mg/kg, intraperitoneally) or vehicle (saline) at 10min and 20h after injury. Shams received anesthesia and craniotomy. We analyzed HMGB1 translocation (protein fractionation and Western blotting), microglial activation (Iba-1 immunohistochemistry), neuronal death (Fluoro-Jade-B [FJB] immunofluorescence), and neuronal cell counts (unbiased stereology). Behavioral assessments included motor and Morris-water maze testing. Nuclear to cytosolic translocation of HMGB1 in the injured brain was attenuated in minocycline versus vehicle-treated rats at 24h (p<0.001). Treatment with minocycline reduced microglial activation in the ipsilateral cortex, hippocampus, and thalamus (p<0.05 vs. vehicle, all regions); attenuated neurodegeneration (FJB-positive neurons) at seven days (p<0.05 vs. vehicle); and increased thalamic neuronal survival at 14 days (naive 22773 +/- 1012 cells/mm(3), CCI + vehicle 11753 +/- 464, CCI + minocycline 17047 +/- 524; p<0.001). Minocycline-treated rats demonstrated delayed motor recovery early after injury but had no injury effect on Morris-water maze whereas vehicle-treated rats performed worse than sham on the final two days of testing (both p<0.05 vs. vehicle). Minocycline globally attenuated HMGB1 translocation and microglial activation in injured brain in a pediatric TBI model and afforded selective thalamic neuroprotection. The HMGB1 translocation and thalamic injury may represent novel mechanistic and regional therapeutic targets in pediatric TBI.

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TI Increased miR-124-3p in microglial exosomes following traumatic brain

injury inhibits neuronal inflammation and contributes to neurite

outgrowth via their transfer into neurons

SO FASEB JOURNAL

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DE extracellular vesicles; glia cells; polarization; PDE4B; mTOR signaling

ID MESENCHYMAL STROMAL CELLS; EXTRACELLULAR VESICLES; PHOSPHODIESTERASE 4B;

IN-VITRO; PATHWAY; POLARIZATION; APOPTOSIS; DISEASE; AKT;

NEUROINFLAMMATION

AB Neuronal inflammation is the characteristic pathologic change of acute neurologic impairment and chronic traumatic encephalopathy after traumatic brain injury (TBI). Inhibiting the excessive inflammatory response is essential for improving the neurologic outcome. To clarify the regulatory mechanism of microglial exosomes on neuronal inflammation in TBI, we focused on studying the impact of microglial exosomal miRNAs on injured neurons in this research. We used a repetitive (r) TBI mousemodel and harvested the injured brain extracts from the acute to the chronic phase of TBI to treat cultured BV2 microglia in vitro. The microglial exosomes were collected for miRNA microarray analysis, which showed that the expression level of miR-124-3p increased most apparently in the miRNAs. We found that miR-124-3p promoted the anti-inflamed M2 polarization in microglia, and microglial exosomal miR-124-3p inhibited neuronal inflammation in scratch-injured neurons. Further, the mammalian target of rapamycin (mTOR) signaling was implicated as being involved in the regulation of miR-124-3p by Gene Ontology and Kyoto Encyclopedia of Genes and Genomes pathway analyses. Using the mTOR activator MHY1485 we confirmed that the inhibitory effect of exosomalmiR-124-3p on neuronal inflammation was exerted by suppressing the activity of mTOR signaling. PDE4B was predicted to be the target gene of miR-124-3p by pathway analysis. We proved that it was directly targeted by miR-124-3p with a luciferase reporter assay. Using a PDE4B overexpressed lentivirus transfection system, we suggested that miR-124-3p suppressed the activity of m TOR signaling mainly through inhibiting the expression of PDE4B. In addition, exosomal miR-124-3p promoted neurite outgrowth after scratch injury, characterized by an increase on the number of neurite branches and total neurite length, and a decreased expression on RhoA and neurodegenerative proteins [A beta-peptide and p-Tau]. It also improved the neurologic outcome and inhibited neuro-inflammation in mice with rTBI. Taken together, increased miR-124-3p in microglial exosomes after TBI can inhibit neuronal inflammation and contribute to neurite outgrowth via their transfer into neurons. miR-124-3p exerted these effects by targeting PDE4B, thus inhibiting the activity of mTOR signaling. Therefore, miR-124-3p could be a promising therapeutic target for interventions of neuronal inflammation after TBI. miRNAs manipulated microglial exosomes may provide a novel therapy for TBI and other neurologic diseases.

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TI Exacerbation of Acute Traumatic Brain Injury by Circulating

Extracellular Vesicles

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DE blood-brain barrier; extracellular vesicles; inflammation; microglia;

traumatic brain injury

ID CENTRAL-NERVOUS-SYSTEM; ACUTE-PHASE RESPONSE; LEUKOCYTE MOBILIZATION;

INFLAMMATORY RESPONSE; FLOW-CYTOMETRY; SPINAL-CORD; MICROPARTICLES;

NEUROINFLAMMATION; MICROVESICLES; MODEL

AB Inflammatory lesions in the brain activate a systemic acute-phase response (APR), which is dependent on the release of extracellular vesicles (EVs) into the circulation. The resulting APR is responsible for regulating leukocyte mobilization and subsequent recruitment to the brain. Factors that either exacerbate or inhibit the APR will also exacerbate or inhibit central nervous system (CNS) inflammation as a consequence and have the potential to influence ongoing secondary damage. Here, we were interested to discover how the circulating EV population changes after traumatic brain injury (TBI) and how manipulation of the circulating EV pool impacts on the outcome of TBI. We found the number of circulating EVs increased rapidly post-TBI, and this was accompanied by an increase in CNS and hepatic leukocyte recruitment. In an adoptive transfer study, we then evaluated the outcomes of TBI after administering EVs derived from either in vitro macrophage or endothelial cell lines stimulated with lipopolysaccharide (LPS), or from murine plasma from an LPS challenge using the air-pouch model. By manipulating the circulating EV population, we were able to demonstrate that each population of transferred EVs increased the APR. However, the characteristics of the response were dependent on the nature of the EVs; specifically, it was significantly increased when animals were challenged with macrophage-derived EVs, suggesting that the cellular origins of EVs may determine their function. Selectively targeting EVs from macrophage/monocyte populations is likely to be of value in reducing the impact of the systemic inflammatory response on the outcome of traumatic CNS injury.

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TI Laquinimod attenuates inflammation by modulating macrophage functions in

traumatic brain injury mouse model

SO JOURNAL OF NEUROINFLAMMATION

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DT Article

DE Laquinimod; Microglia; Peripherally derived monocytes; Traumatic brain

injury

ID EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS; PLACEBO-CONTROLLED TRIAL;

CENTRAL-NERVOUS-SYSTEM; MULTIPLE-SCLEROSIS; ORAL LAQUINIMOD; MICE;

DISEASE; CELLS; MILD; MICROGLIA

AB Background: Traumatic brain injury (TBI) is a critical public health and socio-economic problem worldwide. A growing body of evidence supports the involvement of inflammatory events in TBI. It has been reported that resident microglia and infiltrating monocytes promote an inflammatory reaction that leads to neuronal death and eventually behavioral and cognitive impairment. Currently, there is no effective treatment for TBI and the development of new therapeutic strategies is a scientific goal of highest priority. Laquinimod, an orally administered neuroimmunomodulator initially developed for the treatment of multiple sclerosis, might be a promising neuroprotective therapy for TBI. Herein, we aim to investigate the hypothesis that laquinimod will reduce the central nervous system (CNS) damage caused by TBI.

Methods: To test our hypothesis, Ccr2(rfp/+) Cx3cr1(gfp/+) mice were submitted to a moderate TBI induced by fluid percussion. Sham controls were submitted only to craniotomy. Mice were treated daily by oral gavage with laquinimod (25 mg/kg) 7 days before and 3 days after TBI. The brains of mice treated or not treated with laquinimod were collected at 3 and 120 days post injury, and brain morphological changes, axonal injury, and neurogenesis were evaluated by microscopy analysis. We also isolated microglia from infiltrating monocytes, and the expression of immune gene mRNAs were analyzed by employing a quantitative NanoString nCounter technique.

Results: Laquinimod prevented ventricle enlargement caused by TBI in the long term. Immunohistochemical analyses revealed decreased axonal damage and restored neurogenesis in the laquinimod-treated TBI group at early stage (3 days post injury). Notably, laquinimod inhibited the monocytes infiltration to the brain. Hierarchial clustering demonstrated that the microglial gene expression from the TBI group treated with laquinimod resembles the sham group more than the TBI-water control group.

Conclusions: Administration of laquinimod reduced lesion volume and axonal damage and restored neurogenesis after TBI. Laquinimod might be a potential therapy strategy to improve TBI long-term prognosis.

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TI Minocycline reduces chronic microglial activation after brain trauma but

increases neurodegeneration

SO BRAIN

LA English

DT Article

DE traumatic brain injury; microglia; minocycline; neurodegeneration;

positron emission tomography

ID NEUROFILAMENT LIGHT PROTEIN; RHESUS-MONKEY; SPINAL-CORD; INJURY;

BINDING; SINGLE; PET; INFLAMMATION; BIOMARKER; EFFICACY

AB Survivors of a traumatic brain injury can deteriorate years later, developing brain atrophy and dementia. Traumatic brain injury triggers chronic microglial activation, but it is unclear whether this is harmful or beneficial. A successful chronic-phase treatment for traumatic brain injury might be to target microglia. In experimental models, the antibiotic minocycline inhibits microglial activation. We investigated the effect of minocycline on microglial activation and neurodegeneration using PET, MRI, and measurement of the axonal protein neurofilament light in plasma. Microglial activation was assessed using C-11-PBR28 PET. The relationships of microglial activation to measures of brain injury, and the effects of minocycline on disease progression, were assessed using structural and diffusion MRI, plasma neurofilament light, and cognitive assessment. Fifteen patients at least 6 months after a moderate-to-severe traumatic brain injury received either minocycline 100 mg orally twice daily or no drug, for 12 weeks. At baseline, C-11-PBR28 binding in patients was increased compared to controls in cerebral white matter and thalamus, and plasma neurofilament light levels were elevated. MRI measures of white matter damage were highest in areas of greater C-11-PBR28 binding. Minocycline reduced 11 C-PBR28 binding (mean Delta white matter binding = -23.30%, 95% confidence interval -40.9 to -5.64%, P = 0.018), but increased plasma neurofilament light levels. Faster rates of brain atrophy were found in patients with higher baseline neurofilament light levels. In this experimental medicine study, minocycline after traumatic brain injury reduced chronic microglial activation while increasing a marker of neurodegeneration. These findings suggest that microglial activation has a reparative effect in the chronic phase of traumatic brain injury.

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TI Differential Response of Neural Cells to Trauma-Induced Swelling In

Vitro

SO NEUROCHEMICAL RESEARCH

LA English

DT Article

DE Cell swelling; Astrocytes; Microglia; Neurons; Cytokines; Glutamate;

Traumatic brain injury

ID FLUID-PERCUSSION INJURY; FREE-RADICAL PRODUCTION; BRAIN-INJURY;

GLUTAMATE TRANSPORTER; CULTURED ASTROCYTES; ENDOTHELIAL-CELLS; EDEMA;

RAT; MICROGLIA; NEURONS

AB Brain edema and the associated increase in intracranial pressure are major consequences of traumatic brain injury (TBI) that accounts for most early deaths after TBI. We recently showed that acute severe trauma to cultured astrocytes results in cell swelling. We further examined whether trauma induces cell swelling in neurons and microglia. We found that severe trauma also caused cell swelling in cultured neurons, whereas no swelling was observed in microglia. While severe trauma caused cell swelling in both astrocytes and neurons, mild trauma to astrocytes, neurons, and microglia failed to cell swelling. Since extracellular levels of glutamate are increased in brain post-TBI and microglia are known to release cytokine, and direct exposure of astrocytes to these molecules are known to stimulate cell swelling, we examined whether glutamate or cytokines have any additive effect on trauma-induced cell swelling. Exposure of cultured astrocytes to trauma caused cell swelling, and such swelling was potentiated by the exposure of traumatized astrocytes to glutamate and cytokines. Conditioned medium (CM) from traumatized astrocytes had no effect on neuronal swelling post-trauma, while CM from traumatized neurons and microglia potentiated the effect of trauma on astrocyte swelling. Further, trauma significantly increased the Na-K-Cl co-transporter (NKCC) activity in neurons, and that inhibition of NKCC activity diminished the trauma-induced neuronal swelling. Our results indicate that a differential sensitivity to trauma-induced cell swelling exists in neural cells and that neurons and microglia are likely to be involved in the potentiation of the astrocyte swelling post-trauma.

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TI Hyperthermia and Mild Traumatic Brain Injury: Effects on Inflammation

and the Cerebral Vasculature

SO JOURNAL OF NEUROTRAUMA

LA English

DT Review; Early Access

DE BBB perturbations; concussion; cytokines; hyperthermia; inflammation;

macrophages; microglia; traumatic brain injury

ID CLOSED-HEAD INJURY; POST-CONCUSSION SYNDROME; WHITE-MATTER; BLOOD-FLOW;

THERAPEUTIC HYPOTHERMIA; BARRIER PERMEABILITY; TAU PATHOLOGY; RODENT

MODEL; MICROGLIA; NEUROINFLAMMATION

AB Mild traumatic brain injury (mTBI) or concussion represents the majority of brain trauma in the United States. The pathophysiology of mTBI is complex and may include both focal and diffuse injury patterns. In addition to altered circuit dysfunction and traumatic axonal injury (TAI), chronic neuroinflammation has also been implicated in the pathophysiology of mTBI. Recently, our laboratory has reported the detrimental effects of mild hyperthermic mTBI in terms of worsening histopathological and behavioral outcomes. To clarify the role of temperature-sensitive neuroinflammatory processes on these consequences, we evaluated the effects of elevated brain temperature (39 degrees C) on altered microglia/macrophage phenotype patterns after mTBI, changes in leukocyte recruitment, and TAI. Sprague-Dawley male rats underwent mild parasagittal fluid-percussion injury under normothermic (37 degrees C) or hyperthermic (39 degrees C) conditions. Cortical and hippocampal regions were analyzed using several cellular and molecular outcome measures. At 24 h, the ratio of iNOS-positive (M1 type phenotype) to arginase-positive (M2 type phenotype) cells after hyperthermic mTBI showed an increase compared with normothermia by flow cytometry. Inflammatory response gene arrays also demonstrated a significant increase in several classes of pro-inflammatory genes with hyperthermia treatment over normothermia. The injury-induced expression of chemokine ligand 2 (Ccl2) and alpha-2-macroglobulin were also increased with hyperthermic mTBI. With western blot analysis, an increase in CD18 and intercellular cell adhesion molecule-1 (ICAM-1) with hyperthermia and a significant increase in Iba1 reactive microglia are reported in the cerebral cortex. Together, these results demonstrate significant differences in the cellular and molecular consequences of raised brain temperature at the time of mTBI. The observed polarization toward a M1-phenotype with mild hyperthermia would be expected to augment chronic inflammatory cascades, sustained functional deficits, and increased vulnerability to secondary insults. Mild elevations in brain temperature may contribute to the more severe and longer lasting consequences of mTBI or concussion reported in some patients.

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TI Trovafloxacin attenuates neuroinflammation and improves outcome after

traumatic brain injury in mice

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Brain injury; Neuroinflammation; Microglia; Pannexin; Hemichannel

ID CONTROLLED CORTICAL IMPACT; BRILLIANT BLUE FCF; PANNEXIN 1; ATP RELEASE;

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INHIBITION

AB Background: Trovafloxacin is a broad-spectrum antibiotic, recently identified as an inhibitor of pannexin-1 (Panx1) channels. Panx1 channels are important conduits for the adenosine triphosphate (ATP) release from live and dying cells that enhances the inflammatory response of immune cells. Elevated extracellular levels ATP released upon injury activate purinergic pathways in inflammatory cells that promote migration, proliferation, phagocytosis, and apoptotic signals. Here, we tested whether trovafloxacin administration attenuates the neuroinflammatory response and improves outcomes after brain trauma.

Methods: The murine controlled cortical impact (CCI) model was used to determine whether in vivo delivery of trovafloxacin has anti-inflammatory and neuroprotective actions after brain trauma. Locomotor deficit was assessed using the rotarod test. Levels of tissue damage markers and inflammation were measured using western blot, qPCR, and immunofluorescence. In vitro assays were used to evaluate whether trovafloxacin blocks ATP release and cell migration in a chemotactic-stimulated microglia cell line.

Results: Trovafloxacin treatment of CCI-injured mice significantly reduced tissue damage markers and improved locomotor deficits. In addition, trovafloxacin treatment significantly reduced mRNA levels of several pro-inflammatory cytokines (IL-1 beta, IL-6, and TNF-alpha), which correlates with an overall reduction in the accumulation of inflammatory cell types (neutrophils, microglia/macrophages, and astroglia) at the injury zone. To determine whether trovafloxacin exerted these effects by direct action on immune cells, we evaluated its effect on ATP release and cell migration using a chemotactic-stimulated microglial cell line. We found that trovafloxacin significantly inhibited both ATP release and migration of these cells.

Conclusion: Our results show that trovafloxacin administration has pronounced anti-inflammatory and neuroprotective effects following brain injury. These findings lay the foundation for future studies to directly test a role for Panx1 channels in pathological inflammation following brain trauma.

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AU Leung, JYK

Bennett, WR

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King, Anna E.

Chung, Roger S.

TI The impact of metallothionein-II on microglial response to tumor

necrosis factor-alpha (TNFα) and downstream effects on neuronal

regeneration

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Metallothionein; Microglia; Low-density lipoprotein receptor-related

protein-1; Low-density lipoprotein receptor-related protein-2; Axon

regeneration; Traumatic brain injury

ID BRAIN-INJURY; CHONDROITINASE ABC; CNS; EXPRESSION

AB Background: The extracellular environment plays an important role in supporting the regeneration of axons after injury. Metallothionein-II (MTII) is a metal-binding protein known for its neuroprotective effect by directly stimulating the growth of axons after injury. Previous studies have shown that MTII also modulates the response of astrocytes and microglia after injury. However, a detailed analysis describing how MTII modulates the interaction between microglia and neurons is lacking.

Methods: We introduced fluorescently labelled MTII into the cortex at the time of needlestick injury to investigate the cellular uptake of MTII using immunohistochemistry with antibodies against cell-type-specific markers. The role of MTII in modulating the effect of microglia on axon outgrowth following an inflammatory response is further investigated using a co-culture model involving primary rodent microglia pre-treated with TNF alpha and primary rodent cortical neurons. The axon lengths were assessed 24 h after the plating of the neurons onto treated microglia. We also utilised siRNA to knockdown the expression of LRP1, which allows us to investigate the role of LRP1 receptors in the MTII-mediated effect of microglia on axon outgrowth.

Results: Fluorescently labelled MTII was found to be associated with neurons, astrocytes and microglia following injury in vivo. Microglia-neuron co-culture experiments demonstrated that exogenous MTII altered the response of microglia to TNF alpha. The neurons plated onto the TNF alpha-stimulated microglia pre-treated with MTII have shown a significantly longer axonal length compare to the TNF alpha-stimulated microglia without the MTII treatment. This suggested that MTII reduce cytokine-stimulated activation of microglia, which would ordinarily impair neurite outgrowth. This inhibitory effect of MTII on activated microglia was blocked by siRNA-mediated downregulation of LRP1 receptor expression in microglia, suggesting that MTII acts via the LRP1 receptor on microglia.

Conclusions: This study demonstrates that exogenous MTII acts via the LRP1 receptor to alter the inflammatory response of microglia following TNF alpha stimulation, providing a more supportive environment for axon growth.

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NR 24

TC 9

Z9 9

U1 0

U2 1

PU BIOMED CENTRAL LTD

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AF Gao, Tielei

Chen, Zhe

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Yuan, Hui

Wang, Yuena

Peng, Xue

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Yang, Jinxia

Xu, Changqing

TI Inhibition of HMGB1 mediates neuroprotection of traumatic brain injury

by modulating the microglia/macrophage polarization

SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS

LA English

DT Article

DE Glycyrrhizin; HMGB1; Microglia/macrophage; M1/M2 phenotype; Traumatic

brain injury

ID MACROPHAGE PHENOTYPE; THERAPEUTIC TARGET; GLYCYRRHIZIN; ACTIVATION;

INFLAMMATION; EXPRESSION; PROTEIN; REPAIR; M1

AB Microglia/Macrophages have a double-edged role in secondary brain damage after traumatic brain injury (TBI) depending on polarization toward proinflammatory M1 or anti-inflammatory M2 phenotypes. Recently, high-mobility group box 1 (HMGB1) was found to influence the polarization of macrophages. In this study, glycyrrhizin (GL), an inhibitor of HMGB1, was used to investigate whether the inhibition of HMGB1 could modulate microglia/macrophage polarization after TBI. The results showed that treatment with GL improved the neurological function recovery, reduced the lesion volume, and inhibited the release and expression of HMGB1 after TBI. In addition, the administration of GL suppressed M1 phenotype activation and promoted M2 phenotype activation of microglia/macrophages. In conclusion, the results suggested that GL attenuated TBI by inhibiting M1 phenotype while inducing M2 phenotype activation of microglia/macrophages, at least partly through inhibiting HMGB1. Also, targeting HMGB1 to modulate the microglia/macrophage polarization should be one potential therapeutic approach for TBI. (C) 2018 Elsevier Inc. All rights reserved.

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PT J

AU Karelina, K

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AF Karelina, Kate

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Weil, Zachary M.

TI Minocycline blocks traumatic brain injury-induced alcohol consumption

and nucleus accumbens inflammation in adolescent male mice

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE Traumatic brain injury; Alcohol; Neuroinflammation; Microglia;

Minocycline

ID VENTRAL TEGMENTAL AREA; MICROGLIAL ACTIVATION; ETHANOL-CONSUMPTION;

SUBSTANCE-ABUSE; DRUG-USE; DRINKING; NEUROINFLAMMATION; INTERLEUKIN-1;

DEGENERATION; MODULATION

AB Alcohol use is a well characterized risk factor for traumatic brain injury (TBI); however, emerging clinical and experimental research suggests that TBI may also be an independent risk factor for the development of alcohol use disorders. In particular, TBIs incurred early in life predict the development of problem alcohol use and increase vulnerability to neuroinflammation as a consequence of alcohol use. Critically, the neuroinflammatory response to alcohol, mediated in large part by microglia, may also function as a driver of further alcohol use. Here, we tested the hypothesis that TBI increases alcohol consumption through microglia-mediated neuroinflammation. Mice were injured as juveniles and alcohol consumption and preference were assessed in a free-choice voluntary drinking paradigm in adolescence. TBI increased alcohol consumption; however, treatment with minocycline, an inhibitor of microglial activation, reduced alcohol intake in TBI mice to sham levels. Moreover, a single injection of ethanol (2 g/kg) significantly increased microglial activation in the nucleus accumbens and microglial expression of the proinfiammatory cytokine IL-1 beta in TBI, but not sham or minocycline-treated, mice. Our data implicate TBI-induced microglial activation as a possible mechanism for the development of alcohol use disorders. (C) 2018 Elsevier Inc. All rights reserved.

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TI Effects of DHA on Hippocampal Autophagy and Lysosome Function After

Traumatic Brain Injury

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article

DE Autophagy; Cortical contusion injury; Docosahexaenoic acid; Lysosome;

Microglial polarization; Secondary injury

ID OXIDATIVE STRESS; CELL-SURVIVAL; CATHEPSIN-D; PROTEIN; RAT;

BIOSYNTHESIS; BIOGENESIS; EXPRESSION; TRANSIENT; MODEL

AB Traumatic brain injury (TBI) triggers endoplasmic reticulum (ER) stress and impairs autophagic clearance of damaged organelles and toxic macromolecules. In this study, we investigated the effects of the post-TBI administration of docosahexaenoic acid (DHA) on improving hippocampal autophagy flux and cognitive functions of rats. TBI was induced by cortical contusion injury in Sprague-Dawley rats, which received DHA (16 mg/kg in DMSO, intraperitoneal administration) or vehicle DMSO (1 ml/kg) with an initial dose within 15 min after the injury, followed by a daily dose for 3 or 7 days. First, RT-qPCR reveals that TBI induced a significant elevation in expression of autophagy-related genes in the hippocampus, including SQSTM1/p62 (sequestosome 1), lysosomal-associated membrane proteins 1 and 2 (Lamp1 and Lamp2), and cathepsin D (Ctsd). Upregulation of the corresponding autophagy-related proteins was detected by immunoblotting and immunostaining. In contrast, the DHA-treated rats did not exhibit the TBI-induced autophagy biogenesis and showed restored CTSD protein expression and activity. T-2-weighted images and diffusion tensor imaging (DTI) of ex vivo brains showed that DHA reduced both gray matter and white matter damages in cortical and hippocampal tissues. DHA-treated animals performed better than the vehicle control group on the Morris water maze test. Taken together, these findings suggest that TBI triggers sustained stimulation of autophagy biogenesis, autophagy flux, and lysosomal functions in the hippocampus. Swift post-injury DHA administration restores hippocampal lysosomal biogenesis and function, demonstrating its therapeutic potential.

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TI Therapeutic time window of multipotent adult progenitor therapy after

traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Microglia; Neuroinflammation; Spatial learning and blood-brain barrier

ID ACTIVATED MICROGLIAL/MACROPHAGE RESPONSE; CONTROLLED CORTICAL IMPACT;

MICROGLIAL ACTIVATION; CELLS; RATS; RECRUITMENT; NEUROINFLAMMATION;

TRANSPLANTATION; EXPRESSION; INFARCTION

AB Background: Traumatic brain injury (TBI) is a major cause of death and disability. TBI results in a prolonged secondary central neuro-inflammatory response. Previously, we have demonstrated that multiple doses (2 and 24 h after TBI) of multipotent adult progenitor cells (MAPC) delivered intravenously preserve the blood-brain barrier (BBB), improve spatial learning, and decrease activated microglia/macrophages in the dentate gyrus of the hippocampus. In order to determine if there is an optimum treatment window to preserve the BBB, improve cognitive behavior, and attenuate the activated microglia/macrophages, we administered MAPC at various clinically relevant intervals.

Methods: We administered two injections intravenously of MAPC treatment at hours 2 and 24 (2/24), 6 and 24 (6/24), 12 and 36 (12/36), or 36 and 72 (36/72) post cortical contusion injury (CCI) at a concentration of 10 million/kg. For BBB experiments, animals that received MAPC at 2/24, 6/24, and 12/36 were euthanized 72 h post injury. The 36/72 treated group was harvested at 96 h post injury.

Results: Administration of MAPC resulted in a significant decrease in BBB permeability when administered at 2/24 h after TBI only. For behavior experiments, animals were harvested post behavior paradigm. There was a significant improvement in spatial learning (120 days post injury) when compared to cortical contusion injury (CCI) in groups when MAPC was administered at or before 24 h. In addition, there was a significant decrease in activated microglia/macrophages in the dentate gyrus of hippocampus of the treated group (2/24) only when compared to CCI.

Conclusions: Intravenous injections of MAPC at or before 24 h after CCI resulted in improvement of the BBB, improved cognitive behavior, and attenuated activated microglia/macrophages in the dentate gyrus.

C1 [Bedi, Supinder S.; Aertker, Benjamin M.; Liao, George P.; Caplan, Henry W.; Bhattarai, Deepa; Mandy, Fanni; Mandy, Franciska; Fernandez, Luis G.; Zelnick, Pamela; Mitchell, Matthew B.; Schiffer, Walter; Johnson, Margaret; Denson, Emma; Prabhakara, Karthik; Xue, Hasen; Smith, Philippa; Uray, Karen; Olson, Scott D.; Cox, Charles S., Jr.] Univ Texas Hlth Sci Ctr Houston, Dept Pediat Surg, Houston, TX 77030 USA.

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TI Neuroprotective Effects of Platonin, a Therapeutic Immunomodulating

Medicine, on Traumatic Brain Injury in Mice after Controlled Cortical

Impact

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE traumatic brain injury; platonin; neuroinflammation; microglial

activation; free radical

ID PHOTOSENSITIZING DYE; INHIBITORY CONTROL; ISCHEMIC-STROKE; APOPTOSIS;

INFLAMMATION; ACTIVATION; BEHAVIOR; DEFICITS; MODELS; CELLS

AB Traumatic brain injury (TBI) is one of the leading causes of mortality worldwide and leads to persistent cognitive, sensory, motor dysfunction, and emotional disorders. TBI-caused primary injury results in structural damage to brain tissues. Following the primary injury, secondary injuries which are accompanied by neuroinflammation, microglial activation, and additional cell death subsequently occur. Platonin, a cyanine photosensitizing dye, has been used to treat trauma, ulcers, and some types of acute inflammation. In the present study, the neuroprotective effects of platonin against TBI were explored in a controlled cortical impact (CCI) injury model in mice. Treatment with platonin ( 200 mu g/kg) significantly reduced the neurological severity score, general locomotor activity, and anxiety-related behavior, and improved the rotarod performance of CCI-injured mice. In addition, platonin reduced lesion volumes, the expression of cleaved caspase-3, and microglial activation in TBI-insulted brains. Platonin also suppressed messenger (m)RNA levels of caspase-3, caspase-1, cyclooxygenase-2, tumor necrosis factor-alpha, interleukin-6, and interleukin-1 alpha. On the other hand, free radical production after TBI was obviously attenuated in platonin-treated mice. Treatment with platonin exhibited prominent neuroprotective properties against TBI in a CCI mouse model through its anti-inflammatory, anti-apoptotic, and anti-free radical capabilities. This evidence collectively indicates that platonin may be a potential therapeutic medicine for use with TBIs.

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TI Effects of Veliparib on Microglial Activation and Functional Outcomes

after Traumatic Brain Injury in the Rat and Pig

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE axonal injury; IL1-beta; MMP9; TNF alpha

ID POLY(ADP-RIBOSE) POLYMERASE INHIBITOR; NAD(+) DEPLETION;

GENE-EXPRESSION; SWINE MODEL; NEURODEGENERATION; INFLAMMATION;

TRANSCRIPTION; NEUROBIOLOGY; DEFICIENT; RECOVERY

AB The inflammation response induced by brain trauma can impair recovery. This response requires several hours to develop fully and thus provides a clinically relevant therapeutic window of opportunity. Poly(ADP-ribose) polymerase inhibitors suppress inflammatory responses, including brain microglial activation. We evaluated delayed treatment with veliparib, a poly(ADPribose) polymerase inhibitor, currently in clinical trials as a cancer therapeutic, in rats and pigs subjected to controlled cortical impact (CCI). In rats, CCI induced a robust inflammatory response at the lesion margins, scattered cell death in the dentate gyrus, and a delayed, progressive loss of corpus callosum axons. Pre-determined measures of cognitive and motor function showed evidence of attentional deficits that resolved after three weeks and motor deficits that recovered only partially over eight weeks. Veliparib was administered beginning 2 or 24 h after CCI and continued for up to 12 days. Veliparib suppressed CCI-induced microglial activation at doses of 3mg/kg or higher and reduced reactive astrocytosis and cell death in the dentate gyrus, but had no significant effect on delayed axonal loss or functional recovery. In pigs, CCI similarly induced a perilesional microglial activation that was attenuated by veliparib. CCI in the pig did not, however, induce detectable persisting cognitive or motor impairment. Our results showed veliparib suppression of CCI-induced microglial activation with a delay-to-treatment interval of at least 24 h in both rats and pigs, but with no associated functional improvement. The lack of improvement in long-term recovery underscores the complexities in translating anti-inflammatory effects to clinically relevant outcomes.

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TI Microglial Inflammasome Activation in Penetrating Ballistic-Like Brain

Injury

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DE inflammasome; microglia; penetrating ballistic-like brain injury;

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ID INNATE IMMUNE-RESPONSE; NLRP3 INFLAMMASOME; CELL-DEATH; LOADING CONTROL;

TOTAL PROTEIN; CEREBROSPINAL-FLUID; GUNSHOT WOUNDS; UNITED-STATES;

GASDERMIN D; BETA-ACTIN

AB Penetrating traumatic brain injury (PTBI) is a significant cause of death and disability in the United States. Inflammasomes are one of the key regulators of the interleukin (IL)-1 beta mediated inflammatory responses after traumatic brain injury. However, the contribution of inflammasome signaling after PTBI has not been determined. In this study, adult male Sprague-Dawley rats were subjected to sham procedures or penetrating ballistic-like brain injury (PBBI) and sacrificed at various time-points. Tissues were assessed by immunoblot analysis for expression of IL-1 beta, IL-18, and components of the inflammasome: apoptosis-associated speck-like protein containing a caspase-activation and recruitment domain (ASC), caspase-1, X-linked inhibitor of apoptosis protein (XIAP), nucleotide-binding oligomerization domain (NOD)-like receptor protein 3 (NLRP3), and gasdermin-D (GSDMD). Specific cell types expressing inflammasome proteins also were evaluated immunohistochemically and assessed quantitatively. After PBBI, expression of IL-1 beta, IL-18, caspase-1, ASC, XIAP, and NLRP3 peaked around 48 h. Brain protein lysates from PTBI animals showed pyroptosome formation evidenced by ASC laddering, and also contained increased expression of GSDMD at 48 h after injury. ASC-positive immunoreactive neurons within the perilesional cortex were observed at 24 h. At 48 h, ASC expression was concentrated in morphologically activated cortical microglia. This expression of ASC in activated microglia persisted until 12 weeks following PBBI. This is the first report of inflammasome activation after PBBI. Our results demonstrate cell-specific patterns of inflammasome activation and pyroptosis predominantly in microglia, suggesting a sustained pro-inflammatory state following PBBI, thus offering a therapeutic target for this type of brain injury.

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TI The inflammatory Continuum of Traumatic Brain injury and Alzheimer's

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LA English

DT Review

DE traumatic brain injury; Alzheimer's disease; neuroinflammation;

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ID AMYLOID PRECURSOR PROTEIN; IMPROVES COGNITIVE FUNCTION; FLUID PERCUSSION

INJURY; TRANSGENIC MOUSE MODEL; CLOSED-HEAD INJURY; A-BETA DEPOSITION;

TAU PATHOLOGY; MICROGLIAL ACTIVATION; APOLIPOPROTEIN-E; AXONAL INJURY

AB The post-injury inflammatory response is a key mediator in long-term recovery from traumatic brain injury (TBI). Moreover, the immune response to TBI, mediated by microglia and macrophages, is influenced by existing brain pathology and by secondary immune challenges. For example, recent evidence shows that the presence of beta-amyloid and phosphorylated tau protein, two hallmark features of AD that increase during normal aging, substantially alter the macrophage response to TBI. Additional data demonstrate that post-injury microglia are "primed" and become hyper-reactive following a subsequent acute immune challenge thereby worsening recovery. These alterations may increase the incidence of neuropsychiatric complications after TBI and may also increase the frequency of neurodegenerative pathology. Therefore, the purpose of this review is to summarize experimental studies examining the relationship between TBI and development of AD-like pathology with an emphasis on the acute and chronic microglial and macrophage response following injury. Furthermore, studies will be high-lighted that examine the degree to which beta-amyloid and tau accumulation as well as pre-and post-injury immune stressors influence outcome after TBI. Collectively, the studies described in this review suggest that the brain's immune response to injury is a key mediator in recovery, and if compromised by previous, coincident, or subsequent immune stressors, post-injury pathology and behavioral recovery will be altered.

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TI MicroRNA let-7c-5p improves neurological outcomes in a murine model of

traumatic brain injury by suppressing neuroinflammation and regulating

microglial activation

SO BRAIN RESEARCH

LA English

DT Article

DE MicroRNA let-7c-5p; Traumatic brain injury; Microglia; Neuroinflammation

ID MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS; KINASE-C-DELTA; NF-KAPPA-B;

WHITE-MATTER; INHIBITION; PROMOTES

AB MicroRNAs (miRNAs) are a class of non-coding small RNAs that regulate the expression of target genes. They derive from pre-miRNAs that are enzymatically processed by dicer to 22 nucleotide mature miRNAs. Members of the pre-miRNA lethal-7 (let-7) are known to regulate cell proliferation and apoptosis. Here, we showed that the level of let-7c-5p, a key member of the let-7 family, was rapidly reduced in the traumatically injured foci in brains of adult C57BL/6J mice and gradually recovered to the pre-injury level 14 days after traumatic brain injury (TBI) induction. This finding led us to test whether upregulating let-7c-5p in murine cerebral tissue by intracerebroventricular injection (ICV) of let-7c-5p mimic could improve the outcomes of mice subjected to controlled cortical impact (CCI). We found that let-7c-5p overexpression attenuated TBI-induced neurological dysfunction and brain edema. The improvements were attributed to let-7c-5p-mediated inhibiting neuroinflammation and attenuation of microglia/-macrophage activation, both inhibiting M1 polarization and enhancing M2 polarization. In vitro experiments, we observed that let-7c-Sp was decreased in primary microglia activated by LPS treatment or oxygen/glucose deprivation (OGD). Transfection of let-7c-5p mimic suppressed the release of inflammatory mediators in cultured activated primary microglia. In addition, the expressions of caspase-3, a let-7c-5p putative target gene, and the PKC-delta which mediates effect of caspase-3 were inhibited by let-7c-5p in a murine model of TBI. Taken together, these results define the biological activities of cerebral let-7c-5p and delineate its therapeutic potential for improving the neurological outcome of TBI. (C) 2018 Elsevier B.V. All rights reserved.

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Gao, Hongzhi

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Hu, Weipeng

TI Valproic Acid Attenuates Traumatic Brain Injury-Induced Inflammation

<i>in Vivo</i>: Involvement of Autophagy and the Nrf2/ARE Signaling

Pathway

SO FRONTIERS IN MOLECULAR NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury; valproic acid; HDAC3; microglia; inflammatory;

autophagy; Nrf2/ARE pathway

ID HEMORRHAGIC-SHOCK; RATS; APOPTOSIS; CELLS; NEUROPROTECTION;

NEUROGENESIS; SUPPRESSION; ACTIVATION; INHIBITION; EXPRESSION

AB Microglial activation and the inflammatory response in the central nervous system (CNS) play important roles in secondary damage after traumatic brain injury (TBI). Transcriptional activation of genes that limit secondary damage to the CNS are mediated by a cis-acting element called the antioxidant responsive element (ARE). ARE is known to associate with the transcription factor NF-E2-related factor 2 (Nrf2), a transcription factor that is associated with histone deacetylases (HDACs). This pathway, known as the Nrf2/ARE pathway, is a critical antioxidative factor pathway that regulates the balance of oxygen free radicals and the inflammatory response, and is also related to autophagic activities. Although valproic acid (VPA) is known to inhibit HDACs, it is unclear whether VPA plays a role in the microglia-mediated neuroinflammatory response after TBI via regulating oxidative stress and autophagy induced by the Nrf2/ARE signaling pathway. In this study, we demonstrate that microglial activation, oxidative stress, autophagy, and the Nrf2/ARE signaling pathway play essential roles in secondary injury following TBI. Treatment with VPA alleviated TBI-induced secondary brain injury, including neurological deficits, cerebral edema, and neuronal apoptosis. Moreover, VPA treatment upregulated the occurrence of autophagy and Nrf2/ARE pathway activity after TBI, and there was an increase in H3, H4 histone acetylation levels, accompanied by decreased transcriptional activity of the HDAC3 promoter in cortical lesions. These results suggest that VPA-mediated up-regulation of autophagy and antioxidative responses are likely due to increased activation of Nrf2/ARE pathway, through direct inhibition of HDAC3. This inhibition further reduces TBI-induced microglial activation and the subsequent inflammatory response, ultimately leading to neuroprotection.

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TI Omega-3 polyunsaturated fatty acid attenuates the inflammatory response

by modulating microglia polarization through SIRT1-mediated

deacetylation of the HMGB1/NF-κB pathway following experimental

traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Traumatic brain injury; Omega-3 polyunsaturated fatty acid; Microglia

polarization; Neuroinflammation; Sirtuin1; HMGB1/NF-kappa B pathway

ID WHITE-MATTER INJURY; NEUROLOGICAL DEFICITS; HMGB1; ACTIVATION; HDAC;

OMEGA-3-FATTY-ACIDS; SUPPRESSES; HEMORRHAGE; INHIBITION; PHENOTYPE

AB Background: Microglial polarization and the subsequent neuroinflammatory response are contributing factors for traumatic brain injury (TBI)-induced secondary injury. High mobile group box 1 (HMGB1) mediates the activation of the NF-kappa B pathway, and it is considered to be pivotal in the late neuroinflammatory response. Activation of the HMGB1/NF-kappa B pathway is closely related to HMGB1 acetylation, which is regulated by the sirtuin (SIRT) family of proteins. Omega-3 polyunsaturated fatty acids (omega-3 PUFA) are known to have antioxidative and anti-inflammatory effects. We previously demonstrated that omega-3 PUFA inhibited TBI-induced microglial activation and the subsequent neuroinflammatory response by regulating the HMGB1/NF-kappa B signaling pathway. However, no studies have elucidated if omega-3 PUFA affects the HMGB1/NF-kappa B pathway in a HMGB1 deacetylation of dependent SIRT1 manner, thus regulating microglial polarization and the subsequent neuroinflammatory response.

Methods: The Feeney DM TBI model was adopted to induce brain injury in rats. Modified neurological severity scores, rotarod test, brain water content, and Nissl staining were employed to determine the neuroprotective effects of omega-3 PUFA supplementation. Assessment of microglia polarization and pro-inflammatory markers, such as tumor necrosis factor (TNF)-alpha, interleukin (IL)-1 beta, IL-6, and HMGB1, were used to evaluate the neuroinflammatory responses and the anti-inflammatory effects of omega-3 PUFA supplementation. Immunofluorescent staining and western blot analysis were used to detect HMGB1 nuclear translocation, secretion, and HMGB1/NF-kappa B signaling pathway activation to evaluate the effects of omega-3 PUFA supplementation. The impact of SIRT1 deacetylase activity on HMGB1 acetylation and the interaction between HMGB1 and SIRT1 were assessed to evaluate anti-inflammation effects of omega-3 PUFAs, and also, whether these effects were dependent on a SIRT1-HMGB1/NF-kappa B axis to gain further insight into the mechanisms underlying the development of the neuroinflammatory response after TBI.

Results: The results of our study showed that omega-3 PUFA supplementation promoted a shift from the M1 microglial phenotype to the M2 microglial phenotype and inhibited microglial activation, thus reducing TBI-induced inflammatory factors. In addition,omega-3 PUFA-mediated downregulation of HMGB1 acetylation and its extracellular secretion was found to be likely due to increased SIRT1 activity. We also found that treatment with omega-3 PUFA inhibited HMGB1 acetylation and induced direct interactions between SIRT1 and HMGB1 by elevating SIRT1 activity following TBI. These events lead to inhibition of HMGB1 nucleocytoplasmic translocation/extracellular secretion and alleviated HMGB1-mediated activation of the NF-kappa B pathway following TBI-induced microglial activation, thus inhibiting the subsequent inflammatory response.

Conclusions: The results of this study suggest that omega-3 PUFA supplementation attenuates the inflammatory response by modulating microglial polarization through SIRT1-mediated deacetylation of the HMGB1/NF-kappa B pathway, leading to neuroprotective effects following experimental traumatic brain injury.

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TI Neuroprotective effects of metformin on traumatic brain injury in rats

associated with NF-κB and MAPK signaling pathway

SO BRAIN RESEARCH BULLETIN

LA English

DT Article

DE Metformin; Traumatic brain injury; Inflammation; Microglia activation

ID OXIDATIVE STRESS; SUBARACHNOID HEMORRHAGE; POSSIBLE INVOLVEMENT;

CEREBRAL-ISCHEMIA; ACTIVATION; MICROGLIA; RESPONSES; MODULATION;

DISEASE; OXYGEN

AB Traumatic brain injury (TBI) triggers a complex sequence of inflammatory responses that contribute to secondary injury. Metformin, a first-line drug used to treat type 2 diabetes, is reported to exhibit potent anti-inflammatory activity on diseases associated with the central nervous system (CNS). The aim of this study is to investigate the potential neuroprotective effects of metformin on acute brain injury after TBI and explore the underlying mechanisms. Male Sprague-Dawley (SD) rats were divided into four groups: sham group, TBI group, TBI + saline (NS) group and TBI + metformin group. A weight-dropping model was employed to induce TBI in rats. Modified neurological severity scores (mNSS) were employed to assess the short-term neurological deficits, neuronal degeneration and apoptosis in the brain tissues were assayed with Fluoro-Jade B and TUNEL staining, immunofluorescence was designed to investigate microglial activation. The mRNA and protein expression levels of pro-inflammatory cytokines such as necrosis factor-alpha (TNF-alpha), interleukin-beta (IL-1 beta) and nterleukin-6 (IL-6) were evaluated by real-time quantitative reverse transcriptase polymerase chain reaction (QPCR) and enzyme-linked immunosorbent assay (ELISA). Western blotting analysis was engaged to examine the expression of NF-kappa B p65 and phosphorylation of ERK1/2 and p38 MAPK. Our results showed that metformin significantly ameliorated neurological deficit, cerebral edema and neuronal apoptosis in rats following TBI. Moreover, metformin administration inhibited microglial activation and decreased the production of pro-inflammatory cytokines including TNF-alpha, IL-beta and IL-6. In addition, metformin inhibited the translocation of NF-kappa B p65 from cytoplasm into the nucleus, as well as the phosphorylation of ERK1/2 and p38 MAPK. This study suggests that metformin administration inhibits microglia activation-mediated inflammation via NF-kappa B and MAPK signaling pathway to improve neurobehavioral function following TBI, which provide a potential therapeutic benefit in treating brain injury.

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TI NADPH oxidases in traumatic brain injury - Promising therapeutic

targets?

SO REDOX BIOLOGY

LA English

DT Review

DE NADPH oxidase; NOX; NOX2; NOX4; NOX1; Traumatic brain injury; TBI; FPI;

CCI; Controlled cortical impact; CHI; ROS; Oxidative stress; Microglia;

Apocynin; gp91ds-tat

ID EXHALED BREATH CONDENSATE; OXIDATIVE STRESS; REACTIVE OXYGEN;

NOX-FAMILY; NAD(P)H OXIDASE; HYDROGEN-PEROXIDE; CLINICAL-TRIALS;

SEX-DIFFERENCES; APOCYNIN; INHIBITION

AB Traumatic brain injury (TBI) is a major cause of death and disability worldwide. Despite intense investigation, no neuroprotective agents for TBI have yet translated to the clinic. Recent efforts have focused on identifying potential therapeutic targets that underlie the secondary TBI pathology that evolves minutes to years following the initial injury. Oxidative stress is a key player in this complex cascade of secondary injury mechanisms and prominently contributes to neurodegeneration and neuroinflammation. NADPH oxidase (NOX) is a family of enzymes whose unique function is to produce reactive oxygen species (ROS). Human post-mortem and animal studies have identified elevated NOX2 and NOX4 levels in the injured brain, suggesting that these two NOXs are involved in the pathogenesis of TBI. In support of this, NOX2 and NOX4 deletion studies have collectively revealed that targeting NOX enzymes can reduce oxidative stress, attenuate neuroinflammation, promote neuronal survival, and improve functional outcomes following TBI. In addition, NOX inhibitor studies have confirmed these findings and demonstrated an extended critical window of efficacious TBI treatment. Finally, the translational potential, caveats, and future directions of the field are highlighted and discussed throughout the review.

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TI The Contribution of Fibrinogen to Inflammation and Neuronal Density in

Human Traumatic Brain Injury

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DE fibrinogen; microglia; neurodegeneration; TBI

ID AXONAL INJURY; BARRIER DISRUPTION; MOUSE MODEL; HEAD-INJURY; MODERATE;

BETA; NEUROPATHOLOGY; ACTIVATION; MICROGLIA; IMMEDIATE

AB Traumatic brain injury (TBI) is a leading cause of death and disability, particularly among the young. Despite this, no disease-specific treatments exist. Recently, blood-brain barrier disruption and parenchymal fibrinogen deposition have been reported in acute traumatic brain injury and in long-term survival; however, their contribution to the neuropathology of TBI remains unknown. The presence of fibrinogena well-documented activator of microglia/macrophagesmay be associated with neuroinflammation, and neuronal/axonal injury. To test this hypothesis, cases of human TBI with survival times ranging from 12h to 13 years (survival <2 months, n=15; survival >1 year, n=6) were compared with uninjured controls (n=15). Tissue was selected from the frontal lobe, temporal lobe, corpus callosum, cingulate gyrus, and brainstem, and the extent of plasma protein (fibrinogen and immunoglobulin G [IgG]) deposition, microglial/macrophage activation (CD68 and ionized calcium-binding adapter molecule 1 [Iba-1] immunoreactivity), neuronal density, and axonal transport impairment (beta-amyloid precursor protein [beta APP] immunoreactivity) were assessed. Quantitative analysis revealed a significant increase in parenchymal fibrinogen and IgG deposition following acute TBI compared with long-term survival and control. Fibrinogen, but not IgG, was associated with microglial/macrophage activation and a significant reduction in neuronal density. Perivascular fibrinogen deposition also was associated with microglial/macrophage clustering and accrual of beta APP in axonal spheroids, albeit rarely. These findings mandate the future exploration of causal relationships between fibrinogen deposition, microglia/macrophage activation, and potential neuronal loss in acute TBI.

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TI Polarization of Microglia to the M2 Phenotype in a Peroxisome

Proliferator-Activated Receptor Gamma-Dependent Manner Attenuates Axonal

Injury Induced by Traumatic Brain Injury in Mice

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DE axonal injury; microglia; neuroinflammation; peroxisome

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ID PPAR-GAMMA; MACROPHAGE ACTIVATION; TNF-ALPHA; INFLAMMATION;

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AB Increasing evidence indicates that activated microglia play an important role in the inflammatory response following traumatic brain injury (TBI). Inhibiting M1 and stimulating M2 activated microglia have demonstrated protective effects in several animal models of central nervous system diseases. However, it is not clear whether the polarization of microglia to M2 attenuates axonal injury following TBI. In this study, we used a lateral fluid percussion injury device to induce axonal injury in mice. Mice were randomly assigned to the sham, TBI, TBI + rosiglitazone (peroxisome proliferator-activated receptor gamma [PPAR-gamma] agonist), and TBI + GW9662 (PPAR-gamma antagonist) groups. Axonal injury was assessed using immunohistochemical staining for beta amyloid precursor protein. The inflammatory response was assessed by enzyme-linked immunosorbent assay, microglia polarization was assessed using specific markers of M1 and M2 microglia, and neurological function was assessed using the neurological severity score. Following TBI, microglia of the M1 phenotype increased significantly, while those of the M2 phenotype decreased. Rosiglitazone-induced PPAR-gamma activation promoted microglia polarization to the M2 phenotype, which reduced the inflammatory response, attenuated axonal injury in the cerebral cortex, and improved neurological function. Conversely, GW9662 inhibited the polarization of microglia to M2 and aggravated inflammation and axonal injury. Our in vitro findings in lipopolysaccharide-induced microglia were consistent with those of our in vivo experiments. In conclusion, the polarization of microglia to the M2 phenotype via PPAR-gamma activation attenuated axonal injury following TBI in mice, which may be a potential therapeutic approach for TBI-induced axonal injury.

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TI A Single Primary Blast-Induced Traumatic Brain Injury in a Rodent Model

Causes Cell-Type Dependent Increase in Nicotinamide Adenine Dinucleotide

Phosphate Oxidase Isoforms in Vulnerable Brain Regions

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE astrocytes; blast injury; 4-hydroxynonenal; microglia; neuron; NADPH

oxidase; oxidative stress; traumatic brain injury

ID CENTRAL-NERVOUS-SYSTEM; NADPH-OXIDASE; OXIDATIVE STRESS; FREE-RADICALS;

LIPID-PEROXIDATION; ACTIVATED MICROGLIA; SCALING RULES; INHIBITION;

NOX2; INFLAMMATION

AB Blast-induced traumatic brain injury (bTBI) is a leading cause of morbidity in soldiers on the battlefield and in training sites with long-term neurological and psychological pathologies. Previous studies from our laboratory demonstrated activation of oxidative stress pathways after blast injury, but their distribution among different brain regions and their impact on the pathogenesis of bTBI have not been explored. The present study examined the protein expression of two isoforms: nicotinamide adenine dinucleotide phosphate (NADPH) oxidase 1 and 2 (NOX1, NOX2), corresponding superoxide production, a downstream event of NOX activation, and the extent of lipid peroxidation adducts of 4-hydroxynonenal (4HNE) to a range of proteins. Brain injury was evaluated 4h after the shock-wave exposure, and immunofluorescence signal quantification was performed in different brain regions. Expression of NOX isoforms displayed a differential increase in various brain regions: in hippocampus and thalamus, there was the highest increase of NOX1, whereas in the frontal cortex, there was the highest increase of NOX2 expression. Cell-specific analysis of changes in NOX expression with respect to corresponding controls revealed that blast resulted in a higher increase of NOX1 and NOX 2 levels in neurons compared with astrocytes and microglia. Blast exposure also resulted in increased superoxide levels in different brain regions, and such changes were reflected in 4HNE protein adduct formation. Collectively, this study demonstrates that primary blast TBI induces upregulation of NADPH oxidase isoforms in different regions of the brain parenchyma and that neurons appear to be at higher risk for oxidative damage compared with other neural cells.

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TI Concentrated Conditioned Media from Adipose Tissue Derived Mesenchymal

Stem Cells Mitigates Visual Deficits and Retinal Inflammation Following

Mild Traumatic Brain Injury

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE TBI; adult stem cells; TSG6; retinal ganglion cells; oxidative stress;

TIMP1; microglia; trans-endothelial electrical resistance

ID STROMAL CELLS; GANGLION-CELLS; LUNG INJURY; MOUSE MODEL; INTRAVITREAL

INJECTION; IFATS COLLECTION; RODENT MODEL; SPINAL-CORD; VISION LOSS;

BLAST

AB Blast concussions are a common injury sustained in military combat today. Inflammation due to microglial polarization can drive the development of visual defects following blast injuries. In this study, we assessed whether anti-inflammatory factors released by the mesenchymal stem cells derived from adipose tissue (adipose stem cells, ASC) can limit retinal tissue damage and improve visual function in a mouse model of visual deficits following mild traumatic brain injury. We show that intravitreal injection of 1 mu L of ASC concentrated conditioned medium from cells pre-stimulated with inflammatory cytokines (ASC-CCM) mitigates loss of visual acuity and contrast sensitivity four weeks post blast injury. Moreover, blast mice showed increased retinal expression of genes associated with microglial activation and inflammation by molecular analyses, retinal glial fibrillary acidic protein (GFAP) immunoreactivity, and increased loss of ganglion cells. Interestingly, blast mice that received ASC-CCM improved in all parameters above. In vitro, ASC-CCM not only suppressed microglial activation but also protected against Tumor necrosis alpha (TNF alpha) induced endothelial permeability as measured by transendothelial electrical resistance. Biochemical and molecular analyses demonstrate TSG-6 is highly expressed in ASC-CCM from cells pre-stimulated with TNF alpha and IFN gamma but not from unstimulated cells. Our findings suggest that ASC-CCM mitigates visual deficits of the blast injury through their anti-inflammatory properties on activated pro-inflammatory microglia and endothelial cells. A regenerative therapy for immediate delivery at the time of injury may provide a practical and cost-effective solution against the traumatic effects of blast injuries to the retina.

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TI The Number of IgG-Positive Neurons in the Rat Hippocampus Increases

after Dosed Traumatic Brain Injury

SO NEUROCHEMICAL JOURNAL

LA English

DT Article

DE traumatic brain injury; IgG; microglia; hippocampus; neuroinflammation;

blood-brain barrier

ID FLUID-PERCUSSION INJURY; EPILEPSY; DYSFUNCTION; CONTRIBUTE; MODEL

AB To evaluate the consequences of traumatic brain injury (TBI), we used a model of lateral fluid percussion brain injury in freely moving male Wistar rats. The immediate response to TBI included development of motor excitation and tonic-clonic seizures. Morphological analysis was performed 7 day after TBI. To localize IgG in the brain, rat brain slices were double stained with antibodies against IgG and NeuN (neuronal marker). To evaluate the state of microglia, we performed staining with Isolectin B4 (a microglial marker). The number of neurons was measured in sections stained using the Nissl method. The results show the IgG accumulation in neurons adjacent to cortical focus of trauma. In the hippocampus, IgG was accumulated in the neurons of the ipsilateral hippocampal CA1 and CA2 fields and the dentate gyrus, while in the contralateral hemisphere IgG was accumulated in the neurons of the CA1 field. These changes were accompanied by activation of microglia in the hippocampus, as well as by a decrease in neuronal density in the dentate gyrus of the ipsilateral hippocampus. The results show that TBI leads to bilateral damage to the hippocampus.

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TI Cellular players that shape evolving pathology and neurodegeneration

following traumatic brain injury

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Review

DE Traumatic brain injury; Alzheimer's disease; Neuroinflammation;

Neurodegeneration; Blood-brain barrier; Microglia; Macrophages;

Astrocytes; TREM2

ID CENTRAL-NERVOUS-SYSTEM; ALZHEIMERS-DISEASE; MOUSE MODEL; MICROGLIAL

RESPONSE; TREM2 DEFICIENCY; MYELOID CELLS-2; INFLAMMATION; MACROPHAGES;

ACTIVATION; SUSTAINS

AB Traumatic brain injury (TBI) is one of the leading causes of death and disability worldwide, and has emerged as a critical risk factor for multiple neurodegenerative diseases, particularly Alzheimer's disease (AD). Flow the inflammatory cascade resulting from mechanical stress, axonal shearing and the loss of neurons and glia following initial impact in TBI, contributes to the development of AD-like disease is unclear. Neuroinflammation, characterized by blood-brain barrier (BBB) dysfunction and activation of brain-resident microglia and astrocytes, resulting in secretion of inflammatory mediators and subsequent recruitment of peripheral immune cells has been the focus of extensive research in attempts to identify drug-targets towards improving functional outcomes post TBI. While knowledge of intricate cellular interactions that shape lesion pathophysiology is incomplete, a major limitation in the field is the lack of understanding of how distinct cell types differentially alter TBI pathology. The aim of this review is to highlight functional differences between populations of bone marrow derived, infiltrating monocytes/macrophages and brain-resident microglia based on differential expression of the chemokine receptors CCR2 and CX(3)CR1. This review will focus on how unique subsets of mononuclear phagocytes shape TBI pathophysiology, neurotoxicity and BBB function, in a disease-stage dependent manner. Additionally, this review summarizes the role of multiple microglia and macrophage receptors, namely CCR2, CX(3)CR1 and Triggering Receptor Expressed on Myeloid Cells-2 (TREM2) in pathological neuroinflammation and neurodegeneration vs. recovery following TBI. TREM2 has been implicated in mediating AD-related pathology, and variants in TREM2 are particularly important due to their correlation with exacerbated neurodegeneration. Finally, this review highlights behavioral outcomes associated with microglial vs. macrophage variances, the need for novel treatment strategies that target unique subpopulations of peripheral macrophages, and the importance of development of therapeutics to modulate inflammatory functions of brain-resident microglia at specific stages of TBI. (C) 2018 Elsevier Inc. All rights reserved.

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Hu, Ziyou

Wu, Bingyi

TI Glia maturation factor beta is required for reactive gliosis after

traumatic brain injury in zebrafish

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Glia maturation factor beta; Reactive gliosis; Traumatic brain injury;

Radial glial cells; Microglia

ID FIBRILLARY ACIDIC PROTEIN; REGENERATIVE RESPONSE; ADULT NEUROGENESIS;

EXPRESSION; PROLIFERATION; CELLS; GMF; INFLAMMATION; INDUCTION;

REGULATOR

AB Gliosis is a hallmark of neural pathology that occurs after most forms of central nervous system (CNS) injuries including traumatic brain injury (TBI). Identification of genes that control gliosis may provide novel treatment targets for patients with diverse CNS injuries. Glia maturation factor beta (GMFB) is crucial in brain development and stress response. In the present study, GMFB was found to be widely expressed in adult zebrafish telencephalon. A gmfb mutant zebrafish was created using CRISPR/cas9. In the uninjured zebrafish telencephalon, glial fibrillary acidic protein (GFAP) fibers in gmfb mutants were disorganized and shorter than wild type zebrafish. After TBI, transformation of quiescent type I radial glial cells (RGC) to proliferative type II RGCs was significantly suppressed in the gmfb mutant. RGC proliferation and hypertrophy post-TBI was reduced in gmfb mutants, indicating that reactive gliosis was attenuated. TBI-induced acute inflammation was also found to be alleviated in the gmfb mutant. Morphological changes also suggest attenuation of microglial reactive gliosis. In a mouse model of TBI, GMFB expression was increased around the injury site. These GMFB + cells were identified as astrocytes and microglia. Taken together, the data suggests that GMFB is not only required for normal development of GFAP fibers in the zebrafish telencephalon, but also promotes reactive gliosis after TBI. Our findings provide novel information to help better understand the reactive gliosis process following TBI.

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TI Unique Properties Associated with the Brain Penetrant Iron Chelator HBED

Reveal Remarkable Beneficial Effects after Brain Trauma

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE blood; edema; microglia; traumatic brain injury

ID INTRACEREBRAL HEMORRHAGE; PROSTANOID RECEPTOR; INDUCED ATTENUATION;

DEFEROXAMINE; INJURY; OVERLOAD; DELETION; OUTCOMES; EDEMA; RATS

AB Iron is postulated to contribute to secondary injury after brain trauma through various pathways including oxidative stress and inflammation. Therefore, one goal is to limit iron toxicity by either directly limiting iron activity, or limiting the secondary cascade mediated by iron, therefore rescuing the brain from damage after trauma. The N,N'-Di(2-hydroxybenzyl)ethylenediamine-N,N'-diacetic acid monohydrochloride (HBED) is a unique iron chelator that has the ability to cross the intact blood-brain barrier; it has a higher affinity to iron, and it has a longer half-life than most commonly used chelators. A controlled-cortical impact model of traumatic brain injury (TBI) was induced in mice. Mice were subcutaneously injected with HBED immediately after TBI, then at 12h after, followed by a twice-a-day regimen until an end-point of 3 days. Neurobehavioral tests were performed daily. Cortical injury volume, hemispheric enlargement, and hippocampal swelling were quantified. Perls' iron immunostaining along with markers of gliosis, oxidative stress, and aquaporin (AQP) 4 were also performed. Data revealed that HBED treatment significantly decreases motor deficits and improves recovery after TBI. It also reduces cortical injury volume by 36.6 +/- 6.8% (p<0.001), hippocampal swelling by 23.4 +/- 3.8% (p<0.05), and total hemispheric volume by 13.3 +/- 2.7% (p<0.01). These effects are related to a reduction in microgliosis and oxidiative stress markers in the impacted corpus callosum area by 39.8 +/- 7.3%, and by 80.5 +/- 0.8% (p<0.05), respectively. AQP4 staining is also attenuated in the hippocampus of HBED-treated mice. Therefore, our results suggest that HBED should be considered as a therapeutic tool to facilitate the recovery process following brain trauma.

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University of Florida; State University System of Florida; University of

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TI ABCA1 haplodeficiency affects the brain transcriptome following

traumatic brain injury in mice expressing human APOE isoforms

SO ACTA NEUROPATHOLOGICA COMMUNICATIONS

LA English

DT Article

DE Traumatic brain injury; Apolipoprotein E; ABCA1; Transcriptome; WGCNA;

Microglia sensome

ID APOLIPOPROTEIN-E; NEUROINFLAMMATION; ACTIVATION; DISEASE; GENE;

MODULATION; GENOTYPE; RECOVERY; MODERATE; PROTEIN

AB Expression of human Apolipoprotein E (APOE) modulates the inflammatory response in an isoform specific manner, with APOE4 isoform eliciting a stionger pro-inflammatory response, suggesting a possible mechanism for worse outcome following traumatic brain injury (TBI). APOE lipidation and stability is modulated by ATP-binding cassette transporter A1 (ABCA1), a transmembrane protein that transports lipids and cholesterol onto APOE. We examined the impact of Abca1 deficiency and APOE isoform expression on the response to TBI using 3-months-old, human APOE3(+/+) (E3/Abca1(+/+)) and APOE4(+/+) (E4/Abca1(+/+)) targeted replacement mice, and APOE3(+/+) and APOE4(+/+) mice with only one functional copy of the Abca1 gene (E3/Abca1(+/-); E4/Abca1(+/-)). TBI-treated mice received a craniotomy followed by a controlled cortical impact (CCI) brain injury in the left hemisphere, sham-treated mice received the same surgical procedure without the impact. We performed RNA-seq using samples from cortices and hippocampi followed by genome-wide differential gene expression analysis. We found that TBI significantly impacted unique transcripts within each group, however, the proportion of unique transcripts was highest in E4/Abca1(+/-) mice. Additionally, we found that Abca1 haplodeficiency increased the expression of microglia sensome genes among only APOE4 injured mice, a response not seen in injured APOE3 mice, nor in either group of sham-treated mice. To identify gene networks, or modules, correlated to TBI, APOE isoform and Abca1 haplodeficiency, we used weighted gene co-expression network analysis (WGCNA). The module that positively correlated to TBI groups was associated with immune response and featured hub genes that were microglia-specific, including Trem2, Tyrobp, Cd68 and Hexb. The modules positively correlated with APOE4 isoform and negatively to Abca1 haplodeficient mice represented "protein translation" and "oxidation-reduction process", respectively. Our results reveal E4/Abca1(+/-) TBI mice have a distinct response to injury, and unique gene networks are associated with APOE isoform, Abca1 insufficiency and injury.

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TI Minocycline plus N-acteylcysteine induces remyelination, synergistically

protects oligodendrocytes and modifies neuroinflammation in a rat model

of mild traumatic brain injury

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article

DE Brain trauma; immunohistochemistry; inflammation; microglia; white

matter; oligodendrocytes

ID WHITE-MATTER INJURY; TUMOR-NECROSIS-FACTOR; MICROGLIA/MACROPHAGE

POLARIZATION; MULTIPLE-SCLEROSIS; WORKING-MEMORY; NITRIC-OXIDE;

CELL-DEATH; ACETYLCYSTEINE; INFLAMMATION; ACTIVATION

AB Mild traumatic brain injury afflicts over 2 million people annually and little can be done for the underlying injury. The Food and Drug Administration-approved drugs Minocycline plus N-acetylcysteine (MINO plus NAC) synergistically improved cognition and memory in a rat mild controlled cortical impact (mCCI) model of traumatic brain injury.(3) The underlying cellular and molecular mechanisms of the drug combination are unknown. This study addressed the effect of the drug combination on white matter damage and neuroinflammation after mCCI. Brain tissue from mCCI rats given either sham-injury, saline, MINO alone, NAC alone, or MINO plus NAC was investigated via histology and qPCR at four time points (2, 4, 7, and 14 days post-injury) for markers of white matter damage and neuroinflammation. MINO plus NAC synergistically protected resident oligodendrocytes and decreased the number of oligodendrocyte precursor cells. Activation of microglia/macrophages (MP/MG) was synergistically increased in white matter two days post-injury after MINO plus NAC treatment. Patterns of M1 and M2 MP/MG were also altered after treatment. The modulation of neuroinflammation is a potential mechanism to promote remyelination and improve cognition and memory. These data also provide new and important insights into how drug treatments can induce repair after traumatic brain injury.

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TI Selective NLRP3 inflammasome inhibitor reduces neuroinflammation and

improves long-term neurological outcomes in a murine model of traumatic

updates brain injury

SO NEUROBIOLOGY OF DISEASE

LA English

DT Article

DE Traumatic brain injury; Neuroinflammation; NLRP3 inflammasome;

Interleukin-1 beta; Microglia; MCC950

ID CEREBROSPINAL-FLUID; ACTIVATION; MICE; NEUROPROTECTION; DISRUPTION;

DEFICITS; CORTEX

AB The nucleotide-binding oligomerization domain (NOD)-like receptor family pyrin domain containing 3 (NLRP3) inflammasome-mediated inflammatory response has emerged as a prominent contributor to the pathophysiological processes of traumatic brain injury (TBI). Recently, a potent, selective, small-molecule NLRP3 inflammasome inhibitor, MCC950, was described. Here, we investigated the effect of MCC950 on inflammatory brain injury and long-term neurological outcomes in a mouse model of TBI. Male C57/BL6 mice were subjected to TBI using the controlled cortical impact injury (CCI) system. Western blotting, flow cytometry, and immunofluorescence assays were utilized to analyze post-traumatic NLRP3 inflammasome expression and determine its cellular source. We found that NLRP3 inflammasome expression was significantly increased in the peri-contusional cortex and that microglia were the primary source of this expression. The effects of MCC950 on mice with TBI were then determined using post-assessments including analyses of neurological deficits, brain water content, traumatic lesion volume, neuroinflammation, blood-brain barrier (BBB) integrity, and cell death. MCC950 treatment resulted in a better neurological outcome after TBI by alleviating brain edema, reducing lesion volume, and improving long-term motor and cognitive functions. The therapeutic window for MCC950 against TBI was as long as 6 h. Furthermore, the neuroprotective effect of MCC950 was associated with reduced microglial activation, leukocyte recruitment, and pro-inflammatory cytokine production. In addition, MCC950 preserved BBB integrity, alleviated TBI-induced loss of tight junction proteins, and attenuated cell death. Notably, the efficacy of MCC950 was abolished in microglia-depleted mice. These results indicate that microglia-derived NLRP3 inflammasome may be primarily involved in the inflammatory response to TBI, and specific NLRP3 inflammasome inhibition using MCC950 may be a promising therapeutic approach for patients with TBI.

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TI Bexarotene protects against neurotoxicity partially through a

PPARγ-dependent mechanism in mice following traumatic brain injury

SO NEUROBIOLOGY OF DISEASE

LA English

DT Article

DE Bexarotene; Traumatic brain injury; Neuron; Astrocyte; Microglia;

Peroxisome proliferator-activated receptor; gamma

ID MICROGLIAL ACTIVATION; INFLAMMATION; POLARIZATION; RECOVERY;

NEUROPROTECTION; DIFFERENTIATION; IMPAIRMENT; ASTROCYTES; DAMAGE

AB Traumatic brain injury (TBI) causes a high rate of mortality and disability worldwide, and there exists almost none effective drugs to protect against TBI. Neurotoxicity occurring after TBI can be derived from microglia and astrocytes, and causes neuronal death and synapse loss. Bexarotene has been demonstrated to protect neurons in CNS diseases. In the present study, we aimed to investigate the potential role of bexarotene in protecting against neurotoxicity after TBI, as well as the underlying mechanism. The controlled cortical impact (CCI) model was established on adult C57BL/6 mice, followed by intraperitoneal administration of bexarotene for 14 consecutive days. We found that bexarotene improved sensorimotor function and cognitive recovery in CCI mice. In addition, bexarotene decreased neuronal death and synapse loss, as well as inhibited apoptotic cascade. Moreover, bexarotene treatment reduced M1 microglia polarization, microglia-derived pro-inflammatory cytokines, and the number of A1 astrocytes after CCI. These effects of bexarotene were partially abolished by T0070907, an antagonist of peroxisome proliferator-activated receptor gamma (PPAR gamma). Additionally, bexarotene enhanced nuclear translocation and transcriptional activity of PPAR gamma. These findings show that bexarotene inhibits neurotoxicity in mice after TBI, at least in part through a PPAR gamma-dependent mechanism.

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TI Teriflunomide Modulates Vascular Permeability and Microglial Activation

after Experimental Traumatic Brain Injury

SO MOLECULAR THERAPY

LA English

DT Article

ID CONTROLLED CORTICAL IMPACT; DE-NOVO SYNTHESIS; AGOUTI RAT MODEL;

RHEUMATOID-ARTHRITIS; T-LYMPHOCYTES; IMMUNOMODULATORY DRUG;

CLINICAL-TRIAL; DENTATE GYRUS; DOUBLE-BLIND; LEFLUNOMIDE

AB Despite intensive research and clinical trials with numerous therapeutic treatments, traumatic brain injury (TBI) is a serious public health problem in the United States. There is no effective FDA-approved treatment to reduce morbidity and mortality associated with TBI. Inflammation plays a pivotal role in the pathogenesis of TBI. We looked to re-purpose existing drugs that reduce immune activation without broad immunosuppression. Teriflunomide, an FDA-approved drug, has been shown to modulate immunological responses outside of its ability to inhibit pyrimidine synthesis in rapidly proliferating cells. In this study, we tested the efficacy of teriflunomide to treat two different injury intensities in rat models of TBI. Our results show that teriflunomide restores blood-brain barrier integrity, decreases inflammation, and increases neurogenesis in the subgranular zone of the hippocampus. While we were unable to detect neurocognitive effects of treatment on memory and special learning abilities after treatment, a 2-week treatment following injury was sufficient to reduce neuroinflammation up to 120 days later.

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TI Traumatic Injury Leads to Inflammation and Altered Tryptophan Metabolism

in the Juvenile Rabbit Brain

SO JOURNAL OF NEUROTRAUMA

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DE IDO; inflammation; microglia; serotonin; TBI

ID TUMOR-NECROSIS-FACTOR; CEREBROSPINAL-FLUID; QUINOLINIC ACID;

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AB Neuroinflammation after traumatic brain injury (TBI) contributes to widespread cell death and tissue loss. Here, we evaluated sequential inflammatory response in the brain, as well as inflammation-induced changes in brain tryptophan metabolism over time, in a rabbit pediatric TBI model. On post-natal days 5-7 (P5-P7), New Zealand white rabbit littermates were randomized into three groups: naive (no injury), sham (craniotomy alone), and TBI (controlled cortical impact). Animals were sacrificed at 6h and 1, 3, 7, and 21 days post-injury for evaluating levels of pro- and anti-inflammatory cytokines, as well as the major components in the tryptophan-kynurenine pathway. We found that 1) pro- and anti-inflammatory cytokine levels in the brain injury area were differentially regulated in a time-dependent manner post-injury; 2) indoleamine 2,3 dioxygeenase 1 (IDO1) was upregulated around the injury area in TBI kits that persisted at 21 days post-injury; 3) mean length of serotonin-staining fibers was significantly reduced in the injured brain region in TBI kits for at least 21 days post-injury; and 4) kynurenine level significantly increased at 7 days post-injury. A significant decrease in serotonin/tryptophan ratio and melatonin/tryptophan ratio at 21 days post-injury was noted, suggesting that tryptophan metabolism is altered after TBI. A better understanding of the temporal evolution of immune responses and tryptophan metabolism during injury and repair after TBI is crucial for the development of novel therapeutic strategies targeting these pathways.

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J9 J NEUROTRAUM

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AU Zhang, FC

Dong, HP

Lv, T

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AF Zhang, Fengchen

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TI Moderate hypothermia inhibits microglia activation after traumatic brain

injury by modulating autophagy/apoptosis and the MyD88-dependent TLR4

signaling pathway

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Apoptosis; Autophagy; Microglial activation; Toll-like receptor;

Traumatic brain injury

ID TOLL-LIKE-RECEPTORS; NF-KAPPA-B; CENTRAL-NERVOUS-SYSTEM; INFLAMMATORY

FACTOR-I; INDUCED CELL-DEATH; AUTOPHAGY PATHWAY; RATS; EXPRESSION;

DAMAGE; TRANSDUCTION

AB Background: Complex mechanisms participate in microglial activation after a traumatic brain injury (TBI). TBI can induce autophagy and apoptosis in neurons and glial cells, and moderate hypothermia plays a protective role in the acute phase of TBI. In the present study, we evaluated the effect of TBI and moderate hypothermia on microglial activation and investigated the possible roles of autophagy/apoptosis and toll-like receptor 4 (TLR4).

Methods: The TBI model was induced with a fluid percussion TBI device. Moderate hypothermia was achieved under general anesthesia by partial immersion in a water bath for 4 h. All rats were killed 24 h after the TBI.

Results: Our results showed downregulation of the microglial activation and autophagy, but upregulation of microglial apoptosis, upon post-TBI hypothermia treatment. The expression of TLR4 and downstream myeloid differentiation primary response 88 (MyD88) was attenuated. Moderate hypothermia reduced neural cell death post-TBI.

Conclusions: Moderate hypothermia can reduce the number of activated microglia by inhibiting autophagy and promoting apoptosis, probably through a negative modulation between autophagy and apoptosis. Moderate hypothermia may attenuate the pro-inflammatory function of microglia by inhibiting the MyD88-dependent TLR4 signaling pathway.

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TI The Importance of Inter-Species Variation in Traumatic Brain

Injury-Induced Alterations of Microglial-Axonal Interactions

SO FRONTIERS IN NEUROLOGY

LA English

DT Article

DE traumatic brain injury; microglia; axonal injury; microglial-neuronal

interaction; rat; micro pig; species differences

ID INTRACRANIAL-PRESSURE; INFLAMMATION; ISOFLURANE; CELLS; ACTIVATION;

RECEPTORS; NEURONS; FLUID; TIME

AB Interactions between microglia and neuronal components are important for normal CNS function. They are also associated with neuroinflammation and many pathological processes and several studies have explored these interactions in terms of phagocytic engulfment. Much progress has also been made in understanding the consequences of chronic neuroinflammatory changes following trauma. However, little is known about acute alterations to these physical non-phagocytic microglial-neuronal interactions following traumatic brain injury (TBI), and particularly to what degree these post-injury interactions may be influenced by the animal species utilized in pre-clinical models of TBI. To investigate these problems, we evaluated the physical interactions between microglia and injured axons acutely (6 h and 1 day) following central fluid percussion injury (cFPI) in both rats and micro pigs. The physical interactions between lba-1+ microglia and either normal MBP+myelinated fibers or APP+ injured axonal swellings in the thalamus were assessed following injury or sham via quantitative image analysis of 3D confocal micrographs. The results indicated that the physical interactions between microglia and injured axonal swellings decreased by nearly half in rats 6 h following cFPI but was consistent with sham control at 1 day post-cFPI. This reduction was also observed in non-injured intact fibers at both timepoints following TBI in the rat. Microglial process interactions with injured axons in the micro pig, however, increased nearly 2-fold compared to interactions with intact axonal segments 1 day post-cFPI. This study shows that the species utilized for in vivo pre-clinical studies influences the manner in which microglial-axonal interactions change following TBI. These species differences can be leveraged to further our understanding of the mechanisms involved in microglial process convergence and how these neuro-immune interactions alter the progression of axonal injury following TBI.

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TI Comparison of the detrimental features of microglia and infiltrated

macrophages in traumatic brain injury: A study using a hypnotic

bromovalerylurea

SO GLIA

LA English

DT Article

DE CCL2; FACS; glycolysis; mitochondria; oxidative stress; ROS

ID NITRIC-OXIDE SYNTHASE; NG2 PROTEOGLYCAN; OXIDATIVE STRESS; NEURONAL

DEATH; CELL-DEATH; STROKE; DAMAGE; NEUROPROTECTION; EXPRESSION;

CONTRIBUTE

AB Microglia and blood-borne macrophages in injured or diseased brains are difficult to distinguish because they share many common characteristics. However, the identification of microglia-specific markers and the use of flow cytometry have recently made it easy to discriminate these types of cells. In this study, we analyzed the features of blood-borne macrophages, and activated and resting microglia in a rat traumatic brain injury (TBI) model. Oxidative injury was indicated in macrophages and neurons in TBI lesions by the presence of 8-hydroxy-2'-deoxyguanosine (8-OHdG). Generation of mitochondrial reactive oxygen species (ROS) was markedly observed in granulocytes and macrophages, but not in activated or resting microglia. Dihydroethidium staining supported microglia not being the major source of ROS in TBI lesions. Furthermore, macrophages expressed NADPH oxidase 2, interleukin-1 beta (IL-1 beta), and CD68 at higher levels than microglia. In contrast, microglia expressed transforming growth factor beta 1 (TGF beta 1), interleukin-6 (IL-6), and tumor necrosis factor alpha at higher levels than macrophages. A hypnotic, bromovalerylurea (BU), which has anti-inflammatory effects, reduced both glycolysis and mitochondrial oxygen consumption. BU administration inhibited chemokine CCL2 expression, accumulation of monocytes/macrophages, 8-OHdG generation, mitochondrial ROS generation, and proinflammatory cytokine expression, and markedly ameliorated the outcome of the TBI model. Yet, BU did not inhibit microglial activation or expression of TGF beta 1 and insulin-like growth factor 1 (IGF-1). These results indicate that macrophages are the major aggravating cell type in TBI lesions, in particular during the acute phase. Activated microglia may even play favorable roles. Reduction of cellular energy metabolism in macrophages and suppression of CCL2 expression in injured tissue may lead to amelioration of TBI.

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FX Ministry of Education; Ehime University; Ehime University

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TI Depression following a traumatic brain injury: uncovering cytokine

dysregulation as a pathogenic mechanism

SO NEURAL REGENERATION RESEARCH

LA English

DT Review

DE concussion; major-depressive disorder; chronic traumatic encephalopathy;

inflammation; tumor necrosis factor alpha; interleukin 1; microglia;

astrocytes; synaptic physiology; N-methyl-D-aspartic acid

ID TUMOR-NECROSIS-FACTOR; SPINAL-CORD-INJURY; HOMEOSTATIC SYNAPTIC

PLASTICITY; RECEPTOR ACCESSORY PROTEIN; LONG-TERM POTENTIATION; GLIAL

TNF-ALPHA; MOUSE MODEL; CEREBROSPINAL-FLUID; ALZHEIMERS-DISEASE;

MULTIPLE-SCLEROSIS

AB A substantial number of individuals have long-lasting adverse effects from a traumatic brain injury (TBI). Depression is one of these long-term complications that influences many aspects of life. Depression can limit the ability to return to work, and even worsen cognitive function and contribute to dementia. The mechanistic cause for the increased depression risk associated with a TBI remains to be defined. As TBI results in chronic neuroinflammation, and priming of glia to a secondary challenge, the inflammatory theory of depression provides a promising framework for investigating the cause of depression following a TBI. Increases in cytokines similar to those seen in depression in the general population are also increased following a TBI. Biomarker levels of cytokines peak within hours-to-days after the injury, yet pro-inflammatory cytokines may still be elevated above physiological levels months-to-years following TBI, which is the time frame in which post-TBI depression can persist. As tumor necrosis factor a and interleukin 1 can signal directly at the neuronal synapse, pathophysiological levels of these cytokines can detrimentally alter neuronal synaptic physiology. The purpose of this review is to outline the current evidence for the inflammatory hypothesis of depression specifically as it relates to depression following a TBI. Moreover, we will illustrate the potential synaptic mechanisms by which tumor necrosis factor a and interleukin 1 could contribute to depression. The association of inflammation with the development of depression is compelling; however, in the context of post-TBI depression, the role of inflammation is understudied. This review attempts to highlight the need to understand and treat the psychological complications of a TBI, potentially by neuroimmune modulation, as the neuropsychiatric disabilities can have a great impact on the rehabilitation from the injury, and overall quality of life.

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Ruan, Xiangcai

TI Administration of Dexmedetomidine inhibited NLRP3 inflammasome and

microglial cell activities in hippocampus of traumatic brain injury rats

SO BIOSCIENCE REPORTS

LA English

DT Article

ID INNATE IMMUNE-RESPONSE; ACTIVATION; EXPRESSION; ISCHEMIA; STRESS

AB The abnormally high nucleotide-binding oligomerization domain (NOD)-like receptor family pyrin domain containing 3 (NLRP3) inflammasome activity is a typical characteristic of traumatic brain injury (TBI). Dexmedetomidine (Dex) is a highly selective alpha-2 adrenergic receptor agonist that inhibits the activation of NLRP3. Thus, it was hypothesized that Dex could attenuate TBI by inhibiting NLRP3 inflammasome activity in hippocampus. Rats were subjected to controlled cortical impact method to induce TBI, and treated with Dex. The effect of Dex treatment on the cognitive function, NLRP3 activity, and microglial activation in rat brain tissues was assessed. The administration of Dex improved performance of TBI rats in Morris water maze (MWM) test, which was associated with the increased neurone viability and suppressed microglia activity. Moreover, the administration of Dex inhibited the neuroinflammation in brain tissue as well as the expressions of NLRP3 and caspase-1. Additionally, Dex and NLRP3 inhibitor, BAY-11-7082 had a synergistic effect in inhibiting NLRP3/caspase-1 axis activity and improving TBI. The findings outlined in the current study indicated that the improvement effect of Dex on TBI was related to its effect on NLRP3 activity.

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TI Immunohistochemical Evaluation of Aquaporin-4 and its Correlation with

CD68, IBA-1, HIF-1, GFAP, and CD15 Expressions in Fatal Traumatic Brain

Injury

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE traumatic brain injury; aquaporin; genetic analysis; edema; computed

tomography (CT) scan; hypoxia; microglia activation

ID REVEALS COMPLEX; GROWTH-FACTOR; EDEMA; WATER; ASTROCYTES; FLUID;

PATHOPHYSIOLOGY; EPIDEMIOLOGY; MECHANISMS; HIF1-ALPHA

AB Traumatic brain injury (TBI) is one of the leading causes of death and disability worldwide. Our understanding of its pathobiology has substantially increased. Following TBI, the following occur, edema formation, brain swelling, increased intracranial pressure, changes in cerebral blood flow, hypoxia, neuroinflammation, oxidative stress, excitotoxicity, and apoptosis. Experimental animal models have been developed. However, the difficulty in mimicking human TBI explains why few neuroprotective strategies, drawn up on the basis of experimental studies, have translated into improved therapeutic strategies for TBI patients. In this study, we retrospectively examined brain samples in 145 cases of death after different survival times following TBI, to investigate aquaporin-4 (AQP4) expression and correlation with hypoxia, and neuroinflammation in human TBI. Antibodies anti-glial fibrillary acid protein (GFAP), aquaporin-4 (AQP4), hypoxia induced factor-1 (HIF-1), macrophage/phagocytic activation (CD68), ionized calcium-binding adapter molecule-1 (IBA-1), and neutrophils (CD15) were used. AQP4 showed a significant, progressive increase between the control group and groups 2 (one-day survival) and 3 (three-day survival). There were further increases in AQP4 immunopositivity in groups 4 (seven-day survival), 5 (14-dayssurvival), and 6 (30-day survival), suggesting an upregulation of AQP4 at 7 to 30 days compared to group 1. GFAP showed its highest expression in non-acute cases at the astrocytic level compared with the acute TBI group. Data emerging from the HIF-1 reaction showed a progressive, significant increase. Immunohistochemistry with IBA-1 revealed activated microglia starting three days after trauma and progressively increasing in the next 15 to 20 days after the initial trauma. CD68 expression demonstrated basal macrophage and phagocytic activation mostly around blood vessels. Starting from one to three days of survival after TBI, an increase in the number of CD68 cells was progressively observed; at 15 and 30 days of survival, CD68 showed the most abundant immunopositivity inside or around the areas of necrosis. These findings need to be developed further to gain insight into the mechanisms through which brain AQP4 is upregulated. This could be of the utmost clinicopathological importance.

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TI The Synthetic Steroid Tibolone Decreases Reactive Gliosis and Neuronal

Death in the Cerebral Cortex of Female Mice After a Stab Wound Injury

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article

DE Astrocytes; Microglia; Neuroinflammation; Neuroprotection; Steroid

receptors; Brain trauma

ID TRAUMATIC BRAIN-INJURY; CORTICAL PERICONTUSIONAL ZONE; RECEPTOR

MODULATORS DECREASE; PROGRAMMED CELL-DEATH; ESTROGEN-RECEPTOR;

MICROGLIAL ACTIVATION; ANDROGEN RECEPTORS; OXIDATIVE STRESS;

UP-REGULATION; ESTRADIOL

AB Previous studies have shown that estradiol reduces reactive gliosis after a stab wound injury in the cerebral cortex. Since the therapeutic use of estradiol is limited by its peripheral hormonal effects, it is of interest to determine whether synthetic estrogenic compounds with tissue-specific actions regulate reactive gliosis. Tibolone is a synthetic steroid that is widely used for the treatment of climacteric symptoms and/or the prevention of osteoporosis. In this study, we have assessed the effect of tibolone on reactive gliosis in the cerebral cortex after a stab wound brain injury in ovariectomized adult female mice. By 7days after brain injury, tibolone reduced the number of glial fibrillary acidic protein (GFAP) immunoreactive astrocytes, the number of ionized calcium binding adaptor molecule 1 (Iba1) immunoreactive microglia, and the number of microglial cells with a reactive phenotype in comparison to vehicle-injected animals. These effects on gliosis were associated with a reduction in neuronal loss in the proximity to the wound, suggesting that tibolone exerts beneficial homeostatic actions in the cerebral cortex after an acute brain injury.

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TI Early Microglial Activation Following Closed-Head Concussive Injury Is

Dominated by Pro-Inflammatory M-1 Type

SO FRONTIERS IN NEUROLOGY

LA English

DT Article

DE microglia; inflammation; polarization; concussion; traumatic brain

injury

ID TRAUMATIC BRAIN-INJURY; WHITE-MATTER; NEURODEGENERATION; FOOTBALL;

STATES; MODEL; MICE; NEUROINFLAMMATION; NEUROPROTECTION; ENCEPHALOPATHY

AB Microglial activation is a pathological hallmark of traumatic brain injury (TBI) Following brain injury, activated microglia/macrophages adopt different phenotypes, generally categorized as M-1, or classically activated, and M-2, or alternatively activated. While the M-1, or pro-inflammatory phenotype is detrimental to recovery, M-2, or the anti-inflammatory phenotype, aids in brain repair. Recent findings also suggest the existence of mixed phenotype following brain injury, where activated microglia simultaneously express both M-1 and M-2 markers. The present study sought to determine microglial activation states at early time points (6-72h) following single or repeated concussive injury in rats. Closed-head concussive injury was modeled in rats using projectile concussive impact injury, with either single or repeated impacts (4 impacts, 1 h apart). Brain samples were examined using immunohistochemical staining, inflammatory gene profiling and real-time polymerase chain reaction analyses to detect concussive injury induced changes in microglial activation and phenotype in cortex and hippocampal regions. Our findings demonstrate robust microglial activation following concussive brain injury. Moreover, we show that multiple concussions induced a unique rod-shaped microglial morphology that was also observed in other diffuse brain injury models. Histological studies revealed a predominance of MHC-Il positive M-1 phenotype in the post-concussive microglial milieu following multiple impacts. Although there was simultaneous expression of M-1 and M-2 markers, gene expression results indicate a clear dominance in M-1 pro-inflammatory markers following both single and repeated concussions. While the increase in M-1 markers quickly resolved after a single concussion, they persisted following repeated concussions, indicating a pro-inflammatory environment induced by multiple concussions that may delay recovery and contribute to long-lasting consequences of concussion.

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TI Sex Differences in Acute Neuroinflammation after Experimental Traumatic

Brain Injury Are Mediated by Infiltrating Myeloid Cells

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE macrophage; microglia; neuroinflammation; sex differences; traumatic

brain injury

ID GENDER-DIFFERENCE; INFARCT VOLUME; HEAD-INJURY; RAT MODEL; MILD;

NEURODEGENERATION; PREDICTORS; MICROGLIA; RECOVERY; MODERATE

AB The inflammatory response to moderate-severe controlled cortical impact (CCI) in adult male mice has been shown to exhibit greater glial activation compared with age-matched female mice. However, the relative contributions of resident microglia and infiltrating peripheral myeloid cells to this sexually dimorphic neuroinflammatory responses remains unclear. Here, 12-week-old male and female C57Bl/6 mice were subjected to sham or CCI, and brain samples were collected at 1, 3, or 7 days post-injury for flow cytometry analysis of cytokines, reactive oxygen species (ROS), and phagocytosis in resident microglia (CD45(int)CD11b+) versus infiltrating myeloid cells (CD45(hi)CD11b+). Motor (rotarod, cylinder test), affect (open field), and cognitive (Y-maze) function tests also were performed. We demonstrate that male microglia had increased phagocytic activity and higher ROS levels in the non-injured brain, whereas female microglia had increased production of tumor necrosis factor (TNF) alpha and interleukin (IL)-1 beta. Following CCI, males showed a significant influx of peripheral myeloid cells by 1 day post-injury followed by proliferation of resident microglia at 3 days. In contrast, myeloid infiltration and microglial activation responses in female CCI mice were significantly reduced. No sex differences were observed for TNF alpha, IL-1 beta, transforming growth factor beta, NOX2, ROS production, or phagocytic activity in resident microglia or infiltrating cells at any time. However, across these functions, infiltrating myeloid cells were significantly more reactive than resident microglia. Female CCI mice also had improved motor function at 1 day post-injury compared with male mice. Thus, we conclude that sexually dimorphic responses to moderate-severe CCI result from the rapid activation and infiltration of pro-inflammatory myeloid cells to brain in male, but not female, mice.

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TI Traumatic brain injury-induced neuronal damage in the somatosensory

cortex causes formation of rod-shaped microglia that promote

astrogliosis and persistent neuroinflammation

SO GLIA

LA English

DT Article

DE astrocytes; CSF1R antagonist; fluid percussion injury; microglia;

neuroinflammation; traumatic brain injury

ID PSYCHIATRIC-DISORDERS; AXONAL INJURY; CELL; ACTIVATION; AXOTOMY;

PROLIFERATION; INFLAMMATION; REACTIVITY; DEFICITS; ATROPHY

AB Microglia undergo dynamic structural and transcriptional changes during the immune response to traumatic brain injury (TBI). For example, TBI causes microglia to form rod-shaped trains in the cerebral cortex, but their contribution to inflammation and pathophysiology is unclear. The purpose of this study was to determine the origin and alignment of rod microglia and to determine the role of microglia in propagating persistent cortical inflammation. Here, diffuse TBI in mice was modeled by midline fluid percussion injury (FPI). Bone marrow chimerism and BrdU pulse-chase experiments revealed that rod microglia derived from resident microglia with limited proliferation. Novel data also show that TBI-induced rod microglia were proximal to axotomized neurons, spatially overlapped with dense astrogliosis, and aligned with apical pyramidal dendrites. Furthermore, rod microglia formed adjacent to hypertrophied microglia, which clustered among layer V pyramidal neurons. To better understand the contribution of microglia to cortical inflammation and injury, microglia were eliminated prior to TBI by CSF1R antagonism (PLX5622). Microglial elimination did not affect cortical neuron axotomy induced by TBI, but attenuated rod microglial formation and astrogliosis. Analysis of 262 immune genes revealed that TBI caused profound cortical inflammation acutely (8 hr) that progressed in nature and complexity by 7 dpi. For instance, gene expression related to complement, phagocytosis, toll-like receptor signaling, and interferon response were increased 7 dpi. Critically, these acute and chronic inflammatory responses were prevented by microglial elimination. Taken together, TBI-induced neuronal injury causes microglia to structurally associate with neurons, augment astrogliosis, and propagate diverse and persistent inflammatory/immune signaling pathways.

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for Brain and Spinal Cord Repair; College of Medicine Dean's Discovery;

NINDS, Grant/Award Number: R56-NS-090311

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TI Neutralization of Interleukin-1β following Diffuse Traumatic Brain

Injury in the Mouse Attenuates the Loss of Mature Oligodendrocytes

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE fluid percussion injury; inflammation; IL-1 beta; microglia;

oligodendrocyte; oligodendrocyte progenitor cells; Olig2; traumatic

brain injury

ID FLUID PERCUSSION INJURY; MICROGLIAL ACTIVATION; INFLAMMATORY RESPONSE;

KINASE-ACTIVITY; AXONAL INJURY; DEATH; EXPRESSION; IL-1-BETA; PATHOLOGY;

CELLS

AB Traumatic brain injury (TBI) commonly results in injury to the components of the white matter tracts, causing post-injury cognitive deficits. The myelin-producing oligodendrocytes (OLs) are vulnerable to TBI, although may potentially be replaced by proliferating oligodendrocyte progenitor cells (OPCs). The cytokine interleukin-1 beta (IL-1 beta) is a key mediator of the complex inflammatory response, and when neutralized in experimental TBI, behavioral outcome was improved. To evaluate the role of IL-1 beta on oligodendrocyte cell death and OPC proliferation, 116 adult male mice subjected to sham injury or the central fluid percussion injury (cFPI) model of traumatic axonal injury, were analyzed at two, seven, and 14 days post-injury. At 30 min post-injury, mice were randomly administered an IL-1 beta neutralizing or a control antibody. OPC proliferation (5-ethynyl 2 '- deoxyuridine (EdU)/Olig2 co-labeling) and mature oligodendrocyte cell loss was evaluated in injured white matter tracts. Microglia/macrophages immunohistochemistry and ramification using Sholl analysis were also evaluated. Neutralizing IL-1 beta resulted in attenuated cell death, indicated by cleaved caspase-3 expression, and attenuated loss of mature OLs from two to seven days post-injury in brain-injured animals. IL-1 beta neutralization also attenuated the early, two day post-injury increase of microglia/macrophage immunoreactivity and altered their ramification. The proliferation of OPCs in brain-injured animals was not altered, however. Our data suggest that IL-1 beta is involved in the TBI-induced loss of OLs and early microglia/macrophage activation, although not the OPC proliferation. Attenuated oligodendrocyte cell loss may contribute to the improved behavioral outcome observed by IL-1 beta neutralization in this mouse model of diffuse TBI.

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TI Transient disruption of mouse home cage activities and assessment of

orexin immunoreactivity following concussive- or blast-induced brain

injury

SO BRAIN RESEARCH

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DT Article

DE Traumatic brain injury; Concussion; Blast brain injury; Neurotrauma;

Activity; Cognition; Orexin; Microglia; Mouse

ID SLEEP-WAKE DISTURBANCES; ANXIETY-LIKE BEHAVIOR; HEAD-INJURY; OPEN-FIELD;

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HYPOCRETIN

AB The employment of explosive weaponry in modern warfare exposes populations to shock wave-induced and impact-related brain injuries. Among the most common clinical complaints resulting from traumatic brain injury (TBI) are sleep-wake disturbances. The current study assessed the acute effects of mild concussive brain injury (CBI) and mild blast wave-induced brain injury (BTBI) on mouse behavior and orexin-A expression. Male C57BL/6J mice were exposed to CBI, BTBI, or sham procedures. Injured animals and their shams were further divided into the following subgroups: 24-h survival in standard group (SG) housing, 72-h survival in SG housing, and 72-h survival in Any-Maze cages (AMc). AMc enabled continuous monitoring of home cage activities. BTBI caused significant but transient decreases in wheel running and ingestive behaviors 24 h post-injury (PI), while CBI transiently decreased running and water intake. BTBI resulted in general hypoactivity in the open field (OF) at both PI time points for SG-housed animals. In contrast, CBI did not cause hypoactivity. Mice subjected to CBI traveled more in the center of the OF at both time points PI, suggesting that CBI caused reduced anxiety in mice. Increased activity in the center of the OF was also seen at 24 h PI after BTBI. CBI treatment caused increased CD11b immunostaining. However, neither injury was accompanied by an alteration in the number of orexin-A hypothalamic neurons. Taken together, shock wave exposure and concussive injury transiently reduced mouse activities, but some differences between the two injuries were seen.

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Activation, Synapse Loss, and Complement-Dependent Memory Deficits

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DT Article

DE traumatic brain injury; complement; C1q; microglia; synapse

ID COGNITIVE FUNCTION; CNS; DYSFUNCTION; INHIBITION; DRIVES; IMPACT

AB Traumatic brain injury (TBI) is of particular concern for the aging community since there is both increased incidence of TBI and decreased functional recovery in this population. In addition, TBI is the strongest environmental risk factor for development of Alzheimer's disease and other dementia-related neurodegenerative disorders. Critical changes that affect cognition take place over time following the initial insult. Our previous work identified immune system activation as a key contributor to cognitive deficits observed in aged animals. Using a focal contusion model in the current study, we demonstrate a brain lesion and cavitation formation, as well as prolonged blood-brain barrier breakdown. These changes were associated with a prolonged inflammatory response, characterized by increased microglial cell number and phagocytic activity 30 days post injury, corresponding to significant memory deficits. We next aimed to identify the injury-induced cellular and molecular changes that lead to chronic cognitive deficits in aged animals, and measured increases in complement initiation components C1q, C3, and CR3, which are known to regulate microglial-synapse interactions. Specifically, we found significant accumulation of C1q on synapses within the hippocampus, which was paralleled by synapse loss 30 days post injury. We used genetic and pharmacological approaches to determine the mechanistic role of complement initiation on cognitive loss in aging animals after TBI. Notably, both genetic and pharmacological blockade of the complement pathway prevented memory deficits in aged injured animals. Thus, therapeutically targeting early components of the complement cascade represents a significant avenue for possible clinical intervention following TBI in the aging population.

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TI Microglial Calcium Release-Activated Calcium Channel Inhibition Improves

Outcome from Experimental Traumatic Brain Injury and Microglia-Induced

Neuronal Death

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE calcium release-activated calcium channel; microglia; store-operated

calcium entry; traumatic brain injury

ID HEAT-SHOCK-PROTEIN; CYCLOSPORINE-A; MILD HYPOTHERMIA; CALCINEURIN

INHIBITORS; NEUROPROTECTIVE ACTION; MOLECULAR-MECHANISMS; EXPERIMENTAL

STROKE; CEREBRAL-ISCHEMIA; TACROLIMUS FK506; DOWN-REGULATION

AB Store-operated Ca2+ entry (SOCE) mediated by calcium release-activated calcium (CRAC) channels contributes to calcium signaling. The resulting intracellular calcium increases activate calcineurin, which in turn activates immune transcription factor nuclear factor of activated T cells (NFAT). Microglia contain CRAC channels, but little is known whether these channels play a role in acute brain insults. We studied a novel CRAC channel inhibitor to explore the therapeutic potential of this compound in microglia-mediated injury. Cultured microglial BV2 cells were activated by Toll-like receptor agonists or IFN gamma. Some cultures were treated with a novel CRAC channel inhibitor (CM-EX-137). Western blots revealed the presence of CRAC channel proteins STIM1 and Orai1 in BV2 cells. CM-EX-137 decreased nitric oxide (NO) release and inducible nitric oxide synthase (iNOS) expression in activated microglia and reduced agonist-induced intracellular calcium accumulation in microglia, while suppressing inflammatory transcription factors nuclear factor kappa B (NF-kappa B) and nuclear factor of activated T cells (NFAT). Male C57/BL6 mice exposed to experimental brain trauma and treated with CM-EX-137 had decreased lesion size, brain hemorrhage, and improved neurological deficits with decreased microglial activation, iNOS and Orai1 and STIM1 levels. We suggest a novel anti-inflammatory approach for managing acute brain injury. Our observations also shed light on new calcium signaling pathways not described previously in brain injury models.

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TI A mathematical model of neuroinflammation in severe clinical traumatic

brain injury

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DT Article

DE Biomarker; Cerebrospinal fluid; Cytokines; Glasgow outcome scale;

Inflammation; Mathematical modeling; Microglia; Patient outcome;

Traumatic brain injury

ID CEREBROSPINAL-FLUID; MICROGLIAL POLARIZATION; INFLAMMATORY RESPONSE; M2

MICROGLIA; ACTIVATION; SURVIVORS; PROFILES; DISEASE; IL-4

AB BackgroundUnderstanding the interdependencies among inflammatory mediators of tissue damage following traumatic brain injury (TBI) is essential in providing effective, patient-specific care. Activated microglia and elevated concentrations of inflammatory signaling molecules reflect the complex cascades associated with acute neuroinflammation and are predictive of recovery after TBI. However, clinical TBI studies to date have not focused on modeling the dynamic temporal patterns of simultaneously evolving inflammatory mediators, which has potential in guiding the design of future immunomodulation intervention studies.MethodsWe derived a mathematical model consisting of ordinary differential equations (ODE) to represent interactions between pro- and anti-inflammatory cytokines, M1- and M2-like microglia, and central nervous system (CNS) tissue damage. We incorporated variables for several cytokines, interleukin (IL)-1, IL-4, IL-10, and IL-12, known to have roles in microglial activation and phenotype differentiation. The model was fit to cerebrospinal fluid (CSF) cytokine data, collected during the first 5days post-injury in n=89 adults with severe TBI. Ensembles of model fits were produced for three patient subgroups: (1) a favorable outcome group (GOS=4,5) and (2) an unfavorable outcome group (GOS=1,2,3) both with lower pro-inflammatory load, and (3) an unfavorable outcome group (GOS=1,2,3) with higher pro-inflammatory load. Differences in parameter distributions between subgroups were ranked using Bhattacharyya metrics to identify mechanistic differences underlying the neuroinflammatory patterns of patient groups with different TBI outcomes.ResultsOptimal model fits to data showed different microglial and damage responses by patient subgroup. Upon comparison of model parameter distributions, unfavorable outcome groups were characterized by either a prolonged, pathophysiological or a transient, sub-physiological course of neuroinflammation.ConclusionBy developing a mathematical characterization of inflammatory processes informed by clinical data, we have created a system for exploring links between acute neuroinflammatory components and patient outcome in severe TBI.

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TI Activation of both transforming growth factor-β and bone morphogenetic

protein signalling pathways upon traumatic brain injury restrains

pro-inflammatory and boosts tissue reparatory responses of reactive

astrocytes and microglia

SO BRAIN COMMUNICATIONS

LA English

DT Article

DE astrocytes; BMP; microglia; TGF beta; traumatic brain injury

ID TGF-BETA; MACROPHAGE PLASTICITY; SCAR FORMATION; RAT-BRAIN; EXPRESSION;

TIME; NEURONS; SYSTEM; CELLS; ROLES

AB Various ligands and receptors of the transforming growth factor-beta superfamily have been found upregulated following traumatic brain injury; however, the role of this signalling system in brain injury pathophysiology is not fully characterized. To address this, we utilized an acute stab wound brain injury model to demonstrate that hallmarks of transforming growth factor-beta superfamily system activation, such as levels of phosphorylated Smads, ligands and target genes for both transforming growth factor-beta and bone morphogenetic protein pathways, were upregulated within injured tissues. Using a bone morphogenetic protein-responsive reporter mouse model, we showed that activation of the bone morphogenetic protein signalling pathway involves primarily astrocytes that demarcate the wound area. Insights regarding the potential role of transforming growth factor-beta superfamily activation in glia cells within the injured tissues were obtained indirectly by treating purified reactive astrocytes and microglia with bone morphogenetic protein-4 or transforming growth factor-beta 1 and characterizing changes in their transcriptional profiles. Astrocytes responded to both ligands with considerably overlapping profiles, whereas, microglia responded selectively to transforming growth factor-beta 1. Novel pathways, crucial for repair of tissue-injury and blood-brain barrier, such as activation of cholesterol biosynthesis and transport, production of axonal guidance and extracellular matrix components were upregulated by transforming growth factor-beta 1 and/or bone morphogenetic protein-4 in astrocytes. Moreover, both ligands in astrocytes and transforming growth factor-beta 1 in microglia shifted the phenotype of reactive glia cells towards the anti-inflammatory and tissue reparatory 'A2'-like and 'M0/M2'-like phenotypes, respectively. Increased expression of selected key components of the in vitro modulated pathways and markers of 'A2'-like astrocytes was confirmed within the wound area, suggesting that these processes could also be modulated in situ by the integrated action of transforming growth factor-beta and/or bone morphogenetic protein-mediated signalling. Collectively, our study provides a comprehensive comparative analysis of transforming growth factor-beta superfamily signalling in reactive astrocytes and microglia and points towards a crucial role of both transforming growth factor-beta and bone morphogenetic protein pathways in modulating the inflammatory and brain injury reparatory functions of activated glia cells.

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TI Inhibition of miR-155 Limits Neuroinflammation and Improves Functional

Recovery After Experimental Traumatic Brain Injury in Mice

SO NEUROTHERAPEUTICS

LA English

DT Article

DE Traumatic brain injury; miR-155; microglial activation;

neuroinflammation; neuroprotection

ID PROGRESSIVE NEURODEGENERATION; MICRORNA-155; ACTIVATION; EXPRESSION;

MICROGLIA; POLARIZATION; HIPPOCAMPUS; CYTOKINE; IMMUNITY; MODELS

AB Micro-RNAs (miRs) are short, noncoding RNAs that negatively regulate gene expression at the post-transcriptional level and have been implicated in the pathophysiology of secondary damage after traumatic brain injury (TBI). Among miRs linked to inflammation, miR-155 has been implicated as a pro-inflammatory factor in a variety of organ systems. We examined the expression profile of miR-155, following experimental TBI (controlled cortical impact) in adult male C57Bl/6 mice, as well as the effects of acute or delayed administration of a miR-155 antagomir on post-traumatic neuroinflammatory responses and neurological recovery. Trauma robustly increased miR-155 expression in the injured cortex over 7days. Similar TBI-induced miR-155 expression changes were also found in microglia/macrophages isolated from the injured cortex at 7days post-injury. A miR-155 hairpin inhibitor (antagomir; 0.5nmol), administered intracerebroventricularly (ICV) immediately after injury, attenuated neuroinflammatory markers at both 1day and 7days post-injury and reduced impairments in spatial working memory. Delayed ICV infusion of the miR-155 antagomir (0.5nmol/day), beginning 24h post-injury and continuing for 6days, attenuated neuroinflammatory markers at 7days post-injury and improved motor, but not cognitive, function through 28days. The latter treatment limited NADPH oxidase 2 expression changes in microglia/macrophages in the injured cortex and reduced cortical lesion volume. In summary, TBI causes a robust and persistent neuroinflammatory response that is associated with increased miR-155 expression in microglia/macrophages, and miR-155 inhibition reduces post-traumatic neuroinflammatory responses and improves neurological recovery. Thus, miR-155 may be a therapeutic target for TBI-related neuroinflammation.

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TC 51

Z9 58

U1 1

U2 11

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JI Neurotherapeutics

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Zhang, Lin

TI VEGF-C Induces Alternative Activation of Microglia to Promote Recovery

from Traumatic Brain Injury

SO JOURNAL OF ALZHEIMERS DISEASE

LA English

DT Article

DE Microglia; polarization; traumatic brain injury; VEGF-C; VEGFR3

ID MACROPHAGE; LYMPHANGIOGENESIS; INFLAMMATION; EXPRESSION; GENE

AB Traumatic brain injury (TBI), a brain disorder that causes death and long-term disability in humans, is increasing in prevalence, though there is a lack of protective or therapeutic strategies for mitigating the damage after TBI and for preserving neurological functionality. Microglia cells play a key role in neuroinflammation following TBI, but their regulation and polarization by a member of the vascular endothelial growth factor (VEGF) family, VEGF-C, is unknown. Here, we show that VEGF-C induced M2 polarization in a murine microglia cell line, BV-2, in vitro, by a mechanism that required signaling from its unique receptor, VEGF receptor 3 (VEGFR3). Moreover, in a TBI model in rats, VEGF-C administration induced M2 polarization of microglia cells, significantly improved motor deficits after experimental TBI, and significantly improved neurological function following TBI, likely through a reduction in cell apoptosis. Together, our data reveal a previously unknown role of VEGF-C/VEGFR3 signaling in the regulation of post-TBI microglia cell polarization, which appears to be crucial for recovery from TBI.

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AU Ni, HQ

Yang, S

Siaw-Debrah, F

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TI Exosomes Derived From Bone Mesenchymal Stem Cells Ameliorate Early

Inflammatory Responses Following Traumatic Brain Injury

SO FRONTIERS IN NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury; bone mesenchymal stem cells; exosomes;

neuroprotection; microglia/macrophage; inflammation

ID STROMAL CELLS; EXTRACELLULAR VESICLES; FUNCTIONAL RECOVERY;

ANIMAL-MODELS; TRANSPLANTATION; REGULATORS; THERAPY

AB Traumatic brain injury (TBI) is a leading cause of mortality and disability worldwide. Although treatment guidelines have been developed, no best treatment option or medicine for this condition exists. Recently, mesenchymal stem cells (MSCs)-derived exosomes have shown lots of promise for the treatment of brain disorders, with some results highlighting the neuroprotective effects through neurogenesis and angiogenesis after TBI. However, studies focusing on the role of exosomes in the early stages of neuroinflammation post-TBI are not sufficient. In this study, we investigated the role of bone mesenchymal stem cells (BMSCs)-exosomes in attenuating neuroinflammation at an early stage post-TBI and explored the potential regulatory neuroprotective mechanism. We administered 30 vg protein of BMSCsexosomes or an equal volume of phosphate-buffered saline (PBS) via the retro-orbital route into C57BL/6 male mice 15 min after controlled cortical impact (CCI)-induced TBI. The results showed that the administration of BMSCs-exosomes reduced the lesion size and improved the neurobehavioral performance assessed by modified Neurological Severity Score (mNSS) and rotarod test. In addition, BMSCs-exosomes inhibited the expression of proapoptosis protein Bcl-2-associated X protein (BAX) and proinflammation cytokines, tumor necrosis factor-alpha (TNF-alpha) and interleukin (IL)-1 beta, while enhancing the expression of the anti-apoptosis protein B-cell lymphoma 2 (BCL2). Furthermore, BMSCs-exosomes modulated microglia/macrophage polarization by downregulating the expression of inducible nitric oxide synthase (INOS) and upregulating the expression of clusters of differentiation 206 (CD206) and arginase-1 (Arg1). In summary, our result shows that BMSCs-exosomes serve a neuroprotective function by inhibiting early neuroinflammation in TBI mice through modulating the polarization of microglia/macrophages. Further research into this may serve as a potential therapeutic strategy for the future treatment of TBI.

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(2016RCA022), and Zhejiang Key Research and Development Project

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NR 46

TC 134

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U1 4

U2 57

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J9 FRONT NEUROSCI-SWITZ

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AU Lee, SW

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TI The role of microglial inflammasome activation in pyroptotic cell death

following penetrating traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Inflammasome; ASC; Pyroptosis; Microglia; Infiltrating leukocytes;

Traumatic brain injury; Penetrating traumatic brain injury; PTBI;

Penetrating ballistic-brain injury; PBBI

ID CIVILIAN GUNSHOT WOUNDS; GASDERMIN D; RAT MODEL; UNITED-STATES; ASC;

NEUROINFLAMMATION; HEAD; NEUROPROTECTION; INHIBITION; PREDICTORS

AB BackgroundTraumatic brain injury remains a significant cause of death and disability in the USA. Currently, there are no effective therapies to mitigate disability except for surgical interventions necessitating a need for continued research into uncovering novel therapeutic targets. In a recent study, we used a rodent model of penetrating traumatic brain injury known as penetrating ballistic-like brain injury (PBBI) to examine the role of innate immunity in post-traumatic secondary injury mechanisms. We previously reported that the inflammasome, a multiprotein complex composed of apoptosis-associated speck-like protein containing card and caspase-1, plays a role in secondary cell death mechanisms after PBBI, including inflammatory cell death (pyroptosis).MethodsIn the current study, we used flow cytometry analysis to evaluate activated microglia and CD11b-positive leukocytes after PBBI and assessed inflammasome activation and pyroptosis of specific cellular populations. Sprague-Dawley male rats underwent PBBI or sham-operated procedures and ipsilateral cortical regions processed for flow cytometry and cellular analysis. Flow cytometry results were compared using one-way ANOVA followed by Tukey's multiple comparisons.ResultsAt 48h following PBBI, there was an increase in activated microglia and infiltrating leukocytes compared to sham controls that were associated with increased caspase-1 activity. Using a florescent probe to identify caspase-1 activity and a fluorescent assay to determine cell viability, evidence for pyroptosis in CD11b+ cells was also determined. Finally, while post-traumatic treatment with an anti-ASC antibody had no effect on the number of activated microglia and infiltrating leukocytes, antibody treatment decreased caspase-1 activity in both resident microglia and infiltrating leukocytes and reduced pyroptotic CD11b+ cell death.ConclusionsThese results provide evidence for inflammasome activation in microglia and infiltrating leukocytes after penetrating traumatic brain injury and a role for pyroptotic cell death in the pathophysiology. In addition to inhibiting neuronal cell death, therapeutic treatments targeting inflammasome activation may also provide beneficial effects by reducing the potentially detrimental consequences of activated microglia and infiltrating CD11b+ leukocytes following penetrating traumatic brain injury.

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TI Neutral Sphingomyelinase Inhibition Alleviates LPS-Induced Microglia

Activation and Neuroinflammation after Experimental Traumatic Brain

Injury

SO JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS

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DT Article

ID IMPROVES FUNCTIONAL RECOVERY; MAPK SIGNALING PATHWAYS; DENDRITIC CELLS;

DOWN-REGULATION; TNF-ALPHA; INFLAMMATION; RELEASE; DISEASE; DEATH;

NEURODEGENERATION

AB Neuroinflammation is one of the key secondary injury mechanisms triggered by traumatic brain injury (TBI). Microglial activation, a hallmark of brain neuroinflammation, plays a critical role in regulating immune responses after TBI and contributes to progressive neurodegeneration and neurologic deficits following brain trauma. Here we evaluated the role of neutral sphingomyelinase (nSMase) in microglial activation by examining the effects of the nSMase inhibitors altenusin and GW4869 in vitro (using BV2 microglia cells and primary microglia), as well as in a controlled cortical injury (CCI) model in adult male C57BL/6 mice. Pretreatment of altenusin or GW4869 prior to lipopolysaccharide (LPS) stimulation for 4 or 24 hours, significantly downregulated gene expression of the pro-inflammatory mediators TNF-alpha, IL-1 beta, IL-6, iNOS, and CCL2 in microglia and reduced the release of nitric oxide and TNF-alpha. These nSMase inhibitors also attenuated the release of microparticles and phosphorylation of p38 MAPK and ERK1/2. In addition, altenusin pretreatment also reduced the gene expression of multiple inflammatory markers associated with microglial activation after experimental TBI, including TNF-alpha, IL-1 beta, IL-6, iNOS, CCL2, CD68, NOX2, and p22(phox). Overall, our data demonstrate that nSMase inhibitors attenuate multiple inflammatory pathways associated with microglial activation in vitro and after experimental TBI. Thus, nSMase inhibitors may represent promising therapeutics agents targeting neuroinflammation.

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TI MiR-124 Enriched Exosomes Promoted the M2 Polarization of Microglia and

Enhanced Hippocampus Neurogenesis After Traumatic Brain Injury by

Inhibiting TLR4 Pathway

SO NEUROCHEMICAL RESEARCH

LA English

DT Article

DE Traumatic brain injury; Exosome; MiR-124; Microglia; Neurogenesis; TLR4

ID INTERLEUKIN-1 RECEPTOR ANTAGONIST; FUNCTIONAL RECOVERY; ADULT

NEUROGENESIS; CONTRIBUTES; MACROPHAGES; ACTIVATION; PROTECTION;

REGULATOR; ABLATION; DELIVERY

AB MicroRNA-124 (miR-124) is a brain specific miRNA that is highly expressed in microglia. The upregulation of miR-124 contributes to M2 polarization of microglia, which is beneficial to neurogenesis. Exosomes are lipid membrane vesicles that can deliver miR-124 into the brain. However, whether miR-124 enriched exosomes (Exo-miR-124) can regulate the polarization of microglia and affect hippocampus neurogenesis after traumatic brain injury (TBI) is unknown. To clarify this, the Exo-miR-124 was first constructed, and then was intravenously administrated into rats via tail vein with the dose of 3x10(9) particles/each rat at 24h post TBI. The polarization of microglia in hippocampus was evaluated through measuring the signature genes and cytokines of M1/M2 phenotype by reverse transcription-polymerase chain reaction (RT-PCR) and enzyme-linked immune sorbent assay (ELISA) at 7/14/21/28 days after TBI. Hippocampus neurogenesis was evaluated through detecting the proliferation marker BrdU/SOX2 and differentiation marker BrdU/NeuN by immunofluorescence (IF) at 7 and 28days after TBI respectively. Neurological function was evaluated by neurological severity score (NSS) and morris water maze (MWM) at 7/14/21/28 and 24-28days after TBI respectively. To explore the underlying mechanisms, the mRNA expression of TLR4 pathway molecules in hippocampus were measured by RT-PCR, and the polarization of microglia and the activation of TLR4 pathway in BV2 cells were measured after exosome treatment as well. Results demonstrated that Exo-miR-124 treatment promoted the M2 polarization of microglia, enhanced neurogenesis in hippocampus, and improved function recovery after TBI. The M2 polarization effect of Exo-miR-124 was produced through inhibiting TLR4 pathway, which was verified in hippocampus and BV2 microglia. In conclusion, Exo-miR-124 treatment promoted M2 polarization of microglia and improved hippocampal neurogenesis and functional recovery after brain injury, which might be a strategy to improve the outcome of TBI.

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TI Mild Closed-Head Injury in Conscious Rats Causes Transient

Neurobehavioral and Glial Disturbances: A Novel Experimental Model of

Concussion

SO JOURNAL OF NEUROTRAUMA

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DE anesthesia; astrocytes; behavior; microglia; mild traumatic brain

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ID TRAUMATIC BRAIN-INJURY; SPORT-RELATED CONCUSSION; ELEVATED PLUS-MAZE;

ANIMAL-MODELS; ANXIETY; ADULT; NEUROINFLAMMATION; DEPRESSION;

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AB Rodent models can provide insights into the most pertinent issues surrounding concussion. Nonetheless, the relevance of some existing models to clinical concussion can be questioned, particularly with regard to the use of surgery and anesthesia and the mechanism and severity of injury. Accordingly, we have co-developed an awake closed-head injury (ACHI) model in rats. Here, we aimed to create a temporal profile of the neurobehavioral and neuropathological effects of a single ACHI. Adolescent male rats were placed in a restraint bag and a steel helmet was positioned over the head such that the impact target was centered over the left parietal cortex. Once positioned on a foam platform, a cortical impactor was used to strike the helmet. Sham animals underwent the same procedure without impact. When compared with sham rats, those given a single ACHI displayed evidence of sensorimotor deficits and reduced exploratory behavior within the first 20 min post-injury; however, these effects were resolved after 24 h. A single ACHI impaired spatial memory on the Y-maze task at both 5 min and 24 h post-ACHI; however, no deficits were apparent at 48 h. Immunostaining revealed region-specific increases in ionized calcium-binding adaptor molecule 1 and glial fibrillary acidic protein expression at 3 days post-impact, with no differences found at either 1 or 14 days. Taken together, our findings indicate that a single ACHI results in transient neurobehavioral and glial disturbances and as such, this model may be a valuable tool for pre-clinical concussion research.

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TI Incretin Mimetics as Rational Candidates for the Treatment of Traumatic

Brain Injury

SO ACS PHARMACOLOGY & TRANSLATIONAL SCIENCE

LA English

DT Review

DE TBI; incretins; GLP-1; GIP; Gcg; glucagon; exendin-4; neurodegeneration;

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ID GLUCAGON-LIKE PEPTIDE-1; NF-KAPPA-B; CLOSED-HEAD INJURY; REVERSES

BEHAVIORAL IMPAIRMENTS; GLP-1/GIP RECEPTOR AGONISTS; GLUTAMATE

TRANSPORTER GLT-1; APOPTOSIS-INDUCING FACTOR; FLUID PERCUSSION INJURY;

LARGE ANIMAL-MODELS; MPTP MOUSE MODEL

AB Traumatic brain injury (TBI) is becoming an increasing public health issue. With an annually estimated 1.7 million TBIs in the United States (U.S.) and nearly 70 million worldwide, the injury, isolated or compounded with others, is a major cause of short- and long-term disability and mortality. This, along with no specific treatment, has made exploration of TBI therapies a priority of the health system. Age and sex differences create a spectrum of vulnerability to TBI, with the highest prevalence among younger and older populations. Increased public interest in the long term effects and prevention of TBI have recently reached peaks, with media attention bringing heightened awareness to sport and war related head injuries. Along with short-term issues, TBI can increase the likelihood for development of long-term neurodegenerative disorders. A growing body of literature supports the use of glucagon-like peptide-1 (GLP-1), glucose-dependent insulinotropic peptide (GIP), and glucagon (Gcg) receptor (R) agonists, along with unimolecular combinations of these therapies, for their potent neurotrophic/neuroprotective activities across a variety of cellular and animal models of chronic neurodegenerative diseases (Alzheimer's and Parkinson's diseases) and acute cerebrovascular disorders (stroke). Mild or moderate TBI shares many of the hallmarks of these conditions; recent work provides evidence that use of these compounds is an effective strategy for its treatment. Safety and efficacy of many incretin-based therapies (GLP-1 and GIP) have been demonstrated in humans for the treatment of type 2 diabetes mellitus (T2DM), making these compounds ideal for rapid evaluation in clinical trials of mild and moderate TBI.

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TI APOE Genotype Specific Effects on the Early Neurodegenerative Sequelae

Following Chronic Repeated Mild Traumatic Brain Injury

SO NEUROSCIENCE

LA English

DT Article

DE APOE4; astrocytes; mild TBI; microglia; phospholipid; tau

ID APOLIPOPROTEIN-E POLYMORPHISM; LOW-DENSITY-LIPOPROTEIN; AMYLOID-BETA

PEPTIDE; ALZHEIMERS-DISEASE; MOUSE MODEL; RISK-FACTOR; OXIDIZED LDL;

ENDOTHELIAL RECEPTOR; GLIAL ACTIVATION; AXONAL INJURY

AB Repeated mild traumatic brain injury (r-mTBI) can potentially manifest into chronic traumatic encephalopathy (CTE). The apolipoprotein E (APOE4) genotype, a well-recognized potent genetic risk factor in age-related neurodegenerative diseases such as Alzheimer's disease, has been linked to worse outcome after TBI in individuals who carry this allele. The underlying molecular modifications triggered by APOE genotype following r-mTBI remain elusive. We addressed the influence of APOE genotype on TBI dependent tau pathology in middle-aged mice. Using a previously established experimental mTBI protocol in a new repetitive injury paradigm, we report the pathological changes that occurred following one-month of repetitive injuries in APOE3/4 gene targeted mice. Firstly, pathological assessment demonstrated evidence of microgliosis and astrogliosis in the corpus callosum of injured animals, but there was no APOE dependent genotype effect on injury. However, in the parietal cortex Iba1-immunoreactivity was significantly increased in injured versus sham APOE3 mice, but not in APOE4 mice. No effects were observed in soluble amyloid levels with injury or interaction with genotype. APOE4 mice showed significant increases in the tau conformational marker MC1, neurofilament H, brain phospholipids, and endothelial specific oxidized low density lipoprotein receptor in cortical homogenates obtained from injured mice compared to sham counterparts. This pilot work suggests APOE3 and APOE4 specific effects following injury in a mouse model of r-mTBI. These changes may underlie the molecular changes that trigger the vulnerability and increased risk of developing neurodegenerative diseases in aged individuals exposed to repetitive mTBI. (C) 2019 IBRO. Published by Elsevier Ltd. All rights reserved.

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TI A Tilted Axis: Maladaptive Inflammation and HPA Axis Dysfunction

Contribute to Consequences of TBI

SO FRONTIERS IN NEUROLOGY

LA English

DT Review

DE traumatic brain injury; psychiatric disorders; neuroinflammation;

microglia; stress; HPA axis; glucocorticoids

ID TRAUMATIC BRAIN-INJURY; CORTICOTROPIN-RELEASING HORMONE;

PITUITARY-ADRENAL AXIS; RECEPTOR-MEDIATED INHIBITION; FORCED-SWIM TEST;

LONG-TERM; SLEEP-DEPRIVATION; MOUSE MODEL; MICROGLIAL ACTIVATION;

NEUROENDOCRINE DYSFUNCTION

AB Each year approximately 1.7 million people sustain a traumatic brain injury (TBI) in the US alone. Associated with these head injuries is a high prevalence of neuropsychiatric symptoms including irritability, depression, and anxiety. Neuroinflammation, due in part to microglia, can worsen or even cause neuropsychiatric disorders after TBI. For example, mounting evidence demonstrates that microglia become "primed" or hyper-reactive with an exaggerated pro-inflammatory phenotype following multiple immune challenges. Microglial priming occurs after experimental TBI and correlates with the emergence of depressive-like behavior as well as cognitive dysfunction. Critically, immune challenges are various and include illness, aging, and stress. The collective influence of any combination of these immune challenges shapes the neuroimmune environment and the response to TBI. For example, stress reliably induces inflammation and could therefore be a gateway to altered neuropathology and behavioral decline following TBI. Given the increasing incidence of stress-related psychiatric disorders after TBI, the degree in which stress affects outcome is of particular interest. This review aims to highlight the role of the hypothalamic-pituitary-adrenal (HPA) axis as a key mediator of stress-immune pathway communication following TBI. We will first describe maladaptive neuroinflammation after TBI and how stress contributes to inflammation through both anti- and pro-inflammatory mechanisms. Clinical and experimental data describing HPA-axis dysfunction and consequences of altered stress responses after TBI will be discussed. Lastly, we will review common stress models used after TBI that could better elucidate the relationship between HPA axis dysfunction and maladaptive inflammation following TBI. Together, the studies described in this review suggest that HPA axis dysfunction after brain injury is prevalent and contributes to the dynamic nature of the neuroinflammatory response to brain injury. Experimental stressors that directly engage the HPA axis represent important areas for future research to better define the role of stress-immune pathways inmediating outcome following TBI.

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TI Angiotensin II Receptor 1 Blockage Limits Brain Damage and Improves

Functional Outcome After Brain Injury in Aged Animals Despite

Age-Dependent Reduction in AT1 Expression

SO FRONTIERS IN AGING NEUROSCIENCE

LA English

DT Article

DE angiotensin II receptor type 1; traumatic brain injury; aged;

inflammation; candesartan; AT1 inhibition; neutrophil granulocyte; M2

microglia polarization

ID DOPAMINERGIC DEGENERATION; ALTERNATIVE ACTIVATION; MOUSE MODEL; TYPE-1

RECEPTOR; GENE-EXPRESSION; RHO-KINASE; RT-PCR; SYSTEM; NORMALIZATION;

INFLAMMATION

AB Traumatic brain injury (TBI) is a frequent pathology associated with poor neurological outcome in the aged population. We recently observed accelerated cerebral inflammation in aged mice in response to TBI. Candesartan is a potent specific inhibitor of angiotensin II receptor type 1 (AT1) which limits cerebral inflammation and brain damage in juvenile animals after experimental TBI. In the present study, we show significantly lower posttraumatic AT1 mRNA levels in aged (21 months) compared to young (2 months) mice. Despite low cerebral At1 expression, pharmacologic blockade by treatment with candesartan [daily, beginning 30 min after experimental TBI by controlled cortical impact (CCI)] was highly effective in both young and aged animals and reduced histological brain damage by -20% after 5 days. In young mice, neurological improvement was enhanced by AT1 inhibition 5 days after CCI. In older animals, candesartan treatment reduced functional impairment already on day 3 after TBI and post-traumatic body weight (BW) loss was attenuated. Candesartan reduced microglia activation (-40%) in young and aged animals, and neutrophil infiltration (-40% to 50%) in aged mice, whereas T-cell infiltration was not changed in either age group. In young animals, markers of anti-inflammatory microglia M2a polarization [arginase 1 (Arg1), chitinase3-like 3 (Ym1)] were increased by candesartan at days 1 and 5 after insult. In older mice 5 days after insult, expression of Arg1 was significantly higher independently of the treatment, whereas Ym1 gene expression was further enhanced by AT1 inhibition. Despite age-dependent posttraumatic differences in At1 expression levels, inhibition of AT1 was highly effective in a posttreatment paradigm. Targeting inflammation with candesartan is, therefore, a promising therapeutic strategy to limit secondary brain damage independent of the age.

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TI Old age increases microglial senescence, exacerbates secondary

neuroinflammation, and worsens neurological outcomes after acute

traumatic brain injury in mice

SO NEUROBIOLOGY OF AGING

LA English

DT Article

DE Aging; Traumatic brain injury; Neuroinflammation; Microglia; Behavior

ID ALZHEIMERS-DISEASE; OXIDATIVE STRESS; ELDERLY-PATIENTS; YOUNG ADULTHOOD;

NEURODEGENERATION; INFLAMMATION; ACTIVATION; DAMAGE; CELLS; DEGRADATION

AB After traumatic brain injury (TBI), individuals aged over 65 years show increased mortality and worse functional outcomes compared with younger persons. As neuroinflammation is a key pathobiological mechanism of secondary injury after TBI, we examined how aging affects post-traumatic microglial responses and functional outcomes. Young (3-month-old) and aged (18-month-old) male C57BL/6 mice were subjected to moderate-level controlled cortical impact or sham surgery, and neurological function was evaluated. At 72 hours after injury, brain, blood, and spleen leukocyte counts were assessed ex vivo using flow cytometry. Aged mice demonstrated more severe deficits in forelimb grip strength, balance and motor coordination, spontaneous locomotor activity, and anxiety-like behavior. These animals also exhibited more robust microglial proliferation and significantly higher numbers of brain-infiltrating leukocytes. Microglia in aged mice showed impairments in phagocytic activity and higher production of interleukin-1 beta (IL-1 beta). Infiltrating myeloid cells in aged TBI mice also had deficits in phagocytosis but showed diminished proinflammatory cytokine production and greater reactive oxygen species production. Expression of several senescence markers (Bcl-2, p16(ink4a), p21(cip1a), lipofuscin, and H2AX [pS139]) was increased with age and/or TBI in both microglia and injured cortex. Although there was no difference in the number of circulating blood neutrophils as a function of age, young mice exhibited more pronounced TBI-induced splenomegaly and splenic myeloid cell expansion. Thus, worse post-traumatic behavioral outcomes in aged animals are associated with exaggerated microglial responses, increased leukocyte invasion, and upregulation of senescence markers. (C) 2019 Elsevier Inc. All rights reserved.

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TI Myeloid-Derived Suppressor Cells Infiltrate the Brain and Suppress

Neuroinflammation in a Mouse Model of Focal Traumatic Brain Injury

SO NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury; inflammation; blood-brain barrier;

myeloid-derived suppressor cells; microglia

ID INFLAMMATION; ADHESION; PET

AB Myeloid-derived suppressor cells (MDSCs) have strong immunosuppressive characteristics, which allow them to limit inflammation and facilitate wound healing and recovery. Although MDSCs are a newly-determined cell type that is gaining attention in the immunology field, their neuroimmunological characteristics remain unstudied. In this study, we explored the suppressive role of MDSCs in cerebral inflammatory reactions after focal traumatic brain injury (TBI) using in vivo imaging. Through morphological, functional, and phenotypic analyses we determined that CD11b(+)/Gr-1(+) cells infiltrating the contusion area are MDSCs. MDSCs are among the first responders to tissue injury, responding even prior to microglial activation. Positron emission tomography imaging of translocator protein results suggest that infiltrating MDSCs suppress neuronal inflammation and interact with resident immune cells, like microglia, following focal TBI. (C) 2019 IBRO. Published by Elsevier Ltd. All rights reserved.

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TI Differential responses to increasing numbers of mild traumatic brain

injury in a rodent closed-head injury model

SO JOURNAL OF NEUROCHEMISTRY

LA English

DT Article

DE functional deficits; microglia; myelin abnormalities; oxidative stress;

repeated mild traumatic brain injury; transmission electron microscopy

ID WHITE-MATTER; SECONDARY DEGENERATION; COGNITIVE IMPAIRMENT; AXONAL

INJURY; WATER-MAZE; VULNERABILITY; CONCUSSION; PATHOLOGY; RATS;

PATHOPHYSIOLOGY

AB Following mild traumatic brain injury (mTBI), further mild impacts can exacerbate negative outcomes. To compare chronic damage and deficits following increasing numbers of repeated mTBIs, a closed-head weight-drop model of repeated mTBI was used to deliver 1, 2 or 3 mTBIs to adult female rats at 24 h intervals. Outcomes were assessed at 3 months following the first mTBI. No gross motor, sensory or reflex deficits were identified (p > 0.05), consistent with current literature. Cognitive function assessed using a Morris water maze revealed chronic memory deficits following 1 and 2, but not 3 mTBI compared to shams (p <= 0.05). Oxidative damage to DNA was assessed immunohistochemically in the dentate hilus of the hippocampus and splenium of the corpus callosum; no changes were observed. IBA1-positive microglia were increased in size in the cortex following 1 mTBI and in the corpus callosum following 2 mTBI compared to shams (p <= 0.05); no changes were observed in the dentate hilus. Glial fibrillary acidic protein (GFAP)-positive astrocyte immunoreactivity was assessed in all three brain regions and no chronic changes were observed. Integrity of myelin ultrastructure in the corpus callosum was assessed using transmission electron microscopy. G ratio was decreased following 2 mTBIs compared to shams (p <= 0.05) at post hoc level only. The changing patterns of damage and deficits following increasing numbers of mTBI may reflect dynamic responses to small numbers of mTBIs or a conditioning effect such that increasing numbers of mTBIs do not necessarily result in worsening pathology. Open science badges This article has received a badge for \*Open Materials\* because it provided all relevant information to reproduce the study in the manuscript. The complete Open Science Disclosure form for this article can be found at the end of the article. More information about the Open Practices badges can be found at . Cover Image for this issue: doi: .

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TI Dissemination of brain inflammation in traumatic brain injury

SO CELLULAR & MOLECULAR IMMUNOLOGY

LA English

DT Review

DE disseminated brain inflammation; traumatic brain injury; microglia;

post-injury neurodegeneration

ID PITUITARY DYSFUNCTION; INNATE IMMUNITY; TAU PATHOLOGY; T-CELLS;

EXPRESSION; NEUROINFLAMMATION; NEUROPROTECTION; MICE; MICROGLIA;

ACCUMULATION

AB Traumatic brain injury (TBI) is recognized as a global health problem due to its increasing occurrence, challenging treatment, and persistent impacts on brain pathophysiology. Neural cell death in patients with TBI swiftly causes inflammation in the injured brain areas, which is recognized as focal brain inflammation. Focal brain inflammation causes secondary brain injury by exacerbating brain edema and neuronal death, while also exerting divergent beneficial effects, such as sealing the damaged limitans and removing cellular debris. Recent evidence from patients with TBI and studies on animal models suggest that brain inflammation after TBI is not only restricted to the focal lesion but also disseminates to remote areas of the brain. The dissemination of inflammation has been detected within days after the primary injury and persists chronically. This state of inflammation may be related to remote complications of TBI in patients, such as hyperthermia and hypopituitarism, and may lead to progressive neurodegeneration, such as chronic traumatic encephalopathy. Future studies should focus on understanding the mechanisms that govern the initiation and propagation of brain inflammation after TBI and its impacts on post-trauma brain pathology.

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TI Inflammation in Traumatic Brain Injury: Roles for Toxic A1 Astrocytes

and Microglial-Astrocytic Crosstalk

SO NEUROCHEMICAL RESEARCH

LA English

DT Article

DE Traumatic brain injury; Inflammation; Astrocyte; Toxic phenotype; Glial

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ID REACTIVE ASTROCYTES; SCAR FORMATION; CELLS; NEUROINFLAMMATION; SENSOR;

MODEL; STEREOLOGY; MECHANISMS; RESPONSES; OUTCOMES

AB Traumatic brain injury triggers neuroinflammation that may contribute to progressive neurodegeneration. We investigated patterns of recruitment of astrocytes and microglia to inflammation after brain trauma by firstly characterising expression profiles over time of marker genes following TBI, and secondly by monitoring glial morphologies reflecting inflammatory responses in a rat model of traumatic brain injury (i.e. the lateral fluid percussion injury). Gene expression profiles revealed early elevation of expression of astrocytic marker glial fibrillary acidic protein relative to microglial marker allograft inflammatory factor 1 (also known as ionized calcium-binding adapter molecule 1). Adult rat brains collected at day 7 after injury were processed for immunohistochemistry with allograft inflammatory factor 1, glial fibrillary acidic protein and complement C3 (marker of bad/disruptive astrocytic A1 phenotype). Astrocytes positive for glial fibrillary acidic protein and complement C3 were significant increased in the injured cortex and displayed more complex patterns of arbourisation with significantly increased bifurcations. Our observations suggested that traumatic brain injury changed the phenotype of microglia from a ramified appearance with long, thin, highly branched processes to a swollen amoeboid shape in the injured cortex. These findings suggest differential glial activation with astrocytes likely undergoing strategic changes in morphology and function. Whilst a detailed analysis is needed of temporal patterns of glial activation, ours is the first evidence of a role for the bad/disruptive astrocytic A1 phenotype in an open head model of traumatic brain injury.

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TI Depletion of microglia immediately following traumatic brain injury in

the pediatric rat: Implications for cellular and behavioral pathology

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Pediatric TBI; Microglia; Clodronate; Neurodegeneration; Spatial

learning; Cortical activity

ID CEREBROSPINAL-FLUID; UNITED-STATES; AXONAL INJURY; MOUSE MODEL;

MINOCYCLINE; ACTIVATION; CHILDREN; MACROPHAGE; MODERATE; CELLS

AB The inflammatory response is a significant component of the pathophysiology of pediatric traumatic brain injury. High levels of inflammatory mediators have been found in the cerebrospinal fluid of brain-injured children which have been linked to poor prognosis. Targeting aspects of the inflammatory response in the hopes of finding a viable post-injury therapeutic option has gained attention. Microglia are largely responsible for perpetuating the injury-induced inflammatory response but in the developing brain they play beneficial roles in both normal and disease states. Following closed head injury in the neonate rat, depletion of microglia with intracerebral injections of liposomes containing clodronate was associated with an increase in neurodegeneration in the early post-injury period (3 days) relative to those injected with empty liposomes suggestive of a decrease in clearance of dying cells. In sham-injured animals, microglia repopulated the clodrosome-mediated depleted brain regions over a period of 2-4 weeks and exhibited morphology typical of a resting phenotype. In brain-injured animals, the repopulated microglia in clodrosome-injected animals exhibited rod-like and amoeboid morphologies. However, fluoro-Jade B reactivity in these brain regions was more extensive than in empty liposome-injected animals suggesting that the active microglia may be unable to clear dying neurons. This was accompanied by an induction of hyperexcitability in the local cortical circuitry. Depletion of microglia within the white matter tracts and the thalamus did not affect the extent of injury-induced traumatic axonal injury. Increased neurodegeneration in the dorsal subiculum was not accompanied by any changes to injury-induced deficits in spatial learning and memory. These data suggest that activation of microglia may be important for removal of dying neurons in the traumatically-injured immature brain.

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TI Pre-injury monocyte/macrophage depletion results in increased

blood-brain barrier permeability after traumatic brain injury

SO JOURNAL OF NEUROSCIENCE RESEARCH

LA English

DT Article

DE clodronate liposomes; granulocytes; macrophage; microglia; monocyte;

traumatic brain injury

ID REGULATES MICROGLIAL ACTIVATION; CEREBRAL EDEMA; SPINAL-CORD;

MACROPHAGES; CELLS; RATS; PLAY

AB Traumatic brain injury (TBI) effects both the brain and the immune system. Circulating monocytes/macrophages (M-o/M-a) after a TBI may play an important role in preserving the blood-brain barrier (BBB), reducing brain edema, and interacting with resident microglia. To elucidate the role of circulating M-o/M-a, we utilized a monocyte/macrophage depletion model in response to TBI in male rats. Clodronate liposomes (CL) were used to deplete circulating M-o/M-a. A controlled cortical impact (CCI) injury model was used to create a TBI. All animals received either CL or PBS liposomes (PL), 48 and 24 hr prior to the procedure, and were sacrificed 72 hr post-injury for analysis of BBB permeability, brain edema, whole blood (M-o/M-a and granulocytes), and/or microglial analysis. Animals undergoing M-o/M-a depletion with CL prior to CCI (CCI-CL) were found to have increased BBB permeability when compared to non-depleted CCI (CCI-PL) animals. At 72 hr following injury, Sham-CL maintained on average an 82% reduction in the whole blood monocytes when compared to Sham-PL (p < 0.001). Monocytes in the whole blood remained significantly lower in CCI-CL animals when compared to CCI-PL (p < 0.001). The number of granulocytes in the whole blood of CCI-CL animals was higher at 3 days when compared to CCI-PL (p < 0.022). Surprisingly, the depletion of M-o/M-a did not affect brain edema. However, the depletion of M-o/M-a did result in a significant decrease in microglia (CCI-CL vs. CCI-PL, p < 0.012). In conclusion, an intact M-o/M-a population is required to repair BBB integrity and microglial response following injury.

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TI Microglia Receptors in Animal Models of Traumatic Brain Injury

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article

DE Microglia; Brain injury; Receptors

ID TUMOR-NECROSIS-FACTOR; CENTRAL-NERVOUS-SYSTEM; METABOTROPIC GLUTAMATE

RECEPTORS; TOLL-LIKE RECEPTORS; PPAR-GAMMA AGONIST;

VASOACTIVE-INTESTINAL-PEPTIDE; IMPROVES FUNCTIONAL RECOVERY; CONTROLLED

CORTICAL IMPACT; PROLIFERATOR-ACTIVATED RECEPTORS; OUTWARD POTASSIUM

CURRENTS

AB Microglia have been implicated as a key mediator of chronic inflammation following traumatic brain injury (TBI). The animal models of TBI vary significantly based on the type of brain injury (focal versus diffuse). This has made it extremely difficult to assess the role of microglia and the window of microglia activation. Hence, the focus of this review is to summarize the time course ofmicroglia activation in various animal models of TBI. The review explores the repertoire of secondary injurymechanisms such as aberrant neurotransmitter release, oxidative stress, blood-brain barrier disruption, and production of pro-inflammatory cytokines that follow microglia activation. Since receptors act as sensors for activation, we highlight certain microglia receptors that have been implicated in TBI pathology, including fractalkine receptor (CX3CR1), purinergic receptor (P2Y12R), Toll-like receptor (TLR4), scavenger receptors, tumor necrosis factor receptor (TNF-1R), interleukin receptor (IL-1R), complement receptors, and peroxisome proliferator-activated receptor (PPAR). In addition to describing their downstream signaling pathways in TBI, we describe the functional consequences of their activation and the implication in behavioral outcomes. Taken together, this review will provide a holistic view of the role of microglia and its receptors in TBI based on animal studies.

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TI Dynamic cell type-specific expression of Nrf2 after traumatic brain

injury in mice

SO EUROPEAN JOURNAL OF NEUROSCIENCE

LA English

DT Article

DE astrocytes; microglia; neurons; NG2 glia; nuclear factor erythroid

2-related factor 2; traumatic brain injury

ID TRANSCRIPTION FACTOR NRF2; ASTROCYTE-SPECIFIC OVEREXPRESSION; OXIDATIVE

STRESS; NF-E2-RELATED FACTOR-2; THERAPEUTIC TARGET; ACTIVATION; NEURONS;

PATHWAY; NEUROPROTECTION; GLUTATHIONE

AB Nrf2 plays a pivotal role in antioxidant response and anti-inflammation after traumatic brain injury (TBI), and its deletion aggravates TBI-induced brain damage. Previous studies have demonstrated that Nrf2 is activated post TBI, but dynamic changes in expression and cell type-specific characteristics remain unclear. In this study, the Feeney weight-drop contusion model was conducted to mimic TBI, and the ipsilateral cerebral cortex was collected at 1, 3, 7 and 14 days post TBI (dpi). Nrf2 protein levels were observed by western blot. Cell type-specific localization of Nrf2 after TBI was detected at different time intervals by double immunofluorescence staining. NeuN, GFAP, IBA1 and NG2 were used as cell type-specific markers to neurons, astrocytes, microglia and NG2 glia, respectively. After TBI, Nrf2 protein levels peaked at 1 dpi. Robust transient Nrf2 accumulation was co-localized with neurons, which was predominant at 1 dpi. Continuous weak Nrf2 expression was detected in activated astrocytes, and the number of double positive cells peaked at 7 dpi. Inducible widespread immunostaining of Nrf2 was observed in the nucleus of the microglia, and the number of Nrf2+ microglia peaked at 7 dpi. In addition, we also explored colocalization of Nrf2 in NG2 glia, in which the percentage of Nrf2+ in NG2 glia reached a climax at 3 dpi. This study reveals that the accumulation of endogenous Nrf2 might mediate different pathophysical roles in neurons and glias after TBI, the cell-type specific and time-dependent expression provide insights to explain the roles of Nrf2 in different neural cells.

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TI An End-to-end System for Automatic Characterization of Iba1

Immunopositive Microglia in Whole Slide Imaging

SO NEUROINFORMATICS

LA English

DT Article

DE Computer-aided detection and diagnosis; Whole slide imaging; Digital

pathology; Image analysis; Classification; Traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; GLASGOW COMA SCALE; HEAD IMPACT MODEL;

INFLAMMATION; PATHOLOGY; CLASSIFICATION; SEGMENTATION; LIMITATIONS;

ACTIVATION; CANCER

AB Traumatic brain injury (TBI) is one of the leading causes of death and disability worldwide. Detailed studies of the microglial response after TBI require high throughput quantification of changes in microglial count and morphology in histological sections throughout the brain. In this paper, we present a fully automated end-to-end system that is capable of assessing microglial activation in white matter regions on whole slide images of Iba1 stained sections. Our approach involves the division of the full brain slides into smaller image patches that are subsequently automatically classified into white and grey matter sections. On the patches classified as white matter, we jointly apply functional minimization methods and deep learning classification to identify Iba1-immunopositive microglia. Detected cells are then automatically traced to preserve their complex branching structure after which fractal analysis is applied to determine the activation states of the cells. The resulting system detects white matter regions with 84% accuracy, detects microglia with a performance level of 0.70 (F1 score, the harmonic mean of precision and sensitivity) and performs binary microglia morphology classification with a 70% accuracy. This automated pipeline performs these analyses at a 20-fold increase in speed when compared to a human pathologist. Moreover, we have demonstrated robustness to variations in stain intensity common for Iba1 immunostaining. A preliminary analysis was conducted that indicated that this pipeline can identify differences in microglia response due to TBI. An automated solution to microglia cell analysis can greatly increase standardized analysis of brain slides, allowing pathologists and neuroscientists to focus on characterizing the associated underlying diseases and injuries.

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TI BpV(pic) confers neuroprotection by inhibiting M1 microglial

polarization and MCP-1 expression in rat traumatic brain injury

SO MOLECULAR IMMUNOLOGY

LA English

DT Article

DE Traumatic brain injury; BpV(pic); Microglia; Neuroinflammation; NF-kappa

B p65; MCP-1

ID PTEN; NEUROINFLAMMATION; PHOSPHATASE; PEPTIDE; BARRIER; CELLS; CORD

AB Traumatic brain injury (TBI) is a major cause of motor and cognitive impairment in young adults. It is associated with high mortality rates and very few effective treatment options. Bisperoxovanadium (pyridine-2-carboxyl) (bpV(pic)] is an commercially available inhibitor of Phosphatase and tensin homolog (PTEN). Previous studies have shown that bpV(pic) has protective effects in central nervous system. However, the role of bpV(pic) in TBI is unclear. In this study we aimed to investigate the neuroprotective role of bpV(pic) in rat TBI model. We found that injection of bpV(pic) significantly reduces brain edema and neurological dysfunction after TBI and this is mediated by AKT pathway. TBI is known to promote the Ml pro-inflammatory phenotype of microglial polarization and this effect is inhibited by bpV(pic) treatment which, instead promotes M2 microglial polarization in vivo and in vitro. We also found evidence of bpV(pic)-regulated neuroinflammation mediated by AKT activation and NF-kappa B p65 inhibition. BpV(pic) treatment also suppressed microglia in the peri-TBI region. MCP-1 is known to recruit monocytes and macrophages to promote inflammation, we show that bpV(pic) can inhibit TBI-induced up-regulation of MCP-1 via the AKT/NF-kappa B p65 signaling pathway. Taken together, our findings demonstrate that bpV(pic) plays a neuroprotective role in rat TBI, which may be achieved by inhibiting Ml microglia polarization and MCP-1 expression by modulating AKT/NF-kappa B p65 signaling pathway.

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TI Increases in miR-124-3p in Microglial Exosomes Confer Neuroprotective

Effects by Targeting FIP200-Mediated Neuronal Autophagy Following

Traumatic Brain Injury

SO NEUROCHEMICAL RESEARCH

LA English

DT Article

DE Traumatic brain injury; Exosomes; Autophagy; miRNA; Microglia

ID NEGATIVELY REGULATE AUTOPHAGY; MESENCHYMAL STROMAL CELLS; EXTRACELLULAR

VESICLES; MICRORNA EXPRESSION; TUMOR-SUPPRESSOR; DOWN-REGULATION;

IN-VITRO; APOPTOSIS; PATHWAY; CANCER

AB In our recent study, we observed consistent increases in miR-124-3p levels in exosomes derived from cultured BV2 microglia which was treated with repetitive traumatic brain injury (rTBI) mouse model brain extracts. To clarify the mechanisms underlying increases in microglia-derived exosomal miR-124-3p and their role in regulating neuronal autophagy after TBI, we investigated the impact of exosomal miR-124-3p on neuronal autophagy in scratch-injured HT22 neurons and rTBI mice. We harvested injured brain extracts from rTBI mice at 3 to 21days post injury (DPI) for the treatment of cultured BV2 microglia in vitro. We observed significant induction of autophagy following TBI in vitro, and that inhibition of activated neuronal autophagy could protect against trauma-induced injury. Our results indicated that co-culture of injured HT22 neurons with miR-124-3p overexpressing BV2 microglia exerted a protective effect by inhibiting neuronal autophagy in scratch-injured neurons. Further research revealed that these effects were achieved mainly via upregulation of exosomal miR-124-3p, and that Focal adhesion kinase family-interacting protein of 200kDa (FIP200) plays a key role in trauma-induced autophagy. Injection of exosomes into the vena caudalis in in vivo experiments revealed that exosomal miR-124-3p was associated with decreases in the modified neurological severity score (mNSS) and improvements in Morris water maze (MWM) test results in rTBI mice. Altogether, our results indicate that increased miR-124-3p in microglial exosomes following TBI may inhibit neuronal autophagy and protect against nerve injury via their transfer into neurons. Thus, treatment with microglial exosomes enriched with miR-124-3p may represent a novel therapeutic strategy for the treatment of nerve injury after TBI.

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TI Depletion of regulatory T cells increases T cell brain infiltration,

reactive astrogliosis, and interferon-γ gene expression in acute

experimental traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Traumatic brain injury; Inflammation; Cytokines; Immune response; T

cells; Astrocytes; Microglia

ID ISCHEMIC-STROKE; INFLAMMATION; SYSTEM; NEUROPROTECTION; ASTROCYTES;

MOTOR; MICE; NEUROINFLAMMATION; PATHOPHYSIOLOGY; PROGRANULIN

AB Background Traumatic brain injury (TBI) is a major cause of death and disability. T cells were shown to infiltrate the brain during the first days after injury and to exacerbate tissue damage. The objective of this study was to investigate the hitherto unresolved role of immunosuppressive, regulatory T cells (Tregs) in experimental TBI. Methods "Depletion of regulatory T cell" (DEREG) and wild type (WT) C57Bl/6 mice, treated with diphtheria toxin (DTx) to deplete Tregs or to serve as control, were subjected to the controlled cortical impact (CCI) model of TBI. Neurological and motor deficits were examined until 5 days post-injury (dpi). At the 5 dpi endpoint, (immuno-) histological, protein, and gene expression analyses were carried out to evaluate the consequences of Tregs depletion. Comparison of parametric or non-parametric data between two groups was done using Student's t test or the Mann-Whitney U test. For multiple comparisons, p values were calculated by one-way or two-way ANOVA followed by specific post hoc tests. Results The overall neurological outcome at 5 dpi was not different between DEREG and WT mice but more severe motor deficits occurred transiently at 1 dpi in DEREG mice. DEREG and WT mice did not differ in the extent of brain damage, blood-brain barrier (BBB) disruption, or neuronal excitotoxicity, as examined by lesion volumetry, immunoglobulin G (IgG) extravasation, or calpain-generated alpha II-spectrin breakdown products (SBDPs), respectively. In contrast, increased protein levels of glial fibrillary acidic protein (GFAP) and GFAP+ astrocytes in the ipsilesional brain tissue indicated exaggerated reactive astrogliosis in DEREG mice. T cell counts following anti-CD3 immunohistochemistry and gene expression analyses of Cd247 (CD3 subunit zeta) and Cd8a (CD8a) further indicated an increased number of T cells infiltrating the brain injury sites of DEREG mice compared to WT. These changes coincided with increased gene expression of pro-inflammatory interferon-gamma (Ifng) in DEREG mice compared to WT in the injured brain. Conclusions The results show that the depletion of Tregs attenuates T cell brain infiltration, reactive astrogliosis, interferon-gamma gene expression, and transiently motor deficits in murine acute traumatic brain injury.

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TI Time-Dependent Changes in Microglia Transcriptional Networks Following

Traumatic Brain Injury

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DT Article

DE traumatic brain injury; microglia; transcriptome; neurodegeneration;

mice; neuroimmunology; neuroinflammation

ID CONTROLLED CORTICAL IMPACT; INFLAMMATORY RESPONSE; COGNITIVE DEFICITS;

CD40 LIGAND; CELL-DEATH; EXPRESSION; HYPOXIA; NEUROINFLAMMATION;

ACTIVATION; MECHANISM

AB The neuroinflammatory response to traumatic brain injury (TBI) is critical to both neurotoxicity and neuroprotection, and has been proposed as a potentially modifiable driver of secondary injury in animal and human studies. Attempts to broadly target immune activation have been unsuccessful in improving outcomes, in part because the precise cellular and molecular mechanisms driving injury and outcome at acute, subacute, and chronic time points after TBI remain poorly defined. Microglia play a critical role in neuroinflammation and their persistent activation may contribute to long-term functional deficits. Activated microglia are characterized by morphological transformation and transcriptomic changes associated with specific inflammatory states. We analyzed the temporal course of changes in inflammatory genes of microglia isolated from injured brains at 2, 14, and 60 days after controlled cortical impact (CCI) in mice, a well-established model of focal cerebral contusion. We identified a time dependent, injury-associated change in the microglial gene expression profile toward a reduced ability to sense tissue damage, perform housekeeping, and maintain homeostasis in the early stages following CCI, with recovery and transition to a specialized inflammatory state over time. This later state starts at 14 days post-injury and is characterized by a biphasic pattern of IFN gamma, IL-4, and IL-10 gene expression changes, with concurrent proinflammatory and anti-inflammatory gene changes. Our transcriptomic data sets are an important step to understand microglial role in TBI pathogenesis at the molecular level and identify common pathways that affect outcome. More studies to evaluate gene expression at the single cell level and focusing on subacute and chronic timepoint are warranted.

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TI Relationship of traumatic brain injury to chronic mental health problems

and dementia in military veterans

SO NEUROSCIENCE LETTERS

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DT Article

DE Alzheimer's disease; Blast; Chronic traumatic encephalopathy; Dementia;

Inflammation; Microglia; Post-traumatic stress disorder; Traumatic brain

injury; TYROBP/DAP12; TREM2

ID DISORDER-RELATED TRAITS; ALZHEIMERS-DISEASE; BLAST EXPOSURE; TREM2

DEFICIENCY; AMYLOID-BETA; MOUSE MODEL; MICROGLIAL RESPONSE;

APOLIPOPROTEIN-E; SERVICE MEMBERS; HEAD-INJURY

AB Traumatic brain injury (TBI) is an unfortunately common event in military life. The conflicts in Iraq and Afghanistan have increased public awareness of TBI in the military. Certain injury mechanisms are relatively unique to the military, the most prominent being blast exposure. Blast-related mild TBI (mTBI) has been of particular concern in the most recent veterans although controversy remains concerning separation of the postconcussion syndrome associated with mTBI from post-traumatic stress disorder. TBI is also a risk factor for the development of neurodegenerative diseases including chronic traumatic encephalopathy (CTE) and Alzheimer's disease (AD). AD, TBI, and CTE are all associated with chronic inflammation. Genome wide association studies (GWAS) have identified multiple genetic loci associated with AD that implicate inflammation and - in particular microglia - as key modulators of the AD- and TBI-related degenerative processes. At the molecular level, recent studies have identified TREM2 and TYROBP/DAP12 as components of a key molecular hub linking inflammation and microglia to the pathophysiology of AD and possibly TBI. Evidence concerning the relationship of TBI to chronic mental health problems and dementia is reviewed in the context of its relevance to military veterans.

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TI Glial response in early stages of traumatic brain injury

SO NEUROSCIENCE LETTERS

LA English

DT Article

DE Traumatic brain injury; Global glial reaction; Microglia; Astrocytes

ID MICROGLIA; NEURODEGENERATION; INFLAMMATION; ACTIVATION; ASTROCYTE

AB Traumatic brain in jury affects a number of individuals per year and is a major cause of worldwide death and disability. Yet, its pathophysiological mechanism remains unclear. It is well-known that glial cells, including microglia and astrocytes, are activated and involved in tissue damage and repair in the peri-lesion regions after traumatic brain injury; however, global glial responses are rarely reported. The purpose of this study was to investigate the global activation of microglia and astrocytes 1 day after traumatic brain injury. To test this, we used a weight drop device to inflict traumatic brain injury on left side of the brain and performed hematoxylineosin staining to detect tissue damage. We used immunohistochemical staining and western blotting to detect the activation of microglia and astrocytes 1 day after TBI. We found that microglia were significantly activated in ipsilateral regions. Interestingly, we found that astrocytes were also significantly activated in the ipsilateral regions, contralateral cortex, and contralateral corpus callosum. These results suggest that a focal damage can cause a global glial reaction.

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TI Acute drivers of neuroinflammation in traumatic brain injury

SO NEURAL REGENERATION RESEARCH

LA English

DT Review

DE traumatic brain injury; inflammation; neuroinflammation; microglia;

macrophage; acute; diffuse brain injury; cytokines; adenosine

5'-triphosphoate; glutamate; calcium

ID TUMOR-NECROSIS-FACTOR; NLRP3 INFLAMMASOME; EXTRACELLULAR ATP;

FACTOR-ALPHA; MATRIX METALLOPROTEINASES; CEREBROSPINAL-FLUID;

CL-COTRANSPORTER; MESSENGER-RNA; WHITE-MATTER; CELL-LINE

AB Neuroinflammation is initiated as a result of traumatic brain injury and can exacerbate evolving tissue pathology. Immune cells respond to acute signals from damaged cells, initiate neuroinflammation, and drive the pathological consequences over time. Importantly, the mechanism(s) of injury, the location of the immune cells within the brain, and the animal species all contribute to immune cell behavior following traumatic brain injury. Understanding the signals that initiate neuroinflammation and the context in which they appear may be critical for understanding immune cell contributions to pathology and regeneration. Within this paper, we review a number of factors that could affect immune cell behavior acutely following traumatic brain injury.

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TI Depletion of Microglia Attenuates Dendritic Spine Loss and Neuronal

Apoptosis in the Acute Stage of Moderate Traumatic Brain Injury in Mice

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE apoptosis; colony-stimulating factor 1 receptor inhibitor; microglia;

spines; traumatic brain injury

ID CELL-DEATH; STRESS; ACCUMULATION; IMPACT; GAP-43; MODEL

AB Microglia are the primary immune cells in the central nervous system and undergo significant morphological and transcriptional changes after traumatic brain injury (TBI). However, their exact contribution to the pathogenesis of TBI is still debated and remains to be elucidated. In the present study, thy-1 GFP mice received a colony-stimulating factor 1 receptor inhibitor (PLX3397) for 21 consecutive days, then were subjected to moderate fluid percussion injury (FPI). Brain samples were collected at 1 day and 3 days after FPI for flow cytometry analysis, immunofluorescence, dendrite spine quantification, terminal deoxynucleotidyl transferase dUTP nick end labeling assay, and Western blot. We found that PLX3397 treatment significantly attenuated the percentages of resident microglia and infiltrated immune cells. Depletion of microglia promoted neurite outgrowth, preserved dendritic spines and reduced total brain cell and neuronal apoptosis after FPI, which was accompanied by decreased the protein levels of endoplasmic reticulum stress marker proteins, C/EBP-homologous protein and inositol-requiring kinase 1 alpha. Taken together, these findings suggest that microglial depletion may exert beneficial effects in the acute stage of FPI.

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JI J. Neurotrauma

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PT J

AU Kumar, D

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AF Kumar, Deepak

Kaira, Sahela Meenakshi

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Singh, Shamsher

TI Mitochondrial dysfunctioning and neuroinflammation: Recent highlights on

the possible mechanisms involved in Traumatic Brain Injury

SO NEUROSCIENCE LETTERS

LA English

DT Review

DE Traumatic brain injury; Glutamate; Excitotoxicity; Neuroinflammatory

markers; Microglia

ID OXIDATIVE STRESS; CLINICAL-TRIALS; NEURONAL LOSS; ACTIVATION;

NEUROPROTECTION; ERYTHROPOIETIN; PERMEABILITY; INFLAMMATION; INHIBITOR;

MICROGLIA

AB Traumatic brain injury (TBI) is the injury to the vasculature of brain while trauma caused by physical, chemical and biological stimuli. TBI is the leading cause of mortality and morbidity around the world. In this, primary insult leads to secondary injury through the involvement and initiation of various pathological processes. The most citable includes excitotoxicity, Blood Brain Barrier (BBB) dysfunction, inflammation, mitochondrial dysfunction, oxidative stress, calcium efflux, microglial mediated release of proinflammatory mediators (cytokine, chemokines, interleukin, tissue necrosis factor etc.). The morphological changes in TBI are proportional to mitochondrial dysfunctioning and microglial activation, which play an assorted role in neurodegeneration following traumatic brain injury. It is also assumed that the release of nitric oxide, activation of microglial cells plays a diversive role in maintaining the physiological and pathological balance. This review cites different pathophysiological mechanisms that are involved in progenesis of secondary injury after primary insult. These targets further are useful to explore the deep molecular mechanisms and to analyse the effectiveness of available drugs. Moreover, the present review reflects the underlying inflammatory cascade responsible for neuronal loss and neurological deficit in TBI.

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Ziebell, JM

AF Holloway, Olivia G.

Canty, Alison J.

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TI Rod microglia and their role in neurological diseases

SO SEMINARS IN CELL & DEVELOPMENTAL BIOLOGY

LA English

DT Review

DE Rod microglia; Ageing; Neurological disease; Traumatic brain injury

(TBI); Neuroinflammation

ID TRAUMATIC BRAIN-INJURY; ALZHEIMERS-DISEASE; REACTIVE MICROGLIA; IN-VIVO;

ACTIVATION; MOUSE; FRACTALKINE; MORPHOLOGY; ADULT; BIOLOGY

AB The striking morphology of microglia is one of their most prominent characteristics, with many studies categorising microglial function based on morphology e.g. ramified, hyper-ramified, activated, or amoeboid. Communications regarding rod microglia in neurological disease are scant, and where reported, these cells are rarely the focus of discussion. These factors make it difficult to determine how widespread these cells are not only through the brain but also across diseases. Studies in experimental diffuse brain injury are the first reports of not only significant numbers of rod microglia, but distinct arrangements of these cells, reminiscent of carriages of a train. This review summarises the available reports of rod microglia in vivo and rod-like microglia in vitro and eludes to possible functions and signalling cascades that may evoke this distinct morphology. More investigations are required to fully elucidate the function that rod microglia play in neurological diseases.

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TI Neurosteroids as regulators of neuroinflammation

SO FRONTIERS IN NEUROENDOCRINOLOGY

LA English

DT Review

DE Neurosteroids; Estrogens; 17 beta-estradiol; Progestogens; Progesterone;

Allopregnanolone; Dehydroepiandrosterone (DHEA); Neuroinflammation;

Microglia; Astrocytes; Multiple sclerosis; Alzheimer's disease;

Parkinson's disease; Traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS;

ESTROGEN-RECEPTOR-ALPHA; CENTRAL-NERVOUS-SYSTEM; NECROSIS-FACTOR-ALPHA;

NEUROACTIVE STEROID-LEVELS; KDA TRANSLOCATOR PROTEIN; MESSENGER-RNA

EXPRESSION; NITRIC-OXIDE SYNTHASE; FEMALE SEX STEROIDS

AB Neuroinflammation is a physiological protective response in the context of infection and injury. However, neuroinflammation, especially if chronic, may also drive neurodegeneration. Neurodegenerative diseases, such as multiple sclerosis (MS), Alzheimer's disease (AD), Parkinson's disease (PD) and traumatic brain injury (TBI), display inflammatory activation of microglia and astrocytes. Intriguingly, the central nervous system (CNS) is a highly steroidogenic environment synthesizing steroids de novo, as well as metabolizing steroids deriving from the circulation. Neurosteroid synthesis can be substantially affected by neuroinflammation, while, in turn, several steroids, such as 17 beta-estradiol, dehydroepiandrosterone (DHEA) and allopregnanolone, can regulate neuroinflammatory responses. Here, we review the role of neurosteroids in neuroinflammation in the context of MS, AD, PD and TBI and describe underlying molecular mechanisms. Moreover, we introduce the concept that synthetic neurosteroid analogues could be potentially utilized for the treatment of neurodegenerative diseases in the future.

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TI Neuroimmune responses in the developing brain following traumatic brain

injury

SO EXPERIMENTAL NEUROLOGY

LA English

DT Review

DE Pediatric TBI; Neuroinflammatory response; Microglia; Developing brain;

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ID CENTRAL-NERVOUS-SYSTEM; CONTROLLED CORTICAL IMPACT; INNATE

IMMUNE-RESPONSE; SPINAL-CORD-INJURY; DURAL MAST-CELLS; GROUP BOX 1;

OXIDATIVE STRESS; CEREBRAL EDEMA; MOUSE MODEL; NEUTROPHIL ACCUMULATION

AB Traumatic brain injury (TBI) is one of the leading causes of both acute and long-term morbidity in the pediatric population, leading to a substantial, long-term socioeconomic burden. Despite the increase in the amount of preclinical and clinical research, treatment options for TBI rely heavily on supportive care with very limited targeted interventions that improve the acute and chronic sequelae of TBI. Other than injury prevention, not much can be done to limit the primary injury, which consists of tissue damage and cellular destruction. Secondary injury is the result of the ongoing complex inflammatory pathways that further exacerbate tissue damage, resulting in the devastating chronic outcomes of TBI. On the other hand, some level of inflammation is essential for neuronal regeneration and tissue repair. In this review article we discuss the various stages of the neuroimmune response in the immature, pediatric brain in the context of normal maturation and development of the immune system. The developing brain has unique features that distinguish it from the adult brain, and the immune system plays an integral role in CNS development. Those features could potentially make the developing brain more susceptible to worse outcomes, both acutely and in the long-term. The neuroinflammatory reaction which is triggered by TBI can be described as a highly intricate interaction between the cells of the innate and the adaptive immune systems. The innate immune system is triggered by non-specific danger signals that are released from damaged cells and tissues, which in turn leads to neutrophil infiltration, activation of microglia and astrocytes, complement release, as well as histamine release by mast cells. The adaptive immune response is subsequently activated leading to the more chronic effects of neuroinflammation. We will also discuss current attempts at modulating the TBI-induced neuroinflammatory response. A better understanding of the role of the immune system in normal brain development and how immune function changes with age is crucial for designing therapies to appropriately target the immune responses following TBI in order to enhance repair and plasticity.

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TI Microglial Phagocytosis of Neurons: Diminishing Neuronal Loss in

Traumatic, Infectious, Inflammatory, and Autoimmune CNS Disorders

SO FRONTIERS IN PSYCHIATRY

LA English

DT Article

DE depression; microglia; neuron; autoimmunity; inflammation; traumatic

brain injury; cytokine; excitotoxicity

ID CENTRAL-NERVOUS-SYSTEM; C-REACTIVE PROTEIN; BLOOD-BRAIN-BARRIER;

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STRESS; ADIPOSE-TISSUE; ENDOTHELIAL DYSFUNCTION; ALZHEIMERS-DISEASE

AB Errors in neuron-microglial interaction are known to lead to microglial phagocytosis of live neurons and excessive neuronal loss, potentially yielding poorer clinical outcomes. Factors that affect neuron-microglial interaction have the potential to influence the error rate. Clinical comorbidities that unfavorably impact neuron-microglial interaction may promote a higher rate of neuronal loss, to the detriment of patient outcome. This paper proposes that many common, clinically modifiable comorbidities have a common thread, in that they all influence neuron-microglial interactions. Comorbidities like traumatic brain injury, infection, stress, neuroinflammation, loss of neuronal metabolic integrity, poor growth factor status, and other factors, all have the potential to alter communication between neurons and microglia. When this occurs, microglial phagocytosis of live neurons can increase. In addition, microglia can shift into a morphological form in which they express major histocompatibility complex II (MHC-II), allowing them to function as antigen presenting cells that present neuronal debris as antigen to invading T cells. This can increase risk for the development of CNS autoimmunity, or can exacerbate existing CNS autoimmunity. The detrimental influence of these comorbidities has the potential to contribute to the mosaic of factors that determine patient outcome in some CNS pathologies that have neuropsychiatric involvement, including TBI and CNS disorders with autoimmune components, where excessive neuronal loss can yield poorer clinical outcomes. Recognition of the impact of these comorbidities may contribute to an understanding of the common clinical observation that many seemingly disparate factors contribute to the overall picture of case management and clinical outcome in these complex disorders. In a clinical setting, knowing how these comorbidities can influence neuron-microglial interaction can help focus surveillance and care on a broader group of potential therapeutic targets. Accordingly, an interest in the mechanisms underlying the influence of these factors on neuron-microglial interactions is appropriate. Neuronmicroglial interaction is reviewed, and the various mechanisms by which these potential comorbidities influence neuro-microglial interaction are described.

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TI Valproic acid affects neuronal fate and microglial function via

enhancing autophagic flux in mice after traumatic brain injury

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DE autophagic flux; inflammation; microglia; traumatic brain injury (TBI);

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ID CELL-DEATH; THERAPEUTIC TARGET; BARRIER DISRUPTION; HEMORRHAGIC-SHOCK;

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POLARIZATION

AB In recent years, many studies have focused on autophagy, an evolutionarily conserved mechanism that relies on lysosomes to achieve cellular metabolic requirements and organelle turnover, and revealed its important role in animal models of traumatic injury. Autophagy is a double-edged sword. Appropriate levels of autophagy can promote the removal of abnormal proteins or damaged organelles, while hyperactivated autophagy can induce autophagic apoptosis. However, recent studies suggest that autophagic flux seems to be blocked after traumatic brain injury (TBI), which contributes to the apoptosis of brain cells. In this study, valproic acid (VPA), which was clinically used for epilepsy treatment, was used to treat TBI. The Morris water maze test, hematoxylin & eosin staining and Nissl staining were first conducted to confirm that VPA treatment had a therapeutic effect on mice after TBI. Western blotting, enzyme-linked immunosorbent assay and immunofluorescence staining were then performed to reveal that VPA treatment reversed TBI-induced blockade of autophagic flux, which was accompanied by a reduced inflammatory response. In addition, the variations in activation and phenotypic polarization of microglia were observed after VPA treatment. Nevertheless, the use of the autophagy inhibitor 3-methyladenine partially abolished VPA-induced neuroprotection and the regulation of microglial function after TBI, resulting in the deterioration of the central nervous system microenvironment and neurological function. Collectively, VPA treatment reversed the TBI-induced blockade of autophagic flux in the mouse brain cortex, subsequently inhibiting brain cell apoptosis and affecting microglial function to achieve the promotion of functional recovery in mice after TBI.

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TI TSG-6 in conditioned media from adipose mesenchymal stem cells protects

against visual deficits in mild traumatic brain injury model through

neurovascular modulation

SO STEM CELL RESEARCH & THERAPY

LA English

DT Article

DE Endothelial; ERG; OKN; Retina; Microglia; Muller; Paracrine; TBI; MSC;

SOD2; Stat3

ID MURINE MODEL; INFLAMMATION; ACTIVATION; MICROGLIA; DEGENERATION;

MINOCYCLINE; EXPRESSION; RESPONSES; MEMORY; BLAST

AB Background Retinal inflammation affecting the neurovascular unit may play a role in the development of visual deficits following mild traumatic brain injury (mTBI). We have shown that concentrated conditioned media from adipose tissue-derived mesenchymal stem cells (ASC-CCM) can limit retinal damage from blast injury and improve visual function. In this study, we addressed the hypothesis that TNF alpha-stimulated gene-6 (TSG-6), an anti-inflammatory protein released by mesenchymal cells, mediates the observed therapeutic potential of ASCs via neurovascular modulation. Methods About 12-week-old C57Bl/6 mice were subjected to 50-psi air pulse on the left side of the head overlying the forebrain resulting in an mTBI. Age-matched sham blast mice served as control. About 1 mu l of ASC-CCM (siControl-ASC-CCM) or TSG-6 knockdown ASC-CCM (siTSG-6-ASC-CCM) was delivered intravitreally into both eyes. One month following injection, the ocular function was assessed followed by molecular and immunohistological analysis. In vitro, mouse microglial cells were used to evaluate the anti-inflammatory effect of ASC-CCM. Efficacy of ASC-CCM in normalizing retinal vascular permeability was assessed using trans-endothelial resistance (TER) and VE-cadherin expression in the presence of TNF alpha (1 ng/ml). Results We show that intravitreal injection of ASC-CCM (siControl-ASC-CCM) but not the TSG-6 knockdown ASC-CCM (siTSG-6-ASC-CCM) mitigates the loss of visual acuity and contrast sensitivity, retinal expression of genes associated with microglial and endothelial activation, and retinal GFAP immunoreactivity at 4 weeks after blast injury. In vitro, siControl-ASC-CCM but not the siTSG-6-ASC-CCM not only suppressed microglial activation and STAT3 phosphorylation but also protected against TNF alpha-induced endothelial permeability as measured by transendothelial electrical resistance and decreased STAT3 phosphorylation. Conclusions Our findings suggest that ASCs respond to an inflammatory milieu by secreting higher levels of TSG-6 that mediates the resolution of the inflammatory cascade on multiple cell types and correlates with the therapeutic potency of the ASC-CCM. These results expand our understanding of innate mesenchymal cell function and confirm the importance of considering methods to increase the production of key analytes such as TSG-6 if mesenchymal stem cell secretome-derived biologics are to be developed as a treatment solution against the traumatic effects of blast injuries and other neurovascular inflammatory conditions of the retina.

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TI Acute peripheral inflammation and post-traumatic sleep differ between

sexes after experimental diffuse brain injury

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ID GENDER-DIFFERENCES; BAYESIAN METHODS; R PACKAGE; CYTOKINES;

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AB Identifying differential responses between sexes following traumatic brain injury (TBI) can elucidate the mechanisms behind disease pathology. Peripheral and central inflammation in the pathophysiology of TBI can increase sleep in male rodents, but this remains untested in females. We hypothesized that diffuse TBI would increase inflammation and sleep in males more so than in females. Diffuse TBI was induced in C57BL/6J mice and serial blood samples were collected (baseline, 1, 5, 7 days post-injury [DPI]) to quantify peripheral immune cell populations and sleep regulatory cytokines. Brains and spleens were harvested at 7DPI to quantify central and peripheral immune cells, respectively. Mixed-effects regression models were used for data analysis. Female TBI mice had 77%-124% higher IL-6 levels than male TBI mice at 1 and 5DPI, whereas IL-1 beta and TNF-alpha levels were similar between sexes at all timepoints. Despite baseline sex differences in blood-measured Ly6C(high) monocytes (females had 40% more than males), TBI reduced monocytes by 67% in TBI mice at 1DPI. Male TBI mice had 31%-33% more blood-measured and 31% more spleen-measured Ly6G(+) neutrophils than female TBI mice at 1 and 5DPI, and 7DPI, respectively. Compared with sham, TBI increased sleep in both sexes during the first light and dark cycles. Male TBI mice slept 11%-17% more than female TBI mice, depending on the cycle. Thus, sex and TBI interactions may alter the peripheral inflammation profile and sleep patterns, which might explain discrepancies in disease progression based on sex.

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TI Molecular imaging of neuroinflammation in patients after mild traumatic

brain injury: a longitudinal <SUP>123</SUP>I-CLINDE single photon

emission computed tomography study

SO EUROPEAN JOURNAL OF NEUROLOGY

LA English

DT Article

DE concussion; inflammation; microglia; mild traumatic brain injury;

neuroinflammation; post-concussion symptoms; post-concussion syndrome;

translocator protein

ID ACTIVATION

AB Background and purpose Neuroinflammation has been proposed as part of the pathogenesis of post-concussion symptoms (PCS), but the inflammatory response of the human brain to mild traumatic brain injury (mTBI) remains unknown. We hypothesized that a neuroinflammatory response is present in mTBI at 1-2 weeks post-injury and persists in patients with PCS. Methods We scanned 14 patients with mTBI without signs of structural damage at 1-2 weeks and 3-4 months post-injury and 22 healthy controls once using the single photon emission computed tomography tracer I-123-CLINDE, which visualizes translocator protein (TSPO), a protein upregulated in active immune cells. PCS was defined as three or more persisting symptoms from the Rivermead Post Concussion Symptoms Questionnaire at 3 months post-injury. Results Across brain regions, patients had significantly higher I-123-CLINDE binding to TSPO than healthy controls, both at 1-2 weeks after the injury in all patients (P = 0.011) and at 3-4 months in the seven patients with PCS (P = 0.006) and in the six patients with good recovery (P = 0.018). When the nine brain regions were tested separately and results were corrected for multiple comparisons, no individual region differed significantly, but all estimated parameters indicated increased I-123-CLINDE binding to TSPO, ranging from 2% to 19% in all patients at 1-2 weeks, 13% to 27% in patients with PCS at 3-4 months and -9% to 17% in patients with good recovery at 3-4 months. Conclusions Neuroinflammation was present in mTBI at 1-2 weeks post-injury and persisted at 3-4 months post-injury with a tendency to be most pronounced in patients with PCS.

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TI Microglial cell-derived interleukin-6 influences behavior and

inflammatory response in the brain following traumatic brain injury

SO GLIA

LA English

DT Article; Early Access

DE behavior; cryolesion; knockout microglial IL-6; neuroinflammation;

transcriptome

ID ASTROCYTE-TARGETED EXPRESSION; TRANSGENIC MICE; FRACTALKINE-RECEPTOR;

COGNITIVE FUNCTION; METALLOTHIONEIN-I; DEFICIENCY LEADS;

GENE-EXPRESSION; BODY-WEIGHT; COLD INJURY; WILD-TYPE

AB Traumatic brain injury (TBI) is a major health problem with high rates of mortality and morbidity worldwide. The response of the brain to TBI is orchestrated by a number of cytokines, including interleukin-6 (IL-6). IL-6 is a major cytokine in the central nervous system and it is produced by different cells, such as neurons, glial cells, and endothelial cells. Since glial cells are one of the most important sources and targets of IL-6, we have examined the role of microglia-derived IL-6 in normal conditions and following a model of TBI, cryolesion of the somatosensorial cortex. To this end, tamoxifen-inducible microglial IL-6-deficient (Il6(Delta Mic), using Cx3cr1(CreER) model) mice and control (Il6(lox/lox)) mice were used. In normal conditions, microglial IL-6 deficiency reduced deambulation and exploratory behavior and decreased anxiety in a sex-dependent manner. The transcriptome profile following cryolesion was dramatically altered 1 day post-lesion in Il6(Delta Mic) compared with Il6(lox/lox) mice. However, the phenotype of Il6(Delta Mic) mice was less compromised in the following days, suggesting that compensatory mechanisms are at play.

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AF Chen, Mingrui

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TI Tanshinone IIA Promotes M2 Microglia by ERβ/IL-10 Pathway and Attenuates

Neuronal Loss in Mouse TBI Model

SO NEUROPSYCHIATRIC DISEASE AND TREATMENT

LA English

DT Article

DE tanshinone IIA; TNA; traumatic brain injury; TBI; microglia;

interleukins; macrophage polarization

ID TRAUMATIC BRAIN-INJURY; FACTOR-KAPPA-B; MACROPHAGE POLARIZATION;

IN-VIVO; CELLS; EXPRESSION; NEURODEGENERATION; ACTIVATION; BETA

AB Purpose: Traumatic brain injury (TBI) is a major cause of morbidity and mortality worldwide. Increasing evidence indicates that activated microglia play an important role in the inflammatory response in TBI. Inhibiting M1 and stimulating M2 activated microglia have protective effects in several animal models of central nervous system (CNS) disorders. In the present study, we investigated whether tanshinone IIA (TNA) protects neurons by shifting microglia polarization in a mouse TBI model and further investigated the mechanism in vitro.

Materials and Methods: Forty C57BL/6 mice were used to investigate the effect of TNA on microglia polarization in TBI. BV-2 cells were used to examine the mechanism of TNA in regulating microglia polarization.

Results: Normal saline (NS), TNA and the combination of TNA with ICI 182,780 (ICI, an estrogen receptor antagonist) were used to treat the TBI mice. After TBI, mice from each group demonstrated functional improvement. The improvement rate in mice treated with TNA was faster than other groups. ICI partially reversed the benefits from TNA treatment. TNA treatment significantly reduced TBI-induced neuronal loss. The number of microglia after TBI was not significantly changed by TNA treatment. However, TNA treatment significantly decreased M1 macrophage markers (iNOS, TNF alpha and IL-1 beta) and increased M2 macrophage markers (CD206, arginase 1 and Ym1). This effect was partially abolished by ICI. TNA treatment downregulated M1 macrophage markers and upregulated M2 macrophage markers in BV-2 cells under LPS stimulation. IL-10 was significantly increased by TNA treatment without a significantly change of IL-4 and IL-13 expression. IL-10 knockdown completely abolished the effect of TNA on microglial M2 polarization.

Conclusion: Taken together, our data demonstrated that TNA attenuates neuronal loss in mouse TBI model and promotes M2 microglia by ER beta/IL-10 pathway. Thus, TNA could be a potential drug for TBI and/or the disorders that caused by microglial over-activation in CNS.

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TI MEK inhibitor trametinib attenuates neuroinflammation and cognitive

deficits following traumatic brain injury in mice

SO AMERICAN JOURNAL OF TRANSLATIONAL RESEARCH

LA English

DT Article

DE Traumatic brain injury; microglia; neuroinflammation; MEK/ERK;

trametinib

ID NF-KAPPA-B; GROWTH-FACTOR; SIGNALING PATHWAYS; MICROGLIAL CELLS;

PROTEIN-KINASES; ERK; ACTIVATION; SURVIVAL; ROLES; BETA

AB Microglia-mediated neuroinflammation is one of the hallmark pathological features following traumatic brain injury (TBI) that contributes to aggravated brain damage and cognitive deficits. These pathologies require novel effective treatments to improve prognosis. Trametinib, a mitogen-activated protein kinase inhibitor approved by the Food and Drug Administration in treating various malignant tumors, has been shown to exert anti-inflammatory effects. The present study demonstrated that TBI mice treated with trametinib exhibited improved cognitive function. Trametinib treatment rescued oligodendrocytes and decreased infiltrating microglial density in the TBI area. Furthermore, this study revealed that ameliorated lipopolysaccharides (LPS) induced inflammatory reaction in microglial cells. Besides, trametinib attenuated inflammation factors expression during the early stages of TBI. In addition, trametinib inhibited LPS-induced microglial chemotactic activity. In conclusion, the results indicate that trametinib efficiently suppresses microglia-induced neuroinflammation and improves cognitive function of TBI mice, providing a potential therapy strategy for TBI patients.

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TI Traumatic brain injury-related inflammatory projection: beyond local

inflammatory responses

SO ACUTE MEDICINE & SURGERY

LA English

DT Review

DE Microglia; neurodegeneration; neuroimaging; neuroinflammation; traumatic

brain injury

ID IN-VIVO; MICROGLIAL ACTIVATION; DISRUPTION; DEMENTIA; MODERATE; PROTEIN;

DISEASE; SINGLE

AB Acute neuroinflammation induced by microglial activation is key for repair and recovery after traumatic brain injury (TBI) and could be necessary for the clearance of harmful substances, such as cell debris. However, recent clinical and preclinical data have shown that TBI causes chronic neuroinflammation, lasting many years in some cases, and leading to chronic neurodegeneration, dementia, and encephalopathy. To evaluate neuroinflammation in vivo, positron-emission tomography has been used to target translocator protein, which is upregulated in activated glial cells. Such studies have suggested that remote neuroinflammation induced by regional microglia persists even after reduced inflammatory responses at the injury site. Furthermore, unregulated inflammatory responses are associated with neurodegeneration. Therefore, elucidation of the role of neuroinflammation in TBI pathology is essential for developing new therapeutic targets for TBI. Treatment of associated progressive disorders requires a deeper understanding of how inflammatory responses to injury are triggered, sustained, and resolved and how they impact neuronal function. In this review, we provide a general overview of the dynamics of immune responses to TBI, from acute to chronic neuroinflammation. We discuss the clinical significance of remote ongoing neuroinflammation, termed "brain injury-related inflammatory projection". We also highlight positron-emission tomography imaging as a promising approach needing further development to facilitate an understanding of post-TBI inflammatory and neurodegenerative processes and to monitor the clinical effects of corresponding new therapeutic strategies.

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TI C-terminal binding proteins 1 and 2 in traumatic brain injury-induced

inflammation and their inhibition as an approach for anti-inflammatory

treatment

SO INTERNATIONAL JOURNAL OF BIOLOGICAL SCIENCES

LA English

DT Article

DE CtBP; proinflammatory transcription; neuroinflammation; microglia

activation; traumatic brain injury

ID DIFFUSE AXONAL INJURY; TRANSCRIPTIONAL ACTIVATION; POTENTIAL MECHANISMS;

ADENOVIRUS E1A; HEAD-INJURY; CTBP; NEUROPROTECTION; DIFFERENTIATION;

IDENTIFICATION; ASSOCIATION

AB Traumatic brain injury (TBI) induces an acute inflammatory response in the central nervous system that involves both resident and peripheral immune cells. The ensuing chronic neuroinflammation causes cell death and tissue damage and may contribute to neurodegeneration. The molecular mechanisms involved in the maintenance of this chronic inflammation state remain underexplored. C-terminal binding protein (CtBP) 1 and 2 are transcriptional coregulators that repress diverse cellular processes. Unexpectedly, we find that the CtBPs can transactivate a common set of proinflammatory genes both in lipopolysaccharide-activated microglia, astrocytes and macrophages, and in a mouse model of the mild form of TBI. We also find that the expression of these genes is markedly enhanced by a single mild injury in both brain and peripheral blood leukocytes in a severity- and time-dependent manner. Moreover, we were able to demonstrate that specific inhibitors of the CtBPs effectively suppress the expression of the CtBP target genes and thus improve neurological outcome in mice receiving single and repeated mild TBIs. This discovery suggests new avenues for therapeutic modulation of the inflammatory response to brain injury.

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TI Plasma osteopontin may predict neuroinflammation and the severity of

pediatric traumatic brain injury

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article; Proceedings Paper

CT Journal-of-Cerebral-Blood-Flow-and-Metabolism (JCBFM) Symposium at

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CL Yokohama, JAPAN

SP Journal Cerebral Blood Flow & Metab

DE Diagnostic biomarker; glial fibrillary acidic protein; microglia;

outcomes; predictive biomarker

AB Traumatic brain injury (TBI) is the leading cause of death in children and adolescents in developed countries, but there are no blood-based biomarkers to support the diagnosis or prognosis of pediatric TBI to-date. Here we report that the plasma levels of osteopontin (OPN), a phosphoprotein chiefly secreted by macrophages and/or activated microglia, may contribute to this goal. In animal models of TBI, while OPN, fibrillary acidic protein (GFAP), and matrix metalloproteinase 9 (MMP-9) were all readily induced by controlled cortical impact in the brains of one-month-old mice, only OPN and GFAP ascended in the blood in correlation with high neurological severity scores (NSS). In children with TBI (three to nine years of age, n = 66), the plasma levels of OPN, but not GFAP, correlated with severe TBI (Glasgow Coma Score <= 8) and intracranial lesions at emergency department. In addition, the plasma OPN levels in severe pediatric TBI patients continued to ascend for 72 h and correlated with mortality and the days requiring ventilator or intensive care unit support, whereas the plasma GFAP levels lacked these properties. Together, these results suggest that plasma OPN outperforms GFAP and may be a neuroinflammation-based diagnostic and prognostic biomarker in pediatric TBI.

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TI Interleukin-1 Beta Neutralization Attenuates Traumatic Brain

Injury-Induced Microglia Activation and Neuronal Changes in the Globus

Pallidus

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE traumatic brain injury; interleukin-1 beta; inflammation; Parkinson's

disease; globus pallidus; axonal injury

ID FLUID PERCUSSION INJURY; RECEPTOR ANTAGONIST; PARKINSONS-DISEASE;

ANIMAL-MODELS; HEAD-INJURY; EXPRESSION; INFLAMMATION; PROTEIN; RISK;

ENCEPHALOPATHY

AB Traumatic brain injury (TBI) increases the risk of delayed neurodegenerative processes, including Parkinson's disease (PD). Interleukin-1beta (IL-1 beta), a key pro-inflammatory cytokine, may promote secondary injury development after TBI. Conversely, neutralizing IL-1 beta was found to improve functional recovery following experimental TBI. However, the mechanisms underlying the behavioral improvements observed by IL-1 beta neutralization are still poorly understood. The present study investigated the role of IL-1 beta on the microglia response and neuronal changes in the globus pallidus in response to diffuse TBI. Mice were subjected to sham injury or the central fluid percussion injury (cFPI) (a model of traumatic axonal injury), and were randomly administered an IL-1 beta neutralizing or a control antibody at 30 min post-injury. The animals were analyzed at 2, 7, or 14 days post-injury. When compared to controls, mice subjected to cFPI TBI had increased microglia activation and dopaminergic innervation in the globus pallidus, and a decreased number of parvalbumin (PV) positive interneurons in the globus pallidus. Neutralization of IL-1 beta attenuated the microglia activation, prevented the loss of PV+ interneurons and normalized dopaminergic fiber density in the globus pallidus of brain-injured animals. These findings argue for an important role for neuro-inflammation in the PD-like pathology observed in TBI.

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TI Aberrant ER Stress Induced Neuronal-IFNβ Elicits White Matter Injury Due

to Microglial Activation and T-Cell Infiltration after TBI

SO JOURNAL OF NEUROSCIENCE

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ID TRAUMATIC BRAIN-INJURY; ENDOPLASMIC-RETICULUM STRESS; INTRACEREBRAL

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AB Persistent endoplasmic reticulum (ER) stress in neurons is associated with activation of inflammatory cells and subsequent neuroinflammation following traumatic brain injury (TBI); however, the underlying mechanism remains elusive. We found that induction of neuronal-ER stress, which was mostly characterized by an increase in phosphorylation of a protein kinase R-like ER kinase (PERK) leads to release of excess interferon (IFN)beta due to atypical activation of the neuronal-STING signaling pathway. IFN beta enforced activation and polarization of the primary microglial cells to inflammatory M1 phenotype with the secretion of a proinflammatory chemokine CXCL10 due to activation of STAT1 signaling. The secreted CXCL10, in turn, stimulated the T-cell infiltration by serving as the ligand and chemoattractant for CXCR3(+)T-helper 1 (Th1) cells. The activation of microglial cells and infiltration of Th1 cells resulted in white matter injury, characterized by impaired myelin basic protein and neurofilament NF200, the reduced thickness of corpus callosum and external capsule, and decline of mature oligodendrocytes and oligodendrocyte precursor cells. Intranasal delivery of CXCL10 siRNA blocked Th1 infiltration but did not fully rescue microglial activation and white matter injury after TBI. However, impeding PERK-phosphorylation through the administration of GSK2656157 abrogated neuronal induction of IFN beta, switched microglial polarization to M2 phenotype, prevented Th1 infiltration, and increased Th2 and Treg levels. These events ultimately attenuated the white matter injury and improved anxiety and depressive-like behavior following TBI.

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TI Microglial polarization in posttraumatic epilepsy: Potential mechanism

and treatment opportunity

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ID TRAUMATIC BRAIN-INJURY; MESENCHYMAL STEM-CELLS; ALTERNATIVE ACTIVATION;

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AB Owing to the complexity of the pathophysiological mechanisms driving epileptogenesis following traumatic brain injury (TBI), effective preventive treatment approaches are not yet available for posttraumatic epilepsy (PTE). Neuroinflammation appears to play a critical role in the pathogenesis of the acquired epilepsies, including PTE, but despite a large preclinical literature demonstrating the ability of anti-inflammatory treatments to suppress epileptogenesis and chronic seizures, no anti-inflammatory treatment approaches have been clinically proven to date. TBI triggers robust inflammatory cascades, suggesting that they may be relevant for the pathogenesis of PTE. A major cell type involved in such cascades is the microglial cells-brain-resident immune cells that become activated after brain injury. When activated, these cells can oscillate between different phenotypes, and such polarization states are associated with the release of various pro- and anti-inflammatory mediators that may influence brain repair processes, and also differentially contribute to the development of PTE. As the molecular mechanisms and key signaling molecules associated with microglial polarization in brain are discovered, strategies are now emerging that can modulate this polarization, promoting this as a potential therapeutic strategy for PTE. In this review, we discuss the relevant literature regarding the polarization of brain-resident immune cells following TBI and attempt to put into perspective a role in epilepsy pathogenesis. Finally, we explore potential strategies that could polarize microglia/macrophages toward a neuroprotective phenotype to mitigate PTE development.

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TI Contributions of Interleukin-1 Receptor Signaling in Traumatic Brain

Injury

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DT Review

DE traumatic brain injury; interleukin-1; interleukin-1 receptor; cytokine;

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ID PROTEIN-KINASE-C; CEREBRAL-ISCHEMIA; MESSENGER-RNA; POSTTRAUMATIC

EPILEPSY; PSYCHIATRIC-DISORDERS; BONE-FRACTURE; MOUSE-BRAIN;

HEAD-INJURY; PHASE-II; ANTAGONIST

AB Traumatic brain injury (TBI) in various forms affects millions in the United States annually. There are currently no FDA-approved therapies for acute injury or the chronic comorbidities associated with TBI. Acute phases of TBI are characterized by profound neuroinflammation, a process that stimulates the generation and release of proinflammatory cytokines including interleukin-1 alpha (IL-1 alpha) and IL-1 beta. Both forms of IL-1 initiate signaling by binding with IL-1 receptor type 1 (IL-1R1), a receptor with a natural, endogenous antagonist dubbed IL-1 receptor antagonist (IL-1Ra). The recombinant form of IL-1Ra has gained FDA approval for inflammatory conditions such as rheumatoid arthritis, prompting interest in repurposing these pharmacotherapies for other inflammatory diseases/injury states including TBI. This review summarizes the currently available preclinical and clinical literature regarding the therapeutic potential of inhibiting IL-1-mediated signaling in the context of TBI. Additionally, we propose specific research areas that would provide a greater understanding of the role of IL-1 signaling in TBI and how these data may be beneficial for the development of IL-1-targeted therapies, ushering in the first FDA-approved pharmacotherapy for acute TBI.

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TI Enhanced Akt/GSK-3β/CREB signaling mediates the anti-inflammatory

actions of mGluR5 positive allosteric modulators in microglia and

following traumatic brain injury in male mice

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ID GLUTAMATE-RECEPTOR 5; NF-KAPPA-B; IMPROVES FUNCTIONAL RECOVERY; SYNTHASE

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ACTIVATION; INFLAMMATION

AB We have previously shown that treatment with a mGluR5 positive allosteric modulator (PAM) is neuroprotective after experimental traumatic brain injury (TBI), limiting post-traumatic neuroinflammation by reducing pro-inflammatory microglial activation and promoting anti-inflammatory and neuroprotective responses. However, the specific molecular mechanisms governing this anti-inflammatory shift in microglia remain unknown. Here we show that the mGluR5 PAM, VU0360172 (VuPAM), regulates microglial inflammatory responses through activation of Akt, resulting in the inhibition of GSK-3 beta. GSK-3 beta regulates the phosphorylation of CREB, thereby controlling the expression of inflammation-related genes and microglial plasticity. The anti-inflammatory action of VuPAM in microglia is reversed by inhibiting Akt/GSK-3 beta/CREB signaling. Using a well-characterized TBI model and CX3CR1(gfp/+) mice to visualize microglia in vivo, we demonstrate that VuPAM enhances Akt/GSK-3 beta/CREB signaling in the injured cortex, as well as anti-inflammatory microglial markers. Furthermore, in situ analysis revealed that GFP + microglia in the cortex of VuPAM-treated TBI mice co-express pCREB and the anti-inflammatory microglial phenotype marker YM1. Taken together, our data show that VuPAM decreases pro-inflammatory microglial activation by modulating Akt/GSK-3 beta/CREB signaling. These findings serve to clarify the potential neuroprotective mechanisms of mGluR5 PAM treatment after TBI, and suggest novel therapeutic targets for post-traumatic neuroinflammation.

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TI Disruption of Midkine gene reduces traumatic brain injury through the

modulation of neuroinflammation

SO JOURNAL OF NEUROINFLAMMATION

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DE Midkine; Traumatic brain injury; Microglia; macrophages; M1; M2

phenotype; Neuroinflammation

ID MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS; BINDING GROWTH-FACTOR;

SPINAL-CORD; T-CELLS; INTRACEREBRAL HEMORRHAGE; DEGENERATION;

REGENERATION; PLEIOTROPHIN; MACROPHAGES; DIFFERENTIATION

AB Background Midkine (MK) is a multifunctional cytokine found upregulated in the brain in the presence of different disorders characterized by neuroinflammation, including neurodegenerative disorders and ischemia. The neuroinflammatory response to traumatic brain injury (TBI) represents a key secondary injury factor that can result in further neuronal injury. In the present study, we investigated the role of endogenous MK in secondary injury, including neuroinflammation, immune response, and neuronal apoptosis activity, after TBI. Methods Wild type (Mdk(+/+)) and MK gene deficient (Mdk(-/-)) mice were subjected to fluid percussion injury for TBI models and compared at 3, 7, and 14 days after TBI, in terms of the following: brain tissue loss, neurological deficits, microglia response, astrocytosis, expression of proinflammatory M1 and anti-inflammatory M2 microglia/macrophage phenotype markers, and apoptotic activity. Results As opposed to Mdk(+/+) mice, Mdk(-/-) mice reported a significantly reduced area of brain tissue loss and an improvement in their neurological deficits. The ratios of the Iba1-immunoreactive microglia/macrophages in the perilesional site were significantly decreased in Mdk(-/-) than in the Mdk(+/+) mice at 3 days after TBI. However, the ratios of the glial fibrillary acidic protein immunoreactive area were similar between the two groups. The M1 phenotype marker (CD16/32) immunoreactive areas were significantly reduced in Mdk(-/-) than in the Mdk(+/+) mice. Likewise, the mRNA levels of the M1 phenotype markers (TNF-alpha, CD11b) were significantly decreased in Mdk(-/-) mice than in Mdk(+/+) mice. Furthermore, flow cytometry analysis identified the M2 markers, i.e., CD163(+) macrophages cells and arginase-1(+) microglia cells, to be significantly higher in Mdk(-/-) than in Mdk(+/+) mice. Finally, the ratios of apoptotic neurons were significantly decreased in the area surrounding the lesion in Mdk(-/-) than in Mdk(+/+) mice following TBI. Conclusion Our findings suggest that MK-deficiency reduced tissue infiltration of microglia/macrophages and altered their polarization status thereby reducing neuroinflammation, neuronal apoptosis, and tissue loss and improving neurological outcomes after TBI. Therefore, targeting MK to modulate neuroinflammation may represent a potential therapeutic strategy for TBI management.

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TI Mossy cell hypertrophy and synaptic changes in the hilus following mild

diffuse traumatic brain injury in pigs

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DT Article

DE Mild traumatic brain injury; Concussion; Microglia; Hippocampus; Mossy

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ID DENTATE GYRUS; AXONAL PATHOLOGY; CONTUSION INJURY; WHITE-MATTER; MODEL;

MICROGLIA; NEURONS; DAMAGE; HYPEREXCITABILITY; INFLAMMATION

AB Background Each year in the USA, over 2.4 million people experience mild traumatic brain injury (TBI), which can induce long-term neurological deficits. The dentate gyrus of the hippocampus is notably susceptible to damage following TBI, as hilar mossy cell changes in particular may contribute to post-TBI dysfunction. Moreover, microglial activation after TBI may play a role in hippocampal circuit and/or synaptic remodeling; however, the potential effects of chronic microglial changes are currently unknown. The objective of the current study was to assess neuropathological and neuroinflammatory changes in subregions of the dentate gyrus at acute to chronic time points following mild TBI using an established model of closed-head rotational acceleration induced TBI in pigs. Methods This study utilized archival tissue of pigs which were subjected to sham conditions or rapid head rotation in the coronal plane to generate mild TBI. A quantitative assessment of neuropathological changes in the hippocampus was performed via immunohistochemical labeling of whole coronal tissue sections at 3 days post-injury (DPI), 7 DPI, 30 DPI, and 1 year post-injury (YPI), with a focus on mossy cell atrophy and synaptic reorganization, in context with microglial alterations (e.g., density, proximity to mossy cells) in the dentate gyrus. Results There were no changes in mossy cell density between sham and injured animals, indicating no frank loss of mossy cells at the mild injury level evaluated. However, we found significant mossy cell hypertrophy at 7 DPI and 30 DPI in anterior (> 16% increase in mean cell area at each time; p = < 0.001 each) and 30 DPI in posterior (8.3% increase; p = < 0.0001) hippocampus. We also found dramatic increases in synapsin staining around mossy cells at 7 DPI in both anterior (74.7% increase in synapsin labeling; p = < 0.0001) and posterior (82.7% increase; p = < 0.0001) hippocampus. Interestingly, these morphological and synaptic alterations correlated with a significant change in microglia in proximity to mossy cells at 7 DPI in anterior and at 30 DPI in the posterior hippocampus. For broader context, while we found that there were significant increases in microglia density in the granule cell layer at 30 DPI (anterior and posterior) and 1 YPI (posterior only) and in the molecular layer at 1 YPI (anterior only), we found no significant changes in overall microglial density in the hilus at any of the time points evaluated post-injury. Conclusions The alterations of mossy cell size and synaptic inputs paired with changes in microglia density around the cells demonstrate the susceptibility of hilar mossy cells after even mild TBI. This subtle hilar mossy cell pathology may play a role in aberrant hippocampal function post-TBI, although additional studies are needed to characterize potential physiological and cognitive alterations.

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NR 54

TC 10

Z9 14

U1 0

U2 0

PU BMC

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J9 J NEUROINFLAMM

JI J. Neuroinflamm.

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PT J

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TI Administration of a 20-Hydroxyeicosatetraenoic Acid Synthesis Inhibitor

Improves Outcome in a Rat Model of Pediatric Traumatic Brain Injury

SO DEVELOPMENTAL NEUROSCIENCE

LA English

DT Article

DE 20-Hydroxyeicosatetraenoic acid;

N-hydroxy-N-4-butyl-2-methylphenylformamidine; Microglia;

Neuroinflammation

ID CEREBRAL-BLOOD-FLOW; OXYGEN-GLUCOSE DEPRIVATION; 20-HETE CONTRIBUTES;

HET0016; NEUROPROTECTION; ISCHEMIA; DYSFUNCTION; PLASTICITY; EDEMA

AB The arachidonic acid pathway metabolite 20-hydroxyeicosatetraenoic acid (20-HETE) contributes to ischemia/reperfusion brain injury. Inhibition of 20-HETE formation can protect the developing brain from global ischemia. Here, we examined whether treatment with the 20-HETE synthesis inhibitor N-hydroxy-N-4-butyl-2-methylphenylformamidine (HET0016) can protect the immature brain from traumatic brain injury (TBI). Male rats at postnatal day 9-10 underwent controlled cortical impact followed by intraperitoneal injection with vehicle or HET0016 (1 mg/kg, 5 min and 3 h post-injury). HET0016 decreased the lesion volume by over 50% at 3 days of recovery, and this effect persisted at 30 days as the brain matured. HET0016 decreased peri-lesion gene expression of proinflammatory cytokines (tumor necrosis factor-alpha [TNF-alpha], interleukin-1 beta [IL-1 beta]) at 1 day and increased reparative cytokine (IL-4, IL-10) expression at 3 days. It also partially preserved microglial ramified processes, consistent with less activation. HET0016 decreased contralateral hindlimb foot faults and improved outcome on the novel object recognition memory task 30 days after TBI. In cultured BV2 microglia, HET0016 attenuated the lipopolysaccharide-evoked increase in release of TNF-alpha. Our data show that HET0016 improves acute and long-term histologic and functional outcomes, in association with an attenuated neuroinflammatory response after contusion of an immature rat brain.

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FU Chongqing Science and Technology Commission grants of China

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NR 47

TC 14

Z9 14

U1 1

U2 4

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J9 DEV NEUROSCI-BASEL

JI Dev. Neurosci.

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PT J

AU Qi, R

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AF Qi, Ruo

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TI Inhibition of miR-429 improves neurological recovery of traumatic brain

injury mice and attenuates microglial neuroinflammation

SO INTERNATIONAL IMMUNOPHARMACOLOGY

LA English

DT Article

DE Microglia; miR-429; Neuroinflammation; Traumatic brain injury; DUSP1

ID DUSP1; MAPK; PHOSPHATASE; CONTRIBUTES; APOPTOSIS; TARGET

AB Background: Neuroinflammation is a common therapeutic target for traumatic brain injury (TBI) due to its contribution to delayed secondary cell death and has the potential to occur for years after the initial insult. Previous studies demonstrate that miR-429 is up-regulated in the brain lesions of TBI mice, while its role in regulating neuroinflammation and brain injury remains largely unknown.

Method: The expression of miR-429 in LPS-activated microglia and microglia in TBI model was detected by RTPCR. The effects of miR-429 inhibitors on LPS-activated microglia in vitro as well as neurological recovery and post-traumatic neuroinflammatory response in TBI model mice were detected in vivo.

Results: LPS and TBI significantly induce the up-expression of miR-429, inflammatory cytokines, MAPK-p38 and phosphorylated NF-kappa B in microglia, which were all inhibited by miR-429 inhibitors. Meanwhile, miR-429 inhibitors also attenuated the neurological impairment in TBI mice. Btoinformatics analysis showed that miR-429 could target and inhibit the expression of dual specificity protein phosphatase 1 (DUSP1), thus inhibiting the expression of MAPK-p38 and phosphorylated NF-kappa B.

Conclusion: miR-429 plays a pro-inflammatory role in activated microglia by targeting DUSP1 signaling pathway. Inhibiting miR-429 can attenuate the inflammatory response of microglia and TBI-mediated brain damage.

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NR 33

TC 15

Z9 17

U1 1

U2 9

PU ELSEVIER

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PT J

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TI Cortical Spreading Depression in the Setting of Traumatic Brain Injury

SO WORLD NEUROSURGERY

LA English

DT Review

DE Astrocytes; Calcium signaling; Cortical spreading depression; Microglia;

Spreading depolarization; Traumatic brain injury

ID ANOXIC DEPOLARIZATION; NITRIC-OXIDE; IN-VIVO; PERSPECTIVES; TRIGGERS;

LACTATE; TARGETS; GLUCOSE; STROKE; DAMAGE

AB Cortical spreading depression (CSD) is a pathophysiologic phenomenon that describes an expanding wave of depolarization within the cortical gray matter. Originally described over 70 years ago, this spreading depression disrupts neuronal and glial ionic equilibrium, leading to increased energy demands that can cause a metabolic crisis. This results in secondary insult, further perpetuating brain injury and neuronal death. Initially not thought to be of clinical significance, the view of CSD was modified with the advent of intracranial electroencephalography, or electrocorticography. With these improved monitoring techniques, CSD has been identified as a major mechanism by which traumatic brain injury (TBI) imparts its negative sequalae. TBI is a heterogenous disease process that runs the gamut of clinical presentations. This includes concussion, epidural and subdural hematoma, diffuse axonal injury, and subarachnoid hemorrhage. Nonetheless, CSD appears to be frequently occurring among the various types of TBI, thus allowing for the potential development of targeted therapies in an otherwise ill-fated patient cohort. Although a complete understanding of the interplay between CSD and TBI has not yet been achieved, the authors recount the efforts that have been employed over the last several decades in an effort to bridge this gap. In addition, our current understanding of the role neuroimmune cells play in CSD is discussed in the context of TBI. Finally, current therapeutic strategies using CSD as a pharmacologic target are explored with respect to their clinical use in patients with TBI.

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AF Jiang, Qian

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Zou, Xin

Yang, Yiping

Huang, Guangying

Zhang, Huaqiu

TI Phillyrin protects mice from traumatic brain injury by inhibiting the

inflammation of microglia via PPARγ signaling pathway

SO INTERNATIONAL IMMUNOPHARMACOLOGY

LA English

DT Article

DE Microglia; Phillyrin; Inflammation; Traumatic brain injury; PPAR gamma

ID NF-KAPPA-B; OXIDATIVE STRESS; WHITE-MATTER; ROSIGLITAZONE; ACTIVATION;

NEUROINFLAMMATION; POLARIZATION; DEGENERATION; EXPRESSION; PHENOTYPE

AB The neuroinflammatory response induced by microglia plays a vital role in causing secondary brain damage after traumatic brain injury (TBI). Previous studies have found that the improved regulation of activated microglia could reduce neurological damage post-TBI. Phillyrin (Phi) is one of the main active ingredients extracted from the fruits of the medicinal plant Forsythia suspensa (Thunb.) with anti-inflammatory effects. Our study attempted to investigate the effects of phillyrin on microglial activation and neuron damage after TBI. The TBI model was applied to induce brain injury in mice, and neurological scores, brain water content, hematoxylin and eosin staining and Nissl staining were employed to determine the neuroprotective effects of phillyrin. Immunofluorescent staining and western blot analysis were used to detect nuclear factor-kappa B (NF-kappa B) and peroxisome proliferator-activated receptor gamma (PPAR gamma) expression and nuclear translocation, and the inflammation-related proteins and mRNAs were assessed by western blot analysis and quantitative real-time PCR. The results revealed that phillyrin not only inhibited the proinflammatory response induced by activated microglia but also attenuated neurological impairment and brain edema in vivo in a mouse TBI model. Additionally, phillyrin suppressed the phosphorylation of NF-kappa B in microglia after TBI insult. These effects of phillyrin were mostly abolished by the antagonist of PPAR gamma. Our results reveal that phillyrin could prominently inhibit the inflammation of microglia via the PPAR gamma signaling pathway, thus leading to potential neuroprotective treatment after traumatic brain injury.

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TI Microglia Adopt Longitudinal Transcriptional Changes After Traumatic

Brain Injury

SO JOURNAL OF SURGICAL RESEARCH

LA English

DT Article

DE TBI; Microglia; FACS; RNA Sequencing

ID ALZHEIMERS-DISEASE; MYELOID CELLS; ACTIVATION; ETIOLOGY; MODEL; MILD

AB Background: Traumatic brain injury (TBI) is an under-recognized public health threat. Even mild brain injuries can lead to long-term neurologic impairment. Microglia play a fundamental role in the development and progression of this ensuing neurologic impairment. Despite this, a microglia-specific injury signature has yet to be identified. We hypothesized that TBI would lead to long-term changes in the transcriptional profile of microglial pathways associated with the development of subsequent neurologic impairment.

Materials and methods: Male C57BL/6 mice underwent TBI via a controlled cortical impact and were followed longitudinally. FACSorted microglia from TBI mice were subjected to Quantiseq 3'-biased RNA sequencing at 7, 30, and 90 d after TBI. K-means clustering on 396 differentially expressed genes was performed, and gene ontology enrichment analysis was used to determine corresponding enriched processes.

Results: Differentially expressed genes in microglia exhibited four main patterns of expression over the course of TBI. In particular, we identified four gene clusters which corresponded to the host defense response, synaptic plasticity, lipid remodeling, and membrane polarization.

Conclusions: Transcriptional profiling within individual populations of microglia after TBI remains a critical unmet research need within the field of TBI. This focused study identified several physiologic processes within microglia that may be associated with development of long-termneurologic impairment after TBI. These data demonstrate the capability of longitudinal transcriptional profiling to uncover potential cell-specific targets for the treatment of TBI. (C) 2019 Elsevier Inc. All rights reserved.

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TI Brain-derived neurotrophic factor fused with a collagen-binding domain

inhibits neuroinflammation and promotes neurological recovery of

traumatic brain injury mice via TrkB signalling

SO JOURNAL OF PHARMACY AND PHARMACOLOGY

LA English

DT Article; Early Access

DE Brain-derived neurotrophic factor fused with a collagen-binding domain;

inflammation; microglia; traumatic brain injury; TrkB

ID NF-KAPPA-B; FACTOR BDNF; NEUROPROTECTION; POLARIZATION; INFLAMMATION;

PROTECTION; MICROGLIA; BDNF/TRKB; PATHWAY

AB Objectives As one of the vital nutrient factors in central nervous system (CNS), brain-derived neurotrophic factor (BDNF) can significantly attenuate neuron damage and promote neurogenesis. Nevertheless, little research has been conducted on regulating the effect of BDNF on the inflammatory response after traumatic brain injury (TBI).

Methods In this study, we used BDNF fused with a collagen-binding domain (CBD-BDNF) to maintain a sufficient concentration of BDNF in the TBI hemisphere, and then, the regulatory effects of BDNF and CBD-BDNF on the inflammatory response of microglia were investigated both on a TBI mice model in vivo and LPS-stimulated microglia experiment in vitro.

Key findings The results revealed that BDNF and CBD-BDNF had similar effects on attenuating the pro-inflammatory reactions but promoting anti-inflammatory responses of microglia induced by LPS in vitro. Furthermore, CBD-BDNF significantly improved the neurological behaviours of TBI mice and alleviated the inflammatory reaction after TBI, while BDNF had weaker effects compared with those of CBD-BDNF. Additionally, the TrkB inhibitor K252a significantly inhibited the above effects of CBD-BDNF.

Conclusions In conclusion, CBD-BDNF can promote the anti-inflammatory function of microglia and neurological recovery of TBI mice through TrkB signalling.

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PT J

AU Soriano, S

Moffet, B

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Villapol, S

AF Soriano, Sirena

Moffet, Bridget

Wicker, Evan

Villapol, Sonia

TI Serum Amyloid A is Expressed in the Brain After Traumatic Brain Injury

in a Sex-Dependent Manner

SO CELLULAR AND MOLECULAR NEUROBIOLOGY

LA English

DT Article; Early Access

DE Neuroinflammation; Brain trauma; Acute phase response; Microglia;

Neutrophil accumulation

ID ALZHEIMERS-DISEASE; SYSTEMIC INFLAMMATION; PROTEIN; CELLS;

NEUROINFLAMMATION; CONSEQUENCES; ACTIVATION; DEPOSITION; MICROGLIA;

CYTOKINES

AB Serum amyloid A (SAA) is an acute phase protein upregulated in the liver after traumatic brain injury (TBI). So far, it has not been investigated whether SAA expression also occurs in the brain in response to TBI. For this, we performed a moderate controlled cortical impact injury in adult male and female mice and analyzed brain, blood, and liver samples at 6 h, 1, 3, and 10 days post-injury (dpi). We measured the levels of SAA in serum, brain and liver by western blot. We also used immunohistochemical techniques combined with in situ hybridization to determine SAA mRNA and protein expression in the brain. Our results revealed higher levels of SAA in the bloodstream in males compared to females at 6 h post-TBI. Liver and serum SAA protein showed a peak of expression at 1 dpi followed by a decrease at 3 to 10 dpi in both sexes. Both SAA mRNA and protein expression colocalize with astrocytes and macrophages/microglia in the cortex, corpus callosum, thalamus, and hippocampus after TBI. For the first time, here we show that SAA is expressed in the brain in response to TBI. Collectively, SAA expression was higher in males compared to females, and in association with the sex-dependent neuroinflammatory response after brain injury. We suggest that SAA could be a crucial protein associated to the acute neuroinflammation following TBI, not only for its hepatic upregulation but also for its expression in the injured brain.

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Houston Methodist Research Institute.

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TI Intravenously Infusing the Secretome of Adipose-Derived Mesenchymal Stem

Cells Ameliorates Neuroinflammation and Neurological Functioning After

Traumatic Brain Injury

SO STEM CELLS AND DEVELOPMENT

LA English

DT Article

DE traumatic brain injury; secretome; neuroinflammation; the polarization

of microglia; neurological function

ID MACROPHAGES; EDEMA; NEUROPROTECTION; INFLAMMATION; POLARIZATION;

MICROGLIA; MODULATE; STROKE; RATS

AB The secretome of mesenchymal stem cell (MSC) offers a series of immunoregulatory properties and is regarded as an effective method of mitigating secondary neuroinflammation induced by traumatic brain injury (TBI). The secretome of adipose-derived MSCs (ASC-ST) was collected under hypoxia conditions. Proteomics data were analyzed by liquid chromatography-tandem mass spectrometry (LC-MS/MS), and concentrations of major components were tested. After the TBI caused by an electric cortical contusion impactor, rats were injected ASC-ST through caudal veins for 7 days. The neurological functional prognosis of TBI rats was significantly improved, and the vasogenic edema of brain tissues that was measured 14 days after TBI was relieved by ASC-ST, corresponding to brain water content levels. ASC-ST ameliorated TBI-induced neuroinflammatory environments that caused the edema, the apoptosis of the neural cells, and the nerve fiber damage by increasing the number of M2 phenotypes present while reducing the number of M1 phenotype microglia present. Furthermore, interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-alpha) levels were reduced, whereas transforming growth factor-beta (TGF-beta) and tumor necrosis factor-stimulated gene 6 protein (TSG-6) levels were increased after secretome treatment. Altogether, ASC-ST is capable of improving neural functioning by modulating TBI-induced neuroinflammation and its related secondary insults. ASC-ST may be one of the most promising candidates for regulating the secondary inflammatory reactions of central nervous systems for clinical use.

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yi/HOF-6668-2023; wang, KiKi/JFZ-3334-2023

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U2 28

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WC Cell & Tissue Engineering; Hematology; Medicine, Research &

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AU Caplan, HW

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AF Caplan, Henry W.

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Zelnick, Pamela

Xue, Hasen

Cox, Charles S.

Bedi, Supinder S.

TI Spatiotemporal Distribution of Microglia After Traumatic Brain Injury in

Male Mice

SO ASN NEURO

LA English

DT Article

DE cell death; inflammation; microglia; neuro degeneration; neuro glia;

neuro immunity; neuro repair; TBI; therapies

ID ACTIVATED MICROGLIAL/MACROPHAGE RESPONSE; UNITED-STATES; THERAPY;

GENDER; SEX; SURVEILLANCE; RECRUITMENT; EXPRESSION; RECEPTOR; IMPACTS

AB Traumatic brain injury (TBI) disrupts the complex arrangement of glia and neuronal cells in the central nervous system. Microglia, the resident immune cells, survey the cellular milieu under homeostatic conditions and play a neuroprotective role via clearance of dead cells and debris such as axons and myelin. Resting (ramified) microglia possess a distinct morphology-small rod-shaped somata with thin processes. After TBI, microglia are activated and transition into an amoeboid morphology. To delineate the spatiotemporal morphological response of microglia after TBI, we used a controlled cortical impact injury model to quantify and characterize microglia at 24 hr and 28 days after TBI in the hippocampus (H) and lateral posterior nucleus of the thalamus (LPNT). Increased numbers of microglia were observed in the H and LPNT at 28 days after controlled cortical impact, but not at 24 hr in comparison to controls. Spatially, controlled cortical impact resulted in an increase of amoeboid microglia bilaterally at 24 hr and 28 days in H and ipsilaterally in LPNT. Temporally, at 28 days, TBI resulted in a significant increase in the number of amoeboid microglia in both H and LPNT. In addition, at 28 days after injury, we observed an increase in translocator protein, a marker for activated microglia, in the ipsilateral thalamus only. TBI results in a spatiotemporal increase in amoeboid microglia in the hippocampus and the LPNT over 28 days. Delineating their spatiotemporal phenotype is critical because it can help identify therapeutic targets with appropriate therapy.

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TI Sustained neuronal and microglial alterations are associated with

diverse neurobehavioral dysfunction long after experimental brain injury

SO NEUROBIOLOGY OF DISEASE

LA English

DT Article

DE Chronic traumatic brain injury; Neurodegeneration; Neuroinflammation;

Cognition; Depression; Microglia

ID UNITED-STATES; FACS; PREVALENCE; BEHAVIOR; ATROPHY; ANXIETY; STRESS;

SINGLE; TASKS; YOUNG

AB Traumatic brain injury (TBI) can cause progressive neurodegeneration, sustained neuroinflammation and chronic neurological dysfunction. Few experimental studies have explored the long-term neurobehavioral and functional cellular changes beyond several months. The present study examined the effects of a single moderate-level TBI on functional outcome 8 months after injury. Male C57BL/6 mice were subjected to controlled cortical impact injury and followed for changes in motor performance, learning and memory, as well as depressive-like and social behavior. We also used a novel flow cytometry approach to assess cellular functions in freshly isolated neurons and microglia from the injured tissue. There were marked and diverse, sustained neurobehavioral changes in injured mice. Compared to sham controls, chronic TBI mice showed long-term deficits in gait dynamics, nest building, spatial working memory and recognition memory. The tail suspension, forced swim, and sucrose consumption tests showed a marked depressive-like phenotype that was associated with impaired sociability. At the cellular level, there were lower numbers of Thy1 (+) Tuj1 (+) neurons and higher numbers of activated CD45(lo)CD11b (+) microglia. Functionally, both neurons and microglia exhibited significantly higher levels of oxidative stress after injury. Microglia exhibited chronic deficits in phagocytosis of E. coin bacteria, and increased uptake of myelin and dying neurons. Living neurons showed decreased expression of synaptophysin and postsynaptic density (PSD)-95, along with greater numbers of microtubule-associated protein light chain 3 (LC3)-positive autophagosomes and increased mitochondrial mass that suggest dysregulation of autophagy. In summary, the late neurobehavioral changes found after murine TBI are similar to those found chronically after moderate-severe human head injury. Importantly, such changes are associated with microglial dysfunction and changes in neuronal activity.

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TI Temporal evolution of heme oxygenase-1 expression in reactive astrocytes

and microglia in response to traumatic brain injury

SO BRAIN HEMORRHAGES

LA English

DT Article

DE Astrocytes; Heme oxygenase-1; Microglia; Traumatic brain injury

ID RAT-BRAIN; TRANSCRIPTIONAL ACTIVATION; CARBON-MONOXIDE; INDUCTION;

PROTEIN; NEURONS; HO-1; GENE; IMPACT

AB Heme oxygenase-1 (HO-1) is an inducible enzyme that catabolizes heme into biliverdin (which is converted to bilirubin), carbon monoxide, and free iron. HO-1 and its downstream molecules have antioxidant and anti-inflammatory functions, making the effects of HO-1 difficult to predict. It is unknown if HO-1 expression has neuroprotective or neurodegenerative sequelae after traumatic brain injury (TBI). In adult male mice, we quantitatively investigated HO-1 expression in reactive astrocytes and microglia in a controlled cortical impact (CCI) model of TBI at 1, 7, 14, and 30 days post-injury (dpi). Immunoglobulin G (IgG) staining for blood-brain barrier (BBB) permeability was significantly increased at 1 and 7 dpi in TBI mice compared to controls. HO-1 expression in astrocytes was significantly increased acutely and sub-acutely (1, 7, 14 dpi) compared to controls. Significantly elevated expression of HO-1 in microglia was only observed at 14 and 30 dpi relative to controls. HO-1 expression remained elevated at 30 dpi following TBI relative to controls. This study for the first time demonstrates that HO-1 is highly expressed in perilesional tissues after TBI, but primarily in cells that contribute to the neuroinflammatory response. Modulating HO-1 expression may provide a path to therapeutic intervention by enhancing the neuroprotective aspects of HO-1.Summary statement:: Heme oxygenase-1 expression after traumatic brain injury in adult male mice occurs first in reactive astrocytes followed by dramatic increases in microglia adjacent to the injury site. (c) 2020 Production and hosting by Elsevier B.V. on behalf of KeAi Communications Co., Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

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TI Pathological Links between Traumatic Brain Injury and Dementia:

Australian Pre-Clinical Research

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article

DE AD; CTE; microglia; plaques; Tau; traumatic brain injury dementia

ID AMYLOID PRECURSOR PROTEIN; DIFFUSE AXONAL INJURY; ALZHEIMERS-DISEASE;

HEAD-INJURY; DYSTROPHIC NEURITES; SODIUM SELENATE; BETA ACCUMULATION;

TAU PATHOLOGY; NEUROFIBRILLARY TANGLES; HYPERPHOSPHORYLATED TAU

AB Traumatic brain injury (TBI) can cause persistent cognitive changes and ongoing neurodegeneration in the brain. Accumulating epidemiological and pathological evidence implicates TBI in the development of Alzheimer's disease, the most common cause of dementia. Further, the TBI-induced form of dementia, called chronic traumatic encephalopathy, shares many pathological hallmarks present in multiple different diseases which cause dementia. The inflammatory and neuritic responses to TBI and dementia overlap, indicating that they may share common pathological mechanisms and that TBI may ultimately cause a pathological cascade culminating in the development of dementia. This review explores Australian pre-clinical research investigating the pathological links between TBI and dementia.

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TI Podoplanin influences the inflammatory phenotypes and mobility of

microglia in traumatic brain injury

SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS

LA English

DT Article

DE Podoplanin; Traumatic brain injury; Microglia; Matrix metalloproteinases

ID WHITE-MATTER; ACTIVATION

AB Traumatic brain injury (TBI) represents a major cause of death and disability worldwide. Exacerbated neuroinflammation following TBI causes secondary injury. Podoplanin (PDPN) is a small transmembrane mucin-like glycoprotein that promotes the inflammatory response in different tissues and cells. However, the contribution of PDPN to neuroinflammation and microglial activation is unknown. Here, we found that PDPN was correlated with microglial activation after TBI in mice. Meanwhile, PDPN expression could be induced by trauma-related stimuli, such as lipopolysaccharide (LPS), ATP, H2O2 and hemoglobin (Hb), in primary microglia. Furthermore, with Hb treatment in vitro, knockdown of PDPN could decrease the proportion of Ml-like microglia and increase the proportion of M2-like microglia via reduced secretion of IL-1 beta and TNF-alpha and increased secretion of IL-10 and TGF-I beta compared to the control microglia. Immunofluorescence also showed that CD86-positive microglia were decreased and CD206-positive microglia were elevated in the PDPN-KD group. Additionally, PDPN knockdown impaired microglial mobility and phagocytosis and decreased the expression of matrix metalloproteinases (mainly MMP2 and MMP9). In summary, PDPN plays an important role in microglia-mediated inflammation and may serve as a potential target for TBI treatment. (C) 2019 Elsevier Inc. All rights reserved.

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AU Si, LL

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Wang, LY

AF Si, Lili

Wang, Haifeng

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TI Suppression of miR-193a alleviates neuroinflammation and improves

neurological function recovery after traumatic brain injury (TBI) in

mice

SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS

LA English

DT Article

DE Traumatic brain injury (TBI); miR-193a; Neuroinflammation; microglial

activation; NLRP3

ID NLRP3 INFLAMMASOME; ACTIVATION; MODELS; DAMAGE

AB Traumatic brain injury (TBI) is a leading cause of morbidity and mortality in the world, and is tightly associated with microglia-regulated neuroinflammation. However, the activation profile of microglia during the pathophysiological responses is still not fully understood. Micro-RNAs (miRs), as noncoding RNAs, are involved in the progression of TBI. In this study, we attempted to explore the effects of miR-193a on TBI using the in vivo and in vitro studies. Following experimental TBI in mice, we found that miR-193a expression was significantly up-regulated in ipsilateral cortical tissues and in the microglia/ macrophages isolated from the ipsilateral cortical tissues, which was accompanied with markedly enhanced expression of pro-inflammatory factors. We then found that miR-193a hairpin inhibitor (antagomir) markedly reduced lesion volume, brain water contents and neuron death in TBI mice induced by the controlled cortical impact (CCI). In addition, cognitive dysfunction in TBI mice was markedly improved after miR-193a antagomir injection. Of note, CCI-induced activation of microglia was repressed by miR-193a inhibition, along with significantly reduced expression of neuroinflammatory markers, which were associated with the blockage of nucleotide-binding oligomerization domain-like receptor family pyrin domain-containing 3 (NLRP3) inflammasome. The anti-neuroinflammation effects of miR-193a suppression were verified in lipopolysaccharide (LPS)-incubated microglial cells transfected with miR-193a inhibitor. In contrast, LPS-induced activation of microglial cells and the expression of pro-inflammatory factors was markedly further accelerated by the transfection of miR-193a mimic. Taken together, TBI resulted in a robust neuroinflammatory response that was closely associated with the up-regulated miR-193a expression mainly in microglia/macrophages; however, miR-193a suppression significantly alleviated post-traumatic neuroinflammation and cognitive dysfunction. Therefore, miR-193a might be a promising therapeutic target for the treatment of TBI-associated neuroinflammation. (C) 2019 Published by Elsevier Inc.

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Yang, YP

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Tian, WD

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AF Long, Xiaobing

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TI Astrocyte-derived exosomes enriched with miR-873a-5p inhibit

neuroinflammation via microglia phenotype modulation after traumatic

brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Exosome; Traumatic brain injury; Microglia; Astrocyte; M1; M2;

miR-873a-5p

ID CONTRIBUTES; GROWTH

AB Background The interaction between astrocytes and microglia plays a vital role in the damage and repair of brain lesions due to traumatic brain injury (TBI). Recent studies have shown that exosomes act as potent mediators involved in intercellular communication. Methods In the current study, the expression of inflammatory factors and miR-873a-5p in the lesion area and oedema area was evaluated in 15 patients with traumatic brain injury. Exosomes secreted by astrocytes were detected by immunofluorescence, Western blot and electron microscopy. A mouse model of TBI and an in vitro model of LPS-induced primary microglia were established to study the protective mechanism of exosomes from miR-873a-5p overexpressing in TBI-induced nerve injury. Results We discovered that exosomes derived from activated astrocytes promote microglial M2 phenotype transformation following TBI. More than 100 miRNAs were detected in these astrocyte-derived exosomes. miR-873a-5p is a major component that was highly expressed in human traumatic brain tissue. Moreover, miR-873a-5p significantly inhibited LPS-induced microglial M1 phenotype transformation and the subsequent inflammation through decreased phosphorylation of ERK and NF-kappa B p65. This effect also greatly improved the modified neurological severity score (mNSS) and attenuated brain injury in a strictly controlled cortical impact mouse model. Conclusions Taken together, our research indicates that miRNAs in the exosomes derived from activated astrocytes play a key role in the astrocyte-microglia interaction. miR-873a-5p, as one of the main components of these astrocyte-derived exosomes, attenuated microglia-mediated neuroinflammation and improved neurological deficits following TBI by inhibiting the NF-kappa B signalling pathway. These findings suggest a potential role for miR-873a-5p in treating traumatic brain injury.

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TI Diagnostic and Therapeutic Potential of TSPO Studies Regarding

Neurodegenerative Diseases, Psychiatric Disorders, Alcohol Use

Disorders, Traumatic Brain Injury, and Stroke: An Update

SO CELLS

LA English

DT Review

DE brain disorders; brain disease; TSPO; microglia; astrocytes; neurons;

microglia activation; cell death; regeneration; PET tracing; drug

development

ID TRANSLOCATOR-PROTEIN TSPO; PERIPHERAL BENZODIAZEPINE-RECEPTOR; 18 KDA

TSPO; POSITRON-EMISSION-TOMOGRAPHY; MICROGLIAL ACTIVATION; IN-VIVO;

ALZHEIMERS-DISEASE; PERMEABILITY TRANSITION; NERVOUS-SYSTEM; MOUSE MODEL

AB Neuroinflammation and cell death are among the common symptoms of many central nervous system diseases and injuries. Neuroinflammation and programmed cell death of the various cell types in the brain appear to be part of these disorders, and characteristic for each cell type, including neurons and glia cells. Concerning the effects of 18-kDa translocator protein (TSPO) on glial activation, as well as being associated with neuronal cell death, as a response mechanism to oxidative stress, the changes of its expression assayed with the aid of TSPO-specific positron emission tomography (PET) tracers' uptake could also offer evidence for following the pathogenesis of these disorders. This could potentially increase the number of diagnostic tests to accurately establish the stadium and development of the disease in question. Nonetheless, the differences in results regarding TSPO PET signals of first and second generations of tracers measured in patients with neurological disorders versus healthy controls indicate that we still have to understand more regarding TSPO characteristics. Expanding on investigations regarding the neuroprotective and healing effects of TSPO ligands could also contribute to a better understanding of the therapeutic potential of TSPO activity for brain damage due to brain injury and disease. Studies so far have directed attention to the effects on neurons and glia, and processes, such as death, inflammation, and regeneration. It is definitely worthwhile to drive such studies forward. From recent research it also appears that TSPO ligands, such as PK11195, Etifoxine, Emapunil, and 2-Cl-MGV-1, demonstrate the potential of targeting TSPO for treatments of brain diseases and disorders.

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TI Microglial Depletion with CSF1R Inhibitor During Chronic Phase of

Experimental Traumatic Brain Injury Reduces Neurodegeneration and

Neurological Deficits

SO JOURNAL OF NEUROSCIENCE

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DT Article

DE CSF1R; functional recovery; microglia; neurodegeneration;

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ID NADPH OXIDASE; NLRP3 INFLAMMASOME; PROGENITOR-CELL; NEURONAL LOSS;

NEUROINFLAMMATION; ACTIVATION; EXPRESSION; DEMENTIA; PREVENTS; RISK

AB Chronic neuroinflammation with sustained microglial activation occurs following severe traumatic brain injury (TBI) and is believed to contribute to subsequent neurodegeneration and neurological deficits. Microglia, the primary innate immune cells in brain, are dependent on colony stimulating factor 1 receptor (CSF1R) signaling for their survival. In this preclinical study, we examined the effects of delayed depletion of chronically activated microglia on functional recovery and neurodegeneration up to 3 months postinjury. A CSF1R inhibitor, Plexxikon (PLX) 5622, was administered to adult male C57BL/6J mice at 1 month after controlled cortical impact to remove chronically activated microglia, and the inhibitor was withdrawn 1-week later to allow for microglial repopulation. Following TBI, the repopulated microglia displayed a ramified morphology similar to that of Sham uninjured mice, whereas microglia in vehicle-treated TBI mice showed the typical chronic posttraumatic hypertrophic morphology. PLX5622 treatment limited TBI-associated neuropathological changes at 3 months postinjury; these included a smaller cortical lesion, reduced hippocampal neuron cell death, and decreased NOX2-and NLRP3 inflammasome-associated neuroinflammation. Furthermore, delayed depletion of chronically activated microglia after TBI led to widespread changes in the cortical transcriptome and altered gene pathways involved in neuroinflammation, oxidative stress, and neuroplasticity. Using a variety of complementary neurobehavioral tests, PLX5622-treated TBI mice also had improved long-term motor and cognitive function recovery through 3 months postinjury. Together, these studies demonstrate that chronic phase removal of neurotoxic microglia after TBI using CSF1R inhibitors markedly reduce chronic neuroinflammation and associated neurodegeneration, as well as related motor and cognitive deficits.

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Z9 179

U1 12

U2 53

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Chen Shulian

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TI Acupuncture stimulation of Yamen (GV 15), Fengfu (GV 16), Baihui (GV

20), Shuigou (GV 26) and Hegu (LI 4) reduces brain microglia activation

in a traumatic brain injury rat model

SO JOURNAL OF TRADITIONAL CHINESE MEDICINE

LA English

DT Article

DE Acupuncture; Brain injuries; traumatic; Microglia; Lysophospholipids;

Receptors; lysophosphatidic acid; Neuroinflammation

ID LYSOPHOSPHATIDIC ACID; NEUROINFLAMMATION; EXPRESSION

AB OBJECTIVE: To evaluate the effect of acupuncture on neuroinflammation in traumatic brain injury (TBI) rats by stimulating Yamen (GV 15), Fengfu (GV 16), Baihui (GV 20), Shuigou (GV 26) and Hegu (LI 4) acupoints and to investigate the mechanism underpinning this effect.

METHODS: A TBI model was induced in Sprague-Dawley rats using Feeney's freefall impact method. Acupuncture to stimulate the Yamen (GV 15), Fengfu (GV 16), Baihui (GV 20), Shuigou (GV 26) and Hegu (LI 4) acupoints was performed on the TBI rats. After 3 consecutive days of acupuncture treatment, we investigated signal molecules, receptors and microglia related to neuroinflammation in brain tissue of the TBI rats and analyzed the possible mechanism underlying the effect of acupuncture on neuroinflammation.

RESULTS: After the acupuncture treatment, ionized calcium binding adaptor molecule 1(lba1), a protein specific to microglia, was investigated. In the cortical layer of damaged brain tissue in TBI rats, the Iba1-positive area was 3.3% +/- 0.9% in the rats that received acupuncture compared with 5.2% +/- 1.4% in the TBI rats that did not receive acupuncture, and the microglia were smaller with more slender protrusions in the acupuncture-treated rats. This result indicates that acupuncture can significantly reduce microglia activation in TBI rats. A possible mechanism for this effect is that acupuncture reduces the expression of autotaxin and lysophosphatidic acid. Together, these constitute the autotaxin-lysophosphatidic acid axis, which induces microglial activation in the brains of TBI rats. Acupuncture treatment may downregulate the expression of Lysophosphatidic acid (LPA) receptor (LPAR) 1 and LPAR2 on the microglial cytomembrane, which affects the microglia activation process.

CONCLUSION: Acupuncture stimulating the Yamen (GV 15), Fengfu (GV 16), Baihui (GV 20), Shuigou (GV 26) and Hegu (LI 4) acupoints can effectively inhibit the development of neuroinflammation after TBI. One possible mechanism for this effect is that acupuncture downregulates LPA synthesis and affects the LPA-LPAR pathway by inhibiting LPAR1 and LPAR2, thereby inhibiting microglial activation and reducing neuroinflammation. (C) 2020 JTCM. All rights reserved.

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NR 30

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Z9 6

U1 0

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PU JOURNAL TRADITIONAL CHINESE MED

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AU Gan, DQ

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TI Application of the Zebrafish Traumatic Brain Injury Model in Assessing

Cerebral Inflammation

SO ZEBRAFISH

LA English

DT Article

DE zebrafish; traumatic brain injury; microglia; inflammation; Cytidine 5

'-Diphosphocholine

ID MACROPHAGE POLARIZATION; CDP-CHOLINE; WILD-TYPE; INCREASE;

INTERLEUKIN-1; ACTIVATION; RESPONSES; PROTECTS; MUTANT; ROLES

AB Traumatic brain injury (TBI) is a major public and socioeconomic problem throughout the world. The establishment of an effective and cost-effective TBI model for developing new therapeutic agents is challenging. Microglia are considered the resident macrophages of the central nervous system (CNS) that normally do not enter the brain. As the primary mediators of the innate immune response in the CNS, microglia play a critical role in neuroinflammation and secondary injury after TBI. In this study, we established an in vivo TBI zebrafish model using Tg(coro1a:EGFP) line where the green fluorescent protein-labeled microglia were present. We demonstrated that microglia accumulated rapidly in response to neuronal injuries. To clear away injured neurons and restore the CNS homeostasis, activated microglia secreted two types of functional cytokines, including pro-inflammatory interleukins (IL) of IL-1 beta and IL-6 and anti-inflammatory factors of IL-4 and IL-10 in the lesioned larvae. Cytidine 5 '-Diphosphocholine (CDP-choline), as an effective and clinical neuroprotective drug, could further activate microglia, expressing high levels of il-1 beta, il-6, il-4, and il-10 in the TBI model. Moreover, CDP-choline reduced neuronal apoptosis and promoted neuronal proliferation around the lesioned site. Based on these results, the TBI model established in this study represents a suitable model for developing new therapeutic agents for CNS-associated diseases.

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NR 44

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U2 6

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JI Zebrafish

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TI The benefits of voluntary physical exercise after traumatic brain injury

on rat's object recognition memory: A comparison of different temporal

schedules

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Traumatic brain injury; Physical exercise; Object recognition memory;

Neuroprotection; Adult neurogenesis; Microglial reactivity

ID CONTROLLED CORTICAL IMPACT; HIPPOCAMPAL NEUROGENESIS; NEUROINFLAMMATION;

EVOLUTION; PATHOLOGY; RECOVERY; WHITE

AB Physical exercise can reduce the cognitive decline associated with traumatic brain injury, yet little is known about the optimal administration schedules. Here, different protocols of voluntary wheel running were evaluated for their effects on object recognition memory (ORM), neuroprotection (NeuN(+) cells), microglial reactivity (Ibal staining) and neurogenesis (DCX+ cells) after controlled cortical impact injury (CCI). CCI-lesioned rats were divided into a sedentary group and three exercise groups: early discontinued exercise (3 weeks of exercise initiated 4 days post-injury, followed by 4 weeks in a sedentary state); delayed exercise (3 weeks of exercise initiated 4 weeks post-injury), and early continuous exercise (7 weeks of exercise starting 4 days post-injury). The deficits induced by CCI in a 24 h ORM test were reversed in the delayed exercise group and reduced in the early discontinued and early continuous groups. The early discontinued protocol also reduced the loss of NeuN(+) cells in the hilus, while attenuated microglial reactivity was found in the dorsal hippocampus of both the early exercising groups. Running at the end of the experiment increased the number of DCX+ cells in the early continuous and delayed groups, and an inverted U-shaped relationship was found between the mean daily exercise time and the amount of neurogenesis. Thus, exercise had benefits on memory both when it was commenced soon and later after injury, although the neural mechanisms implicated differed. Accordingly, the effects of exercise on memory and neurogenesis appear to not only depend on the specific temporal schedule but also, they may be influenced by the amount of daily exercise.

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TI Inhibiting endogenous tissue plasminogen activator enhanced neuronal

apoptosis and axonal injury after traumatic brain injury

SO NEURAL REGENERATION RESEARCH

LA English

DT Article

DE apoptosis; astrocytes; axonal injury; inflammation; microglia; nerve

regeneration; neural regeneration; neuronal injury; neurons;

neuroserpin; tissue plasminogen activator; traumatic brain injury

ID MICROGLIAL ACTIVATION; PATTERNS; DEFICITS; BARRIER; PROTEIN; STROKE

AB Tissue plasminogen activator is usually used for the treatment of acute ischemic stroke, but the role of endogenous tissue plasminogen activator in traumatic brain injury has been rarely reported. A rat model of traumatic brain injury was established by weight-drop method. The tissue plasminogen activator inhibitor neuroserpin (5 mu L, 0.25 mg/mL) was injected into the lateral ventricle. Neurological function was assessed by neurological severity score. Neuronal and axonal injuries were assessed by hematoxylin-eosin staining and Bielschowsky silver staining. Protein level of endogenous tissue plasminogen activator was analyzed by western blot assay. Apoptotic marker cleaved caspase-3, neuronal marker neurofilament light chain, astrocyte marker glial fibrillary acidic protein and microglial marker Iba-1 were analyzed by immunohistochemical staining. Apoptotic cell types were detected by immunofluorescence double labeling. Apoptotic cells in the damaged cortex were detected by terminal deoxynucleotidyl transferase-mediated digoxigenin-dUTP-biotin nick-end labeling staining. Degenerating neurons in the damaged cortex were detected by Fluoro-Jade B staining. Expression of tissue plasminogen activator was increased at 6 hours, and peaked at 3 days after traumatic brain injury. Neuronal apoptosis and axonal injury were detected after traumatic brain injury. Moreover, neuroserpin enhanced neuronal apoptosis, neuronal injury and axonal injury, and activated microglia and astrocytes. Neuroserpin further deteriorated neurobehavioral function in rats with traumatic brain injury. Our findings confirm that inhibition of endogenous tissue plasminogen activator aggravates neuronal apoptosis and axonal injury after traumatic brain injury, and activates microglia and astrocytes. This study was approved by the Biomedical Ethics Committee of Animal Experiments of Shaanxi Province of China in June 2015.

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TI DPP-4 inhibitor reduces striatal microglial deramification after

sensorimotor cortex injury induced by external force impact

SO FASEB JOURNAL

LA English

DT Article; Early Access

DE controlled cortical impact; dorsolateral striatum; microglial

ramification; sitagliptin; traumatic brain injury; vildagliptin

ID DIPEPTIDYL PEPTIDASE-4 INHIBITOR; BRAIN MITOCHONDRIAL-FUNCTION; TYPE-2

DIABETES-MELLITUS; COGNITIVE DEFICITS; RAT MODEL; VILDAGLIPTIN;

SITAGLIPTIN; NEURODEGENERATION; POLYPEPTIDE; RELEASE

AB Dipeptidyl peptidase-4 inhibitors (or gliptins), a class of antidiabetic drugs, have recently been shown to have protective actions in the central nervous system. Their cellular and molecular mechanisms responsible for these effects are largely unknown. In the present study, two structurally different gliptins, sitagliptin and vildagliptin, were examined for their therapeutic actions in a controlled cortical impact (CCI) model of moderate traumatic brain injury (TBI) in mice. Early post-CCI treatment with sitagliptin, but not vildagliptin, significantly reduced body asymmetry, locomotor hyperactivity, and brain lesion volume. Sitagliptin attenuated post-CCI microglial deramification in the ipsilateral dorsolateral (DL) striatum, while vildagliptin had no effect. Sitagliptin also reduced striatal expression of galectin-3 and monocyte chemoattractant protein 1(MCP-1), and increased the cortical and striatal levels of the anti-inflammatory cytokine IL-10 on the ipsilateral side. These data support a differential protective effect of sitagliptin against TBI, possibly mediated by an anti-inflammatory effect in striatum to preserve connective network. Both sitagliptin and vildagliptin produced similar increases of active glucagon-like peptide-1 (GLP-1) in blood and brain. Increasing active GLP-1 may not be the sole molecular mechanisms for the neurotherapeutic effect of sitagliptin in TBI.

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TI The NLRP3 inflammasome in traumatic brain injury: potential as a

biomarker and therapeutic target

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Review

DE Neuroinflammation; TBI; Concussion; Mild traumatic brain injury; Chronic

traumatic encephalopathy; Microglia; IL-1 beta; Cytokine; Caspase-1;

IL-18

ID INNATE IMMUNE-RESPONSE; PROTEIN-3 INFLAMMASOME; EXTRACELLULAR VESICLES;

AMYLOID-BETA; RISK-FACTOR; CELL-DEATH; K+ EFFLUX; ACTIVATION;

NEUROINFLAMMATION; INHIBITOR

AB There is a great clinical need to identify the underlying mechanisms, as well as related biomarkers, and treatment targets, for traumatic brain injury (TBI). Neuroinflammation is a central pathophysiological feature of TBI. NLRP3 inflammasome activity is a necessary component of the innate immune response to tissue damage, and dysregulated inflammasome activity has been implicated in a number of neurological conditions. This paper introduces the NLRP3 inflammasome and its implication in the pathogenesis of neuroinflammatory-related conditions, with a particular focus on TBI. Although its role in TBI has only recently been identified, findings suggest that priming and activation of the NLRP3 inflammasome are upregulated following TBI. Moreover, recent studies utilizing specific NLRP3 inhibitors have provided further evidence that this inflammasome is a major driver of neuroinflammation and neurobehavioral disturbances following TBI. In addition, there is emerging evidence that circulating inflammasome-associated proteins may have utility as diagnostic biomarkers of neuroinflammatory conditions, including TBI. Finally, novel and promising areas of research will be highlighted, including the potential involvement of the NLRP3 inflammasome in mild TBI, how factors such as biological sex may affect NLRP3 activity in TBI, and the use of emerging biomarker platforms. Taken together, this review highlights the exciting potential of the NLRP3 inflammasome as a target for treatments and biomarkers that may ultimately be used to improve TBI management.

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TI Pediatric Traumatic Brain Injury Causes Long-Term Deficits in Adult

Hippocampal Neurogenesis and Cognition

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE adult neurogenesis; cognition; inflammation; microglia; pediatric TBI

ID NEURAL STEM-CELLS; ENVIRONMENTAL ENRICHMENT; MICROGLIAL ACTIVATION;

VENTRAL HIPPOCAMPUS; CRITICAL PERIOD; DENTATE GYRUS; INFLAMMATION;

PROLIFERATION; RECOVERY; OUTCOMES

AB Young children who have sustained severe traumatic brain injury (TBI) can suffer from debilitating neurocognitive deficits. Impairment of adult hippocampal neurogenesis is associated with cognitive deficits and depression. Very few studies have investigated the adult hippocampal neurogenesis after pediatric TBI. Here, we evaluated long-term cognition, adult hippocampal neurogenesis, and microglial activation in a rabbit pediatric TBI model. On Post-natal Day 5-7 (P5-7), New Zealand white rabbits from the same litter were randomized into naive, sham (craniotomy alone), and TBI (controlled cortical impact). Bromodeoxyuridine (BrdU, 50 mg/kg, intraperitoneally) was administered at 1-month post-injury, once/daily for 5 consecutive days. Novel object recognition and spontaneous alternation in T-maze tests were performed at 2 months post-injury to measure the cognitive functions. The animals were euthanized after behavioral tests at 3 months of age to evaluate adult hippocampal neurogenesis and microglial activation. We found that: 1) pediatric TBI caused significant deficits in hippocampal dependent cognitive functions; 2) the survival rates of adult-born neurons at both ipsilateral and contralateral hippocampus significantly decreased in the TBI group; 3) TBI induced ectopic migration of adult-born neurons at the dorsal dentate gyrus in both ipsilateral and contralateral hippocampus; 4) TBI increased astrogenesis in the hilus of the dentate gyrus; and 5) TBI results in abnormal microglial activation. In conclusion, pediatric TBI causes prolonged neuroinflammation and dysregulation of the adult hippocampal neurogenesis through young adulthood, which might be responsible for the cognitive deficits. Protection of adult hippocampal neurogenesis may potentially improve outcomes.

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TI The Delayed Neuroprotective Effect of Methylene Blue in Experimental Rat

Brain Trauma

SO ANTIOXIDANTS

LA English

DT Article

DE traumatic brain injury; neuroprotection; methylene blue; microglial

cells

ID INDUCED NEUROLOGICAL DEFICIT; BARRIER DISRUPTION; INJURY;

NEURODEGENERATION; DAMAGE; MODEL

AB After traumatic brain injury (TBI), an increase in dysfunction of the limbs contralateral to injury focus was observed. Using different behavioral tests, we found that a single intravenous injection of methylene blue (MB, 1 mg/kg) 30 min after the injury reduced the impairment of the motor functions of the limbs from 7 to 120 days after TBI. Administration of methylene blue 30 min after the injury and then monthly (six injections in total) was the most effective both in terms of preservation of limb function and duration of therapeutic action. This therapeutic effect was clearly manifested from the seventh day and continued until the end of the experiment-by the 180th day after TBI. MB is known to possess antioxidant properties; it has a protective effect against TBI by promoting autophagy and minimizing lesion volume in the first two weeks after TBI. Studies of the brains on the 180th day after TBI demonstrated that the monthly treatment of animals with MB statistically significantly prevented an increase in the density of microglial cells in the ipsilateral hemisphere and a decrease in the thickness of the corpus callosum in the contralateral hemisphere in comparison with untreated animals. However, on the 180th day after TBI, the magnetic resonance imaging scan of the animal brains did not show a significant reduction in the volume of the lesion in MB-treated animals. These findings are important for understanding the development of the long-term effects of TBI and expand the required therapeutic window for targeted neuroprotective interventions.

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TI Neurons in Subcortical Oculomotor Regions are Vulnerable to Plasma

Membrane Damage after Repetitive Diffuse Traumatic Brain Injury in Swine

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE microglia; NeuN; oculomotor; permeability; TBI

ID SPORT-RELATED CONCUSSION; CLOSED-HEAD INJURY; AXONAL INJURY; BASAL

GANGLIA; ANIMAL-MODELS; EYE FIELD; PERMEABILITY; CALCIUM; DEGENERATION;

CONNECTIONS

AB Oculomotor deficits, such as insufficiencies in accommodation, convergence, and saccades, are common following traumatic brain injury (TBI). Previous studies in patients with mild TBI attributed these deficits to insufficient activation of subcortical oculomotor nuclei, although the exact mechanism is unknown. A possible cause for neuronal dysfunction in these regions is biomechanically induced plasma membrane permeability. We used our established porcine model of head rotational TBI to investigate whether cell permeability changes occurred in subcortical oculomotor areas following single or repetitive TBI, with repetitive injuries separated by 15 min, 3 days, or 7 days. Swine were subjected to sham conditions or head rotational acceleration in the sagittal plane using a HYGE pneumatic actuator. Two hours prior to the final injury, the cell-impermeant dye Lucifer Yellow was injected into the ventricles to diffuse throughout the interstitial space to assess plasmalemmal permeability. Animals were sacrificed 15 min after the final injury for immunohistological analysis. Brain regions examined for cell membrane permeability included caudate, substantia nigra pars reticulata, superior colliculus, and cranial nerve oculomotor nuclei. We found that the distribution of permeabilized neurons varied depending on the number and spacing of injuries. Repetitive injuries separated by 15 min or 3 days resulted in the most permeability. Many permeabilized cells lost neuron-specific nuclear protein reactivity, although no neuronal loss occurred acutely after injury. Microglia contacted and appeared to begin phagocytosing permeabilized neurons in repetitively injured animals. These pathologies within oculomotor areas may mediate transient dysfunction and/or degeneration that may contribute to oculomotor deficits following diffuse TBI.

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J9 J NEUROTRAUM

JI J. Neurotrauma

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DI 10.1089/neu.2019.6738

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WC Critical Care Medicine; Clinical Neurology; Neurosciences

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SC General & Internal Medicine; Neurosciences & Neurology

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ER

PT J

AU Lier, J

Ondruschka, B

Bechmann, I

Dressler, J

AF Lier, Julia

Ondruschka, Benjamin

Bechmann, Ingo

Dressler, Jan

TI Fast microglial activation after severe traumatic brain injuries

SO INTERNATIONAL JOURNAL OF LEGAL MEDICINE

LA English

DT Article; Early Access

DE Chronic traumatic encephalopathy; Immunohistochemistry; Inflammation;

Microglia; Traumatic brain injury

ID CONSEQUENCES; TIME; NSE

AB Traumatic brain injury is among the leading causes of death in individuals under 45 years of age. However, since trauma mechanisms and survival times differ enormously, the exact mechanisms leading to the primary and secondary injury and eventually to death after traumatic brain injury (TBI) remain unclear. Several studies showed the versatile functions of microglia, the innate macrophages of the brain, following a TBI. Earlier being characterized as rather neurotoxic, neuroprotective capacities were recently demonstrated, therefore, making microglia one of the key players following TBI. Especially in cases with only short survival times, immediate microglial reactions are of great forensic interest in questions of wound age estimation. Using standardized immunohistochemical methods, we examined 8 cases which died causatively of TBI with survival times between minutes and 7 days and 5 control cases with cardiovascular failure as the cause of death to determine acute changes in microglial morphology and antigen expression after TBI. In this pilot study, we detected highly localized changes in microglial morphology already early after traumatic damage, e.g., activated microglia and phagocyted erythrocytes in the contusion areas in cases with minute survival. Furthermore, an altered antigen expression was observed with increasing trauma wound age, showing similar effects like earlier transcriptomic studies. There is minute data on the direct impact of shear forces on microglial morphology. We were able to show localization-depending effects on microglial morphology causing localized dystrophy and adjacent activation. While rodent studies are widespread, they fail to mimic the exact mechanisms in human TBI response. Therefore, more studies focusing on cadaveric samples need to follow to thoroughly define the mechanisms leading to cell destruction and eventually evaluate their forensic value.

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Wang CF, 2019, J NEUROTRAUM, DOI 10.1089/neu.2019.6460

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TC 19

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U2 6

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Tian, N

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Wang, WJ

Liao, WX

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Liao, Rujia

TI Withaferin A alleviates traumatic brain injury induced secondary brain

injury via suppressing apoptosis in endothelia cells and modulating

activation in the microglia

SO EUROPEAN JOURNAL OF PHARMACOLOGY

LA English

DT Article

DE Withaferin A; Traumatic brain injury; Endothelia cell; Microglia;

Activation; Secondary brain injury

ID WITHANIA-SOMNIFERA; HEAD-INJURY; NEUROPROTECTION; INFLAMMATION;

INHIBITION; EXPRESSION; PROTECTS; MODEL; CARE

AB Traumatic brain injury (TBI) is a major public health concern with high rates of morbidity and mortality worldwide. Currently used medications, though effective, are also associated with several adverse effects. Development of effective neuroprotective agents with fewer side-effects would be of clinical value. Previous studies have shown that withaferin compounds have a potential neuroprotective effect in nervous system disorders. However, the effect of withaferin compounds, especially withaferin A (WFA), on traumatic brain injury is unclear. In the present study, both in vivo and in vitro models were used to assess whether WFA could exert a neuroprotective effect after TBI and were used to explore the associated mechanisms. The results showed that WFA significantly improved neurobehavioral function in a dose-dependent fashion and alleviated histological alteration of injury to tissues in TBI mice. In vitro models of TBI revealed that dose-dependent WFA treatment increased the viability of SH-SY5Y cells. In addition, WFA treatment could attenuate blood-brain barrier disruption and brain edema via suppressing apoptosis in endothelial cells. Furthermore, both our in vivo and in vitro results reveal that WFA treatment could significantly reduce levels of several neuroinflammation cytokines (IL-1 beta, IL-6, and TNF-alpha), which correlate with an overall reduction in microglial activation. These data suggest that the neuroprotection by WFA is, at least in part, related to regulation of microglial activation and inhibition of vascular endothelial cell apoptosis. Taken together, these findings support further investigation of WFA as a promising therapeutic agent for promoting functional recovery after traumatic brain injury.

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NR 50

TC 13

Z9 14

U1 2

U2 26

PU ELSEVIER

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PT J

AU Caplan, HW

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TI Human cord blood-derived regulatory T-cell therapy modulates the central

and peripheral immune response after traumatic brain injury

SO STEM CELLS TRANSLATIONAL MEDICINE

LA English

DT Article; Early Access

DE cell therapy; CNS trauma; cord blood; microglia; neuroinflammation;

regenerative medicine; TBI; Treg; t-SNE

ID MICROGLIA; NEUROINFLAMMATION; INFLAMMATION; EXPRESSION; DISEASE

AB Traumatic brain injury (TBI) causes a profound inflammatory response within the central nervous system and peripheral immune system, which contributes to secondary brain injury and further morbidity and mortality. Preclinical investigations have demonstrated that treatments that downregulate microglia activation and polarize them toward a reparative/anti-inflammatory phenotype have improved outcomes in preclinical models. However, no therapy to date has translated into proven benefits in human patients. Regulatory T cells (Treg) have been shown to downregulate pathologic immune responses of the innate and adaptive immune system across a variety of pathologies. Furthermore, cellular therapy has been shown to augment host Treg responses in preclinical models; yet, studies investigating the use of Treg as a therapeutic for TBI are lacking. In a rodent TBI model, we demonstrate that human umbilical cord blood Treg modulate the central and peripheral immune response after injury in vitro and in vivo.

C1 [Caplan, Henry W.; Prabhakara, Karthik S.; Kumar, Akshita; Toledano-Furman, Naama E.; Martin, Cecilia; Carrillo, Louis; Moreno, Nicolas F.; Bordt, Andrea S.; Olson, Scott D.; Cox, Charles S., Jr.] Univ Texas Hlth Sci Ctr Houston, Dept Pediat Surg, McGovern Med Sch, 6431 Fannin St,MSE R162, Houston, TX 77030 USA.

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TC 22

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JI Stem Cells Transl. Med.

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WC Cell & Tissue Engineering

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Arner, Elias S. J.

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TI Characterization of More Selective Central Nervous System

Nrf2-Activating Novel Vinyl Sulfoximine Compounds Compared to Dimethyl

Fumarate

SO NEUROTHERAPEUTICS

LA English

DT Article; Early Access

DE Nrf2; NF kappa B; HIF; dimethyl fumarate; multiple sclerosis; redox

regulation; pTRAF; sulfoximine; traumatic brain injury; microglia

ID PLACEBO-CONTROLLED PHASE-3; TRAUMATIC BRAIN-INJURY; OXIDATIVE STRESS;

ORAL BG-12; CELLS; PATHWAY; ACID; NRF2; DEFENSE; ROS

AB The Nrf2 transcription factor is a key regulator of redox reactions and considered the main target for the multiple sclerosis (MS) drug dimethyl fumarate (DMF). However, exploration of additional Nrf2-activating compounds is motivated, since DMF displays significant off-target effects and has a relatively poor penetrance to the central nervous system (CNS). We de novo synthesized eight vinyl sulfone and sulfoximine compounds (CH-1-CH-8) and evaluated their capacity to activate the transcription factors Nrf2, NF kappa B, and HIF1 in comparison with DMF using the pTRAF platform. The novel sulfoximine CH-3 was the most promising candidate and selected for further comparison in vivo and later an experimental model for traumatic brain injury (TBI). CH-3 and DMF displayed comparable capacity to activate Nrf2 and downstream transcripts in vitro, but with less off-target effects on HIF1 from CH-3. This was verified in cultured microglia and oligodendrocytes (OLs) and subsequently in vivo in rats. Following TBI, DMF lowered the number of leukocytes in blood and also decreased axonal degeneration. CH-3 preserved or increased the number of pre-myelinating OL. While both CH-3 and DMF activated Nrf2, CH-3 showed less off-target effects and displayed more selective OL associated effects. Further studies with Nrf2-acting compounds are promising candidates to explore potential myelin protective or regenerative effects in demyelinating disorders.

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AF Glotfelty, Elliot J.

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TI Glucagon-like peptide-1 (GLP-1)-based receptor agonists as a treatment

for Parkinson's disease

SO EXPERT OPINION ON INVESTIGATIONAL DRUGS

LA English

DT Review; Early Access

DE Glucagon-like peptide-1 (GLP-1); glucose-dependent insulinotropic

peptide (GIP); glucagon (Gcg); Parkinson's disease; incretin mimetics;

neurodegeneration; microglia; brain trauma

ID MPTP MOUSE MODEL; INSULIN-RESISTANCE; GLP-1 RECEPTOR; BODY-WEIGHT; RAT

MODEL; OXYNTOMODULIN; MECHANISMS; ALZHEIMERS; PATHOLOGY; TARGET

AB Introduction: Accumulating evidence supports the evaluation of glucagon-like peptide-1 (GLP-1) receptor (R) agonists for the treatment of the underlying pathology causing Parkinson's Disease (PD). Not only are these effects evident in models of PD and other neurodegenerative disorders but recently in a randomized, double-blind, placebo-controlled clinical trial, a GLP-1R agonist has provided improved cognition motor functions in humans with moderate PD. Areas covered: In this mini-review, we describe the development of GLP-1R agonists and their potential therapeutic value in treating PD. Many GLP-1R agonists are FDA approved for the treatment of metabolic disorders, and hence can be rapidly repositioned for PD. Furthermore, we present preclinical data offering insights into the use of monomeric dual- and tri-agonist incretin-based mimetics for neurodegenerative disorders. These drugs combine active regions of GLP-1 with those of glucose-dependent insulinotropic peptide (GIP) and/or glucagon (Gcg). Expert opinion: GLP-1Ragonists offer a complementary and enhanced therapeutic value to other drugs used to treat PD. Moreover, the use of the dual- or tri-agonist GLP-1-based mimetics may provide combinatory effects that are even more powerful than GLP-1R agonism alone. We advocate for further investigations into the repurposing of GLP-1R agonists and the development of classes of multi-agonists for PD treatment.

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TI Extracellular Vesicles miRNA Cargo for Microglia Polarization in

Traumatic Brain Injury

SO BIOMOLECULES

LA English

DT Review

DE TBI; neuroinflammation; microglia; Extracellular Vesicles; Exosomes;

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ID CIRCULATING MICROPARTICLES; MICRORNA BIOGENESIS; CONTAINING EXOSOMES;

SIGNALING PATHWAY; CEREBRAL-CORTEX; STROMAL CELLS; MOUSE-BRAIN;

IN-VITRO; INFLAMMATION; MICROVESICLES

AB Traumatic brain injury (TBI) is one of the major causes of death and disability worldwide, and despite its high dissemination, effective pharmacotherapies are lacking. TBI can be divided into two phases: the instantaneous primary mechanical injury, which occurs at the moment of insult, and the delayed secondary injury, which involves a cascade of biological processes that lead to neuroinflammation. Neuroinflammation is a hallmark of both acute and chronic TBI, and it is considered to be one of the major determinants of the outcome and progression of disease. In TBI one of the emerging mechanisms for cell-cell communication involved in the immune response regulation is represented by Extracellular Vesicles (EVs). These latter are produced by all cell types and are considered a fingerprint of their generating cells. Exosomes are the most studied nanosized vesicles and can carry a variety of molecular constituents of their cell of origin, including microRNAs (miRNAs). Several miRNAs have been shown to target key neuropathophysiological pathways involved in TBI. The focus of this review is to analyze exosomes and their miRNA cargo to modulate TBI neuroinflammation providing new strategies for prevent long-term progression of disease.

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TI Tizoxanide mitigates inflammatory response in LPS-induced

neuroinflammation in microglia <i>via</i> restraining p38/MAPK pathway

SO EUROPEAN REVIEW FOR MEDICAL AND PHARMACOLOGICAL SCIENCES

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DT Article

DE Tizoxanide; Neuroinflammation; Microglia; p38/MAPK pathway; Traumatic

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ID NITAZOXANIDE; PHARMACOKINETICS; VIRUS

AB OBJECTIVE: Traumatic brain injury (TBI) induced neuroinflammation is featured as excessive glial inflammatory activation and violent neurologic destruction and dysfunction. Massive microglia activation in situ and disrupt of blood-brain barrier contribute to severely collapsed nervous system. Tizoxanide (TIZ), a synthetic thiazolide derivative agent possessing a broad-spectrum anti-infective effect, currently shows a potential resistance against pathogens like bacteria, virus and parasites, while its underlying role in neuroinflammation is elusive. The study aimed to explore the effect of TIZ on neuroinflammation In vitro microglia.

MATERIALS AND METHODS: Primary microglia were accepted to neuroinflammatory activation via lipopolysaccharide (LPS) administration. TIZ was conducted to pretreatment of microglia. Cell viability, inflammatory cytokines, chemotaxis, nitric oxide release, inflammation-related enzymes, and mitogen-activated protein kinase (MAPK) pathway activation in microglia were investigated respectively.

RESULTS: We demonstrated that TIZ administration attenuates inflammatory cytokines and chemokines through quantitative real-time polymerase chain reaction (qRT-PCR) and enzyme-linked immunosorbent assay (ELISA) of medium supernatant. In addition, TIZ reduces pro-inflammatory mediators and nitric oxide release in microglia. Furtherly, TIZ inhibits the level of p38/MAPK pathway in LPS stimuli, indicating that TIZ negatively regulates neuroinflammation via inhibiting p38/MAPK pathway.

CONCLUSIONS: TIZ is verified to be an anti-inflammation effect on neuroinflammation in microglia via downregulation of p38/MAPK pathway, which restrains inflammation by reduced inflammatory cytokines, chemokines and mediators and decreased nitric oxide release. To summarize, TIZ is considered to be a promising reagent to alleviate neuroinflammation targeting microglia in nervous system injury.

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TI Neuroinflammatory mechanisms of post-traumatic epilepsy

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DE Traumatic brain injury; TBI; Astrocytes; Microglia; Cytokines;

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ID TRAUMATIC BRAIN-INJURY; DENTATE GRANULE CELLS; HILAR BASAL DENDRITES;

ACUTE-PHASE RESPONSE; MICROGLIAL ACTIVATION; INDUCED SEIZURES; KINDLING

EPILEPTOGENESIS; INTERLEUKIN-1 RECEPTOR; GENE POLYMORPHISMS; GLUTAMATE

UPTAKE

AB Background Traumatic brain injury (TBI) occurs in as many as 64-74 million people worldwide each year and often results in one or more post-traumatic syndromes, including depression, cognitive, emotional, and behavioral deficits. TBI can also increase seizure susceptibility, as well as increase the incidence of epilepsy, a phenomenon known as post-traumatic epilepsy (PTE). Injury type and severity appear to partially predict PTE susceptibility. However, a complete mechanistic understanding of risk factors for PTE is incomplete. Main body From the earliest days of modern neuroscience, to the present day, accumulating evidence supports a significant role for neuroinflammation in the post-traumatic epileptogenic progression. Notably, substantial evidence indicates a role for astrocytes, microglia, chemokines, and cytokines in PTE progression. Although each of these mechanistic components is discussed in separate sections, it is highly likely that it is the totality of cellular and neuroinflammatory interactions that ultimately contribute to the epileptogenic progression following TBI. Conclusion This comprehensive review focuses on the neuroinflammatory milieu and explores putative mechanisms involved in the epileptogenic progression from TBI to increased seizure-susceptibility and the development of PTE.

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TI Up-regulation of CHMP4B alleviates microglial necroptosis induced by

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AB Microglial cells are key component of central nervous system (CNS) and mediate the immune response of the brain under physiological or pathological conditions. It tends to activate into a pro-inflammatory M1 phenotype after traumatic brain injury (TBI) and promote secondary brain damage. Recently, necroptosis was found to promote microglial activation and neuroinflammation after TBI. However, the mechanism and specific interventions of microglial necroptosis after TBI remain poorly investigated. Here, we reported that overexpress the charged multivesicular body protein 4b (CHMP4B) which is a core member of the endosomal sorting required for transport complex III (ESCRT-III) significantly decreased the level of necroptosis in microglia, improved neurological function recovery and protected against cell death after TBI. Further investigation showed that forkhead transcription factor O1 (FOXO1) was a crucial transcription factor that increased CHMP4B transcription by binding to the promoter region, thereby inhibiting necroptosis in microglia. Collectively, our findings demonstrated that CHMP4B relieved microglial necroptosis and neuroinflammation after TBI, and promote the recovery of nerve function. FOXO1 is an important factor in promoting CHMP4B expression. This study provides the novel viewpoint for TBI prevention and treatment.

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TI Cosyntropin Attenuates Neuroinflammation in a Mouse Model of Traumatic

Brain Injury

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DT Article

DE traumatic brain injury; ACTH; microglia; behavior; neuroinflammation;

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ID MELANOCYTE-STIMULATING HORMONE; ADRENOCORTICOTROPIC HORMONE;

CEREBRAL-ISCHEMIA; MESSENGER-RNA; GLUCOCORTICOIDS MODULATE; MELANOCORTIN

RECEPTORS; SYNAPTIC PLASTICITY; RAT HIPPOCAMPUS; HEAD-INJURY; ACTHAR GEL

AB Aim: Traumatic brain injury (TBI) is a leading cause of mortality/morbidity and is associated with chronic neuroinflammation. Melanocortin receptor agonists including adrenocorticotropic hormone (ACTH) ameliorate inflammation and provide a novel therapeutic approach. We examined the effect of long-acting cosyntropin (CoSyn), a synthetic ACTH analog, on the early inflammatory response and functional outcome following experimental TBI. Methods: The controlled cortical impact model was used to induce TBI in mice. Mice were assigned to injury and treatment protocols resulting in four experimental groups including sham + saline, sham + CoSyn, TBI + saline, and TBI + CoSyn. Treatment was administered subcutaneously 3 h post-injury and daily injections were given for up to 7 days post-injury. The early inflammatory response was evaluated at 3 days post-injury through the evaluation of cytokine expression (IL1 beta and TNF alpha) and immune cell response. Quantification of immune cell response included cell counts of microglia/macrophages (Iba1+ cells) and neutrophils (MPO+ cells) in the cortex and hippocampus. Behavioral testing (n= 10-14 animals/group) included open field (OF) and novel object recognition (NOR) during the first week following injury and Morris water maze (MWM) at 10-15 days post-injury. Results: Immune cell quantification showed decreased accumulation of Iba1+ cells in the perilesional cortex and CA1 region of the hippocampus for CoSyn-treated TBI animals compared to saline-treated. Reduced numbers of MPO+ cells were also found in the perilesional cortex and hippocampus in CoSyn treated TBI mice compared to their saline-treated counterparts. Furthermore, CoSyn treatment reduced IL1 beta expression in the cortex of TBI mice. Behavioral testing showed a treatment effect of CoSyn for NOR with CoSyn increasing the discrimination ratio in both TBI and Sham groups, indicating increased memory performance. CoSyn also decreased latency to find platform during the early training period of the MWM when comparing CoSyn to saline-treated TBI mice suggesting moderate improvements in spatial memory following CoSyn treatment. Conclusion: Reduced microglia/macrophage accumulation and neutrophil infiltration in conjunction with moderate improvements in spatial learning in our CoSyn treated TBI mice suggests a beneficial anti-inflammatory effect of CoSyn following TBI.

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TI LncRNA HOTAIR Participates in Microglia Activation and Inflammatory

Factor Release by Regulating the Ubiquitination of MYD88 in Traumatic

Brain Injury

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AB Traumatic brain injury (TBI) is one of the leading causes of death worldwide. Long non-coding RNAs (LncRNAs) have been reported to be closely associated with various diseases, but their roles in TBI has not been fully elucidated. The purpose of this study was to elucidate the underlying mechanism of LncRNA HOTAIR in TBI-induced microglial activation and inflammatory factor release. In vivo mouse TBI model and in vitro microglia activation model were established by Feeney's free-fall impact method and by LPS stimulation, respectively. The expression of LncRNA HOTAIR in activated microglia was detected by qRT-PCR. After shRNA knocked down, the expressions of LncRNA HOTAIR and microglia activation marker Iba-1 in microglia were detected by qRT-PCR and Western blot and by ELISA that detected the concentration of inflammatory factor in cell culture supernatants. The relationship between LncRNA HOTAIR and MYD88 in mouse microglia BV2 cells was observed by RNA pull-down assay. Furthermore, the effect of LncRNA HOTAIR on MYD88 stability was assessed by cycloheximide (CHX)-chase and by immunoprecipitation and ubiquitination assays that analyzed MYD88 ubiquitination. LncRNA HOTAIR was abnormally highly expressed in activated microglia. By Western blot and ELISA, the knockdown of LncRNA HOTAIR in microglia significantly repressed microglia activation and inflammatory factor release. By RNA pull-down assay, LncRNA HOTAIR could bind to MYD88 protein. Besides, by cycloheximide (CHX)-chase and immunoprecipitation and ubiquitination assays, the overexpression of the LncRNA HOTAIR enhanced the stability of MYD88 protein and inhibited Nrdp1-mediated ubiquitination of MYD88 protein. After the transfection of shRNA-HOTAIR and shRNA-HOTAIR+pcDNA-MYD88 into microglia, shRNA-HOTAIR could significantly inhibit the activation of microglia and the release of inflammatory factors, while these effects were reversed after the transfection of pcDNA-MYD88. Our experimental data indicated that LncRNA HOTAIR was highly expressed in activated microglia, and our further studies had found that the interference with LncRNA HOTAIR could repress microglia activation and inflammatory factor release via promoting Nrdp1-mediated ubiquitination of MYD88 protein.

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TI Glial Activation in the Thalamus Contributes to Vestibulomotor Deficits

Following Blast-Induced Neurotrauma

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LA English

DT Article

DE thalamus; amygdala; blast; vestibulomotor; microglia; astrocytes;

traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; FUNCTIONAL RECOVERY; OXIDATIVE STRESS; EXPOSURE;

MECHANISMS; ANXIETY; MODEL; CONSEQUENCES; MACROPHAGES; IMPAIRMENT

AB Vestibular impairment has become a frequent consequence following blast-related traumatic brain injury (bTBI) in military personnel and Veterans. Behavioral outcomes such as depression, fear and anxiety are also common comorbidities of bTBI. To accelerate pre-clinical research and therapy developments, there is a need to study the link between behavioral patterns and neuropathology. The transmission of neurosensory information often involves a pathway from the cerebral cortex to the thalamus, and the thalamus serves crucial integrative functions within vestibular processing. Pathways from the thalamus also connect with the amygdala, suggesting thalamic and amygdalar contributions to anxiolytic behavior. Here we used behavioral assays and immunohistochemistry to determine the sub-acute and early chronic effects of repeated blast exposure on the thalamic and amygdala nuclei. Behavioral results indicated vestibulomotor deficits at 1 and 3 weeks following repeated blast events. Anxiety-like behavior assessments depicted trending increases in the blast group. Astrogliosis and microglia activation were observed upon post-mortem pathological examination in the thalamic region, along with a limited glia response in the amygdala at 4 weeks. These findings are consistent with a diffuse glia response associated with bTBI and support the premise that dysfunction within the thalamic nuclei following repeated blast exposures contribute to vestibulomotor impairment.

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TI High-resolution and differential analysis of rat microglial markers in

traumatic brain injury: conventional flow cytometric and bioinformatics

analysis

SO SCIENTIFIC REPORTS

LA English

DT Article

ID MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS; ALZHEIMERS-DISEASE;

ACTIVATION; RECEPTOR; CELLS; IDENTIFICATION; MACROPHAGES; EXPRESSION;

MECHANISM

AB Traumatic brain injury (TBI) results in a cascade of cellular responses, which produce neuroinflammation, partly due to microglial activation. Transforming from surveying to primed phenotypes, microglia undergo considerable molecular changes. However, specific microglial profiles in rat remain elusive due to tedious methodology and limited availability of reagents. Here, we present a flow cytometry-based analysis of rat microglia 24 h after TBI using the controlled cortical impact model, validated with a bioinformatics approach. Isolated microglia are analyzed for morphological changes and their expression of activation markers using flow cytometry, traditional gating-based analysis methods and support the data by employing bioinformatics statistical tools. We use CD45, CD11b/c, and p2y12 receptor to identify microglia and evaluate their activation state using CD32, CD86, RT1B, CD200R, and CD163. The results from logic-gated flow cytometry analysis was validated with bioinformatics-based analysis and machine learning algorithms to detect quantitative changes in morphology and marker expression in microglia due to activation following TBI.

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TI Catastrophic consequences: can the feline parasite<i>Toxoplasma

gondii</i>prompt the purrfect neuroinflammatory storm following

traumatic brain injury?

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Review

DE Parasite; Infection; Neuroinflammation; Immune response; Microglia;

Astrocytes

ID NECROSIS-FACTOR-ALPHA; INTERLEUKIN-1 RECEPTOR ANTAGONIST; ANXIETY-LIKE

BEHAVIOR; T-CELL INFILTRATION; TOXOPLASMA-GONDII; INTERFERON-GAMMA;

OXIDATIVE STRESS; INFLAMMATORY RESPONSE; HYPERPHOSPHORYLATED TAU;

LEUKOCYTE INFILTRATION

AB Traumatic brain injury (TBI) is one of the leading causes of morbidity and mortality worldwide; however, treatment development is hindered by the heterogenous nature of TBI presentation and pathophysiology. In particular, the degree of neuroinflammation after TBI varies between individuals and may be modified by other factors such as infection.Toxoplasma gondii, a parasite that infects approximately one-third of the world's population, has a tropism for brain tissue and can persist as a life-long infection. Importantly, there is notable overlap in the pathophysiology between TBI andT.gondiiinfection, including neuroinflammation. This paper will review current understandings of the clinical problems, pathophysiological mechanisms, and functional outcomes of TBI andT.gondii, before considering the potential synergy between the two conditions. In particular, the discussion will focus on neuroinflammatory processes such as microglial activation, inflammatory cytokines, and peripheral immune cell recruitment that occur duringT.gondiiinfection and after TBI. We will present the notion that these overlapping pathologies in TBI individuals with a chronicT.gondiiinfection have the strong potential to exacerbate neuroinflammation and related brain damage, leading to amplified functional deficits. The impact of chronicT.gondiiinfection on TBI should therefore be investigated in both preclinical and clinical studies as the possible interplay could influence treatment strategies.

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TI TMEM119 as a specific marker of microglia reaction in traumatic brain

injury in postmortem examination

SO INTERNATIONAL JOURNAL OF LEGAL MEDICINE

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DT Article; Early Access

DE Cerebrospinal fluid; Forensic neuropathology; Forensic

neurotraumatology; Immunohistochemistry; Immunocytochemistry; Biomarker

ID PERIVASCULAR MACROPHAGES; SPINAL-CORD; EXPRESSION; BIOMARKERS;

ACTIVATION; REVEALS; CELLS

AB The aim of the present study was a refined analysis of neuroinflammation including TMEM119 as a useful microglia-specific marker in forensic assessments of traumatic causes of death, e.g., traumatic brain injury (TBI). Human brain tissue samples were obtained from autopsies and divided into cases with lethal TBI (n = 25) and subdivided into three groups according to their trauma survival time and compared with an age-, gender-, and postmortem interval-matched cohort of sudden cardiovascular fatalities as controls (n = 23). Brain tissue samples next to cortex contusions and surrounding white matter as well as samples of the ipsilateral uninjured brain stem and cerebellum were collected and stained immunohistochemically with antibodies against TMEM119, CD206, and CCR2. We could document the highest number of TMEM119-positive cells in acute TBI death with highly significant differences to the control numbers. CCR2-positive monocytes showed a significantly higher cell count in the cortex samples of TBI cases than in the controls with an increasing number of immunopositive cells over time. The number of CD206-positive M2 microglial cells increased survival time-dependent. After 3 days of survival, the cell number increased significantly in all four regions investigated compared with controls. In sum, we validate a specific and robustly expressed as well as fast reacting microglia marker, TMEM119, which distinguishes microglia from resident and infiltrating macrophages and thus offers a great potential for the estimation of the minimum survival time after TBI.

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TI Polysialic acid and Siglec-E orchestrate negative feedback regulation of

microglia activation

SO CELLULAR AND MOLECULAR LIFE SCIENCES

LA English

DT Article; Early Access

DE Immune balance; Inflammatory activation; Innate immune response;

Traumatic brain injury; Sialic acid-binding immunoglobulin-like lectins

ID TRAUMATIC BRAIN-INJURY; SYNCAM 1; CELLS; POLYSIALYLATION; NEUROPILIN-2;

MACROPHAGES; SUPPRESSION; MODULATION; GENERATION; RECEPTORS

AB Polysialic acid (polySia) emerges as a novel regulator of microglia activity. We recently identified polysialylated proteins in the Golgi compartment of murine microglia that are released in response to inflammatory stimulation. Since exogenously added polySia is able to attenuate the inflammatory response, we proposed that the release of polysialylated proteins constitutes a mechanism for negative feedback regulation of microglia activation. Here, we demonstrate that translocation of polySia from the Golgi to the cell surface can be induced by calcium depletion of the Golgi compartment and that polysialylated proteins are continuously released for at least 24 h after the onset of inflammatory stimulation. The latter was unexpected, because polySia signals detected by immunocytochemistry are rapidly depleted. However, it indicates that the amount of released polySia is much higher than anticipated based on immunostaining. This may be crucial for microglial responses during traumatic brain injury (TBI), as we detected polySia signals in activated microglia around a stab wound in the adult mouse brain. In BV2 microglia, the putative polySia receptor Siglec-E is internalized during lipopolysaccharide (LPS)-induced activation and in response to polySia exposure, indicating interaction. Correspondingly, CRISPR/Cas9-mediated Siglec-E knockout prevents inhibition of pro inflammatory activation by exogenously added polySia and leads to a strong increase of the LPS response. A comparable increase of LPS-induced activation has been observed in microglia with abolished polySia synthesis. Together, these results indicate that the release of the microglia-intrinsic polySia pool, as implicated in TBI, inhibits the inflammatory response by acting as atrans-activating ligand of Siglec-E.

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TI Glia Maturation Factor (GMF) Regulates Microglial Expression Phenotypes

and the Associated Neurological Deficits in a Mouse Model of Traumatic

Brain Injury

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article; Early Access

DE Glia maturation factor; Neuroinflammation; M1-like; M2-like; Microglia;

macrophage; Polarization; Traumatic brain injury

ID CENTRAL-NERVOUS-SYSTEM; OXIDATIVE STRESS; FACTOR-BETA; APOPTOSIS; CELLS;

DAMAGE; NEUROTOXICITY; RECOGNITION; ACTIVATION; INHIBITOR

AB Traumatic brain injury (TBI) induces inflammatory responses through microglial activation and polarization towards a more inflammatory state that contributes to the deleterious secondary brain injury. Glia maturation factor (GMF) is a pro-inflammatory protein that is responsible for neuroinflammation following insult to the brain, such as in TBI. We hypothesized that the absence of GMF in GMF-knockout (GMF-KO) mice would regulate microglial activation state and the M1/M2 phenotypes following TBI. We used the weight drop model of TBI in C57BL/6 mice wild-type (WT) and GMF-KO mice. Immunofluorescence staining, Western blot, and ELISA assays were performed to confirm TBI-induced histopathological and neuroinflammatory changes. Behavioral analysis was done to check motor coordination ability and cognitive function. We demonstrated that the deletion of GMF in GMF-KO mice significantly limited lesion volume, attenuated neuronal loss, inhibited gliosis, and activated microglia adopted predominantly anti-inflammatory (M2) phenotypes. Using an ELISA method, we found a gradual decrease in pro-inflammatory cytokines (TNF-alpha and IL-6) and upregulation of anti-inflammatory cytokines (IL-4 and IL-10) in GMF-KO mice compared with WT mice, thus, promoting the transition of microglia towards a more predominantly anti-inflammatory (M2) phenotype. GMF-KO mice showed significant improvement in motor ability, memory, and cognition. Overall, our results demonstrate that GMF deficiency regulates microglial polarization, which ameliorates neuronal injury and behavioral impairments following TBI in mice and concludes that GMF is a regulator of neuroinflammation and an ideal therapeutic target for the treatment of TBI.

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TI Sex-dependent effects of GPER activation on neuroinflammation in a rat

model of traumatic brain injury

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article

DE GPER; Microglia; Neuroinflammation; Estrogen; Traumatic brain injury

ID PROTEIN-COUPLED RECEPTOR; INFLAMMATORY RESPONSE; ESTROGEN; GPR30;

17-BETA-ESTRADIOL; NEUROPROTECTION; ALPHA; EXPRESSION; MICROGLIA;

ISCHEMIA

AB The G protein-coupled estrogen receptor (GPER) plays a role in estrogen-mediated neuroprotection and has been considered a potential therapeutic target for treating various neurological diseases. It is increasingly recognized that sex is a biological variable affecting treatment outcomes and efficacy, and that neuroinflammation is a key secondary injury mechanism following brain injury, though it is unknown whether the neuroprotective effects exerted by GPER involve modulation of inflammatory processes. The aim of this study was to investigate whether activation of GPER has a sex-dependent effect on neuroinflammation following traumatic brain injury (TBI), a sexually dimorphic disease. In male and ovariectomized (OVX) female rats, the GPER agonist, G1, inhibited the upregulated expression of pro-inflammatory cytokines (IL-1 beta, IL-6, and TNF-alpha), increased the expression of the anti-inflammatory cytokine IL-4, and shifted microglia/macrophage polarization toward the M2 phenotype. In gonadally-intact females, G1 caused more pro-inflammatory (IL-6 and TNF-alpha) and less anti-inflammatory cytokine (IL-4) production, without altering microglia/macrophage polarization. Estradiol supplementation blocked the effects of G1 in OVX females. We also found that post-injury GPER expression was increased in males and OVX females but not in intact females. G1 administration increased Akt phosphorylation in males and OVX females, but had no significant effect in intact females, while Akt inhibition blocked the effects of G1 in males and OVX females. These results indicate that G1 exerts anti-inflammatory effects in males and OVX females but not in intact females; these sex-specific effects are dependent on circulating estrogen levels and are partially mediated through Akt signaling. Future studies are needed to elucidate the relevant molecular mechanisms, especially in females. A better understanding of the sex differences in treatment efficacy with GPER agonists may help improve personalized therapeutic strategies for males and pre- and postmenopausal females with TBI.

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TI TGFβ1 alleviates axonal injury by regulating microglia/macrophages

alternative activation in traumatic brain injury

SO BRAIN RESEARCH BULLETIN

LA English

DT Article

DE TGF beta 1; Traumatic brain injury; Axonal injury;

Microglia/macrophages; Classical activation; Alternative activation

ID BETA-R-I; NEUROINFLAMMATORY RESPONSES; WHITE-MATTER; MICROGLIA;

POLARIZATION; INFLAMMATION; MACROPHAGES

AB Traumatic brain injury (TBI) causes substantial mortality and long-term disability worldwide. TGF beta 1 is a unique molecular and functional signature in microglia, but the role of TGF beta 1 in TBI is not clear. The purpose of this study was to investigate the role of TGF beta 1 in TBI. The weight dropping device was used to establish TBI model of rats. Hematoxylin eosin staining and Bielschowsky silver staining were used to assess tissue loss. Beam walking and muscle strength tests were used to assess neurological deficits. Immunohistochemical staining was used to assess axonal injures. Western blotting was used to detect expression of related proteins. RT-PCR was used to detect expression of cytokines. Immunofluorescence staining was used to assess the microglia/macrophages activation. We observed obvious axonal injury and microglia/macrophages activation in the peri-lesion cortex. The expression of inflammatory cytokines was markedly high after TBI. The expression of TGF beta 1 and TGF beta RI were significantly reduced after TBI. TGF beta 1 promoted the functional recovery and alleviated axonal injury 1 day after TBI. TGF beta 1 promoted microglia/macrophages polarizing to alternative activation and alleviated neuroinflammation. These effects of TGF beta 1 could be inhibited by LY2109761, the inhibitor of TGFRI/II. These results suggested that TGF beta 1 played a protective role in axonal injury and could be a potential therapeutic target in early stages following TBI.

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TI The Bidirectional Relationship Between Sleep and Inflammation Links

Traumatic Brain Injury and Alzheimer's Disease

SO FRONTIERS IN NEUROSCIENCE

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DT Review

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EXTRACELLULAR TAU

AB Traumatic brain injury (TBI) and Alzheimer's disease (AD) are diseases during which the fine-tuned autoregulation of the brain is lost. Despite the stark contrast in their causal mechanisms, both TBI and AD are conditions which elicit a neuroinflammatory response that is coupled with physical, cognitive, and affective symptoms. One commonly reported symptom in both TBI and AD patients is disturbed sleep. Sleep is regulated by circadian and homeostatic processes such that pathological inflammation may disrupt the chemical signaling required to maintain a healthy sleep profile. In this way, immune system activation can influence sleep physiology. Conversely, sleep disturbances can exacerbate symptoms or increase the risk of inflammatory/neurodegenerative diseases. Both TBI and AD are worsened by a chronic pro-inflammatory microenvironment which exacerbates symptoms and worsens clinical outcome. Herein, a positive feedback loop of chronic inflammation and sleep disturbances is initiated. In this review, the bidirectional relationship between sleep disturbances and inflammation is discussed, where chronic inflammation associated with TBI and AD can lead to sleep disturbances and exacerbated neuropathology. The role of microglia and cytokines in sleep disturbances associated with these diseases is highlighted. The proposed sleep and inflammation-mediated link between TBI and AD presents an opportunity for a multifaceted approach to clinical intervention.

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TI Microglia and Macrophages in the Pathological Central and Peripheral

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DT Review

DE traumatic brain injury; brain infarction; carbon monoxide poisoning;

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ID COLONY-STIMULATING FACTOR; TRAUMATIC BRAIN-INJURY; DELAYED NEURONAL

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ANALYSIS REVEALS; VIABLE NEURONS; MOUSE MODEL; GM-CSF

AB Microglia, the immunocompetent cells in the central nervous system (CNS), have long been studied as pathologically deteriorating players in various CNS diseases. However, microglia exert ameliorating neuroprotective effects, which prompted us to reconsider their roles in CNS and peripheral nervous system (PNS) pathophysiology. Moreover, recent findings showed that microglia play critical roles even in the healthy CNS. The microglial functions that normally contribute to the maintenance of homeostasis in the CNS are modified by other cells, such as astrocytes and infiltrated myeloid cells; thus, the microglial actions on neurons are extremely complex. For a deeper understanding of the pathophysiology of various diseases, including those of the PNS, it is important to understand microglial functioning. In this review, we discuss both the favorable and unfavorable roles of microglia in neuronal survival in various CNS and PNS disorders. We also discuss the roles of blood-borne macrophages in the pathogenesis of CNS and PNS injuries because they cooperatively modify the pathological processes of resident microglia. Finally, metabolic changes in glycolysis and oxidative phosphorylation, with special reference to the pro-/anti-inflammatory activation of microglia, are intensively addressed, because they are profoundly correlated with the generation of reactive oxygen species and changes in pro-/anti-inflammatory phenotypes.

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LA English

DT Article

DE traumatic brain injury; post-traumatic epilepsy; epileptiform

discharges; electrocorticograms; local field potentials; neocortex;

hippocampus; microglia; neurodegeneration

ID DELTA BRUSH; EEG; SEIZURE; HIPPOCAMPAL; MECHANISMS; EPILEPSY;

NEUROINFLAMMATION; EPIDEMIOLOGY; FREQUENCY; SOCIETY

AB Background: In humans, early pathological activity on invasive electrocorticograms (ECoGs) and its putative association with pathomorphology in the early period of traumatic brain injury (TBI) remains obscure. Methods: We assessed pathological activity on scalp electroencephalograms (EEGs) and ECoGs in patients with acute TBI, early electrophysiological changes after lateral fluid percussion brain injury (FPI), and electrophysiological correlates of hippocampal damage (microgliosis and neuronal loss), a week after TBI in rats. Results: Epileptiform activity on ECoGs was evident in 86% of patients during the acute period of TBI, ECoGs being more sensitive to epileptiform and periodic discharges. A "brush-like" ECoG pattern superimposed over rhythmic delta activity and periodic discharge was described for the first time in acute TBI. In rats, FPI increased high-amplitude spike incidence in the neocortex and, most expressed, in the ipsilateral hippocampus, induced hippocampal microgliosis and neuronal loss, ipsilateral dentate gyrus being most vulnerable, a week after TBI. Epileptiform spike incidence correlated with microglial cell density and neuronal loss in the ipsilateral hippocampus. Conclusion: Epileptiform activity is frequent in the acute period of TBI period and is associated with distant hippocampal damage on a microscopic level. This damage is probably involved in late consequences of TBI. The FPI model is suitable for exploring pathogenetic mechanisms of post-traumatic disorders.

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TI The Development of Adolescent Chronic Pain following Traumatic Brain

Injury and Surgery: The Role of Diet and Early Life Stress

SO DEVELOPMENTAL NEUROSCIENCE

LA English

DT Review

DE Microglia; Adverse childhood experiences; HPA axis; Neuroinflammation

ID HIGH-FAT DIET; OXYGEN SPECIES ROS; CHRONIC POSTSURGICAL PAIN; MATERNAL

SEPARATION; RISK-FACTORS; RAT MODEL; POSTTRAUMATIC HEADACHES;

MUSCULOSKELETAL PAIN; CHILDHOOD ABUSE; PRENATAL STRESS

AB Pain is evolutionarily necessary for survival in that it reduces tissue damage by signaling the body to respond to a harmful stimulus. However, in many circumstances, acute pain becomes chronic, and this is often dysfunctional. Adolescent chronic pain is a growing epidemic with an unknown etiology and limited effective treatment options. Given that the relationship between acute pain and chronic pain is not straightforward, there is a need to better understand the factors that contribute to the chronification of pain. Since early life factors are critical to a variety of outcomes in the developmental and adolescent periods, they pose promise as potential mechanisms that may underlie the transition from acute to chronic pain. This review examines two early life factors: poor diet and adverse childhood experiences (ACEs); they may increase susceptibility to the development of chronic pain following surgical procedures or traumatic brain injury (TBI). Beyond their high prevalence, surgical procedures and TBI are ideal models toprospectivelyunderstand mechanisms underlying the transition from acute to chronic pain. Common themes that emerged from the examination of poor diet and ACEs as mechanisms underlying this transition included: prolonged inflammation and microglia activation leading to sensitization of the pain system, and stress-induced alterations to hypothalamic-pituitary-adrenal axis function, where cortisol is likely playing a role in the development of chronic pain. These areas provide promising targets for interventions, the development of diagnostic biomarkers, and suggest that biological treatment strategies should focus on regulating the neuroinflammatory and stress responses in an effort to modulate and prevent the development of chronic pain.

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AU Padilla-Zambrano, HS

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Herrera-Martinez, MP

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Moscote-Salazar, LR

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TI The Role of Microglia in Cerebral Traumatic Injury and its Therapeutic

Implications

SO INDIAN JOURNAL OF NEUROTRAUMA

LA English

DT Review; Early Access

DE microglia; traumatic brain injury; inflammation; neurodegeneration;

diffuse axonal injury

ID BRAIN-INJURY; ACTIVATION; CONSEQUENCES; PLASTICITY

AB Microglia have a variety of functions in the brain such as synaptic remodeling, damage repair of the central nervous system (CNS), and CNS' inflammatory response to peripheral infections. The response depends on the type of insult and infection and includes a range of variety of activation states, the duration of which will decide the outcome. In response to traumatic brain injury (TBI), early activation can lead to early restoration of function, while prolonged and continuous activation can cause neurodegeneration states. Current evidence, however, states that this may not be the case. In this article, we discuss this seldom understood topic of microglia response to TBI, and analyze their distribution, function and possible sites of manipulation. Animal studies have allowed genetic and pharmacological manipulations of microglia activation, in order to define their role. Microglia activation can be remote to the site of injury, and thus their manipulation may play a significant role in the response to any trauma.

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TI Role of Insulin in Neurotrauma and Neurodegeneration: A Review

SO FRONTIERS IN NEUROSCIENCE

LA English

DT Review

DE Alzheimer's disease; insulin; inflammation; microglia; neurons;

Parkinson's disease; spinal cord injury; traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; CENTRAL-NERVOUS-SYSTEM; TIGHT GLYCEMIC CONTROL;

SPINAL-CORD-INJURY; CEREBRAL GLUCOSE-METABOLISM; AMYLOID PRECURSOR

PROTEIN; CORTICAL IMPACT INJURY; C-REACTIVE PROTEIN; INTRANASAL INSULIN;

ALZHEIMERS-DISEASE

AB Insulin is a hormone typically associated with pancreatic release and blood sugar regulation. The brain was long thought to be "insulin-independent," but research has shown that insulin receptors (IR) are expressed on neurons, microglia and astrocytes, among other cells. The effects of insulin on cells within the central nervous system are varied, and can include both metabolic and non-metabolic functions. Emerging data suggests that insulin can improve neuronal survival or recovery after trauma or during neurodegenerative diseases. Further, data suggests a strong anti-inflammatory component of insulin, which may also play a role in both neurotrauma and neurodegeneration. As a result, administration of exogenous insulin, either via systemic or intranasal routes, is an increasing area of focus in research in neurotrauma and neurodegenerative disorders. This review will explore the literature to date on the role of insulin in neurotrauma and neurodegeneration, with a focus on traumatic brain injury (TBI), spinal cord injury (SCI), Alzheimer's disease (AD) and Parkinson's disease (PD).

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TI MSC-derived exosomes promote recovery from traumatic brain injury via

microglia/macrophages in rat

SO AGING-US

LA English

DT Article

DE human adipose-derived mesenchymal stem cells; microglia; exosomes;

traumatic brain injury; neurogenesis

ID MESENCHYMAL STEM-CELLS; EXTRACELLULAR VESICLES; STROMAL CELLS;

IMMUNE-SYSTEM; NEUROGENESIS; MICROGLIA; BIODISTRIBUTION; DYSFUNCTION;

SECRETION; RESPONSES

AB Traumatic brain injury (TBI) is a leading cause of morbidity and mortality in young individuals worldwide. There is currently no effective clinical treatment for TBI, but mesenchymal stem cell-derived exosomes have exhibited promising therapeutic effects. In this study, we performed intracerebroventricular microinjection of human adipose mesenchymal stem cell (hADSC)-derived exosomes (hADSC-ex) in a weight-drop-induced TBI rat model. We found that hADSC-ex promoted functional recovery, suppressed neuroinflammation, reduced neuronal apoptosis, and increased neurogenesis in TBI rats. The therapeutic effects of hADSC-ex were comparable to those of hADSC. Sequential in vivo imaging revealed increasing aggregation of DiR-labeled hADSC-ex in the lesion area. Immunofluorescent staining of coronal brain sections and primary mixed neural cell cultures revealed distinct overlap between CM-DiI-labeled hADSC-ex and microglia/macrophages, indicating that hADSC-ex were mainly taken up by microglia/macrophages. In a lipopolysaccharide-induced inflammatory model, hADSC-ex suppressed microglia/macrophage activation by inhibiting nuclear factor kappa B and P38 mitogen-activated protein kinase signaling. These data suggest that hADSC-ex specifically enter microglia/macrophages and suppress their activation during brain injury, thereby inhibiting inflammation and facilitating functional recovery. They also offer new insight into the cellular targeting, uptake and migration of hADSC-ex, and provide a theoretical basis for new therapeutic strategies for central nervous system diseases.

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TI Repetitive Mild Traumatic Brain Injuries in Mice during Adolescence

Cause Sexually Dimorphic Behavioral Deficits and Neuroinflammatory

Dynamics

SO JOURNAL OF NEUROTRAUMA

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DE concussion; development; microglia; MRI; neuroinflammation

ID WIDESPREAD MICROGLIAL ACTIVATION; ANXIETY-LIKE BEHAVIOR; AMYLOID-BETA;

HIGH-SCHOOL; COMPLEMENT CASCADE; PERSISTENT CHANGES; TELOMERE LENGTH;

SOCIAL-BEHAVIOR; SEX-DIFFERENCES; CONCUSSION

AB Adolescent brain injuries have devastating impacts on lifelong health given that adolescence is a critical period for brain development. Adolescents are susceptible to mild traumatic brain injuries (mTBIs) acquired from collisions in contact sports, which are often sustained in a repetitive nature (repetitive mild traumatic brain injuries; RmTBIs), and cause compounding, sexually dimorphic neurological deficits. Neuroinflammation accompanies RmTBIs and may be a central driving force for chronic neurological decline. To date, the impact of neuroinflammation and sex-specific dynamics during adolescent RmTBIs has been understudied. A lateral impact model (LIM) was developed that mimics the biomechanical forces commonly experienced in human mTBIs. Here, we report novel sexually dimorphic neurobehavioral and -inflammatory responses using LIM to model adolescent RmTBIs. We first subjected adolescent male C57Bl/6 mice to one, three, or five RmTBIs at 24-h intervals and quantified neurobehavioral deficits, and brain volumetric and structural changes by magnetic resonance imaging. Five RmTBIs caused significant motor deficits, increased brain volume in cortex, hippocampus, and corpus callosum, and reduced white matter integrity in the corpus callosum. We then compared neurobehavioral deficits in adolescent male and female mice and observed sex-specific deficits in motor function, whereas both sexes had dysfunction in learning and memory. Flow cytometric quantification of neuroinflammatory responses revealed time- and sex-dependent infiltration of peripheral macrophages and T cells and male-specific decreases in microglia number. Using immunohistochemistry, we report specific microglia density decreases in male mice in the motor cortex and thalamus. We show novel neuroinflammatory responses after adolescent brain injuries that expands the current understanding of RmTBI pathophysiology in this critical neurodevelopmental period.

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TI Sex-Dependent Pathology in the HPA Axis at a Sub-acute Period After

Experimental Traumatic Brain Injury

SO FRONTIERS IN NEUROLOGY

LA English

DT Article

DE hypothalamic-pituitary-adrenal axis; diffuse traumatic brain injury;

sex-differences; glucocorticoid receptors; neuroinflammation;

astrocytosis; microglia; diffuse axonal injury

ID GONADAL HORMONE REPLACEMENT; RAT ADRENAL-CORTEX; ADRENOCORTICAL

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NEUROENDOCRINE; EXPRESSION

AB Over 2.8 million traumatic brain injuries (TBIs) are reported in the United States annually, of which, over 75% are mild TBIs with diffuse axonal injury (DAI) as the primary pathology. TBI instigates a stress response that stimulates the hypothalamic-pituitary-adrenal (HPA) axis concurrently with DAI in brain regions responsible for feedback regulation. While the incidence of affective symptoms is high in both men and women, presentation is more prevalent and severe in women. Few studies have longitudinally evaluated the etiology underlying late-onset affective symptoms after mild TBI and even fewer have included females in the experimental design. In the experimental TBI model employed in this study, evidence of chronic HPA dysregulation has been reported at 2 months post-injury in male rats, with peak neuropathology in other regions of the brain at 7 days post-injury (DPI). We predicted that mechanisms leading to dysregulation of the HPA axis in male and female rats would be most evident at 7 DPI, the sub-acute time point. Young adult age-matched male and naturally cycling female Sprague Dawley rats were subjected to midline fluid percussion injury (mFPI) or sham surgery. Corticotropin releasing hormone, gliosis, and glucocorticoid receptor (GR) levels were evaluated in the hypothalamus and hippocampus, along with baseline plasma adrenocorticotropic hormone (ACTH) and adrenal gland weights. Microglial response in the paraventricular nucleus of the hypothalamus indicated mild neuroinflammation in males compared to sex-matched shams, but not females. Evidence of microglia activation in the dentate gyrus of the hippocampus was robust in both sexes compared with uninjured shams and there was evidence of a significant interaction between sex and injury regarding microglial cell count. GFAP intensity and astrocyte numbers increased as a function of injury, indicative of astrocytosis. GR protein levels were elevated 30% in the hippocampus of females in comparison to sex-matched shams. These data indicate sex-differences in sub-acute pathophysiology following DAI that precede late-onset HPA axis dysregulation. Further understanding of the etiology leading up to late-onset HPA axis dysregulation following DAI could identify targets to stabilize feedback, attenuate symptoms, and improve efficacy of rehabilitation and overall recovery.

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TI The role of NLRP3 in traumatic brain injury and its regulation by

pioglitazone

SO JOURNAL OF NEUROSURGERY

LA English

DT Article

DE astrocytes; inflammasome; microglia; NLRP3; pioglitazone; traumatic

brain injury

ID NF-KAPPA-B; INFLAMMASOME ACTIVATION; CEREBROSPINAL-FLUID; PROTEIN;

EDEMA; MICE; ULTRASOUND; MICROGLIA; MICROBUBBLES; MODEL

AB OBJECTIVE Perilesional edema is a predominant mechanism underlying secondary brain injury after traumatic brain injury (TBI). Perilesional edema is characterized by inflammation, production of proinflammatory cytokines, and migration of peripheral immune cells into the brain. The nucleotide-binding domain and leucine-rich repeat (NLR) family pyrin domain- containing 3 protein (NLRP3) is a key component of secondary injury. Pioglitazone regulates NLRP3 and other inflammatory cytokines. In the present study, the role of NLRP3 and the pharmacological effects of pioglitazone were investigated in animal TBI models.

METHODS Brain contusion was induced in a weight drop model involving 3 groups of mice: C57 BL/6 (sham group), NLRP3 knockout (K/O group), and pioglitazone-treated mice (treatment group). The percentage of brain water content of the 3 groups of mice was compared over a period of time. Western blot, immunohistochemistry, and immunofluorescence analyses were conducted to investigate NLRP3-related inflammasomes and the effects of pioglitazone in the TBI models.

RESULTS Brain edema was the highest on day 3 after TBI in the sham group. Brain edema in both the K/O and the treatment groups was lower than in the sham group. In Western blot, the expression of inflammasomes was higher after TBI in the sham group, but the expression of interleukin-1 beta, caspase-1, and NLRP3 was decreased significantly following treatment with pioglitazone. The expression of GFAP (glial fibrillary acidic protein) and Iba1 was decreased in both the K/O and treatment groups. In addition, confocal microscopy revealed a decrease in microglial cell and astrocyte activation following pioglitazone therapy.

CONCLUSIONS The inflammasome NLRP3 plays a pivotal role in regulating cerebral edema and secondary inflammation. Interestingly, pioglitazone reduced cerebral edema and immune response after TBI by downregulating the effects of NLRP3. These results suggest that the clinical application of pioglitazone may be a neuroprotective strategy in TBI.

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TI Estrogen Attenuates Traumatic Brain Injury by Inhibiting the Activation

of Microglia and Astrocyte-Mediated Neuroinflammatory Responses

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article; Early Access

DE Traumatic brain injury (TBI); 17 beta-estradiol (E2); Neuroinflammation;

Microglia; Astrocyte; Neuroprotection

ID NEUROPROTECTION; RATS; INFLAMMATION; MODEL

AB Traumatic brain injury (TBI), which leads to high mortality and morbidity, is a prominent public health problem worldwide. Neuroinflammation involving microglia and astrocyte activation has been demonstrated to play critical role in the secondary injury induced by TBI. A1 astrocytes, which are induced by activated microglia, can directly kill neurons by secreting neurotoxic complement C3. Estrogen has been proved to possess neuroprotective effects, but the effect and underlying mechanism of estrogen on TBI-induced neuroinflammatory injury remain largely unclear. In this study, we constructed an adult male mouse model of TBI and immediately after injury treated the mice with 17 beta-estradiol (E2) (100 mu g/kg, once every day via intraperitoneal injection) for 3 days. We found that E2 treatment significantly alleviated TBI-induced neurological deficits, neuronal injuries, and brain edema and significantly inhibited Iba1 and GFAP expression, which are markers of microglia and astrocyte activation, respectively. E2 treatment also significantly inhibited TLR4 and NF-kappa B protein expression, and significantly reduced the expression of theproinflammatory factorsIL-1 beta, IL-6, and TNF-alpha. Moreover, E2 treatment significantly decreased the number of complement C3d/GFAP-positive cells and complement C3d protein expression. Taking these results together, we concluded that E2 treatment dramatically alleviates TBI neuroinflammatory injury by inhibiting TLR4/NF-kappa B pathway-mediated microglia and astrocyte activation and neuroinflammation and reducing A1-phenotype neurotoxic astrocyte activation. Our findings indicate that E2 treatment may be a potential therapy strategy for TBI-induced neuroinflammation injury.

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TI Proton extrusion during oxidative burst in microglia exacerbates

pathological acidosis following traumatic brain injury

SO GLIA

LA English

DT Article; Early Access

DE acidosis; chronic neurodegeneration; microglia; neuroinflammation;

traumatic brain injury

ID ACUTE NEUROINFLAMMATION; PH; CHANNELS; NEUTROPHIL; BEHAVIOR; DAMAGE

AB Acidosis is among the least studied secondary injury mechanisms associated with neurotrauma. Acute decreases in brain pH correlate with poor long-term outcome in patients with traumatic brain injury (TBI), however, the temporal dynamics and underlying mechanisms are unclear. As key drivers of neuroinflammation, we hypothesized that microglia directly regulate acidosis after TBI, and thereby, worsen neurological outcomes. Using a controlled cortical impact model in adult male mice we demonstrate that intracellular pH in microglia and extracellular pH surrounding the lesion site are significantly reduced for weeks after injury. Microglia proliferation and production of reactive oxygen species (ROS) were also increased during the first week, mirroring the increase in extracellular ROS levels seen around the lesion site. Microglia depletion by a colony stimulating factor 1 receptor (CSF1R) inhibitor, PLX5622, markedly decreased extracellular acidosis, ROS production, and inflammation in the brain after injury. Mechanistically, we identified that the voltage-gated proton channel Hv1 promotes oxidative burst activity and acid extrusion in microglia. Compared to wildtype controls, microglia lacking Hv1 showed reduced ability to generate ROS and extrude protons. Importantly, Hv1-deficient mice exhibited reduced pathological acidosis and inflammation after TBI, leading to long-term neuroprotection and functional recovery. Our data therefore establish the microglial Hv1 proton channel as an important link that integrates inflammation and acidosis within the injury microenvironment during head injury.

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TI Microglia dynamics in adolescent traumatic brain injury

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DE Synaptic pruning; Glia; Pathophysiology; White matter; Brain maturation;

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AB Repetitive, mild traumatic brain injuries (RmTBIs) are increasingly common in adolescents and encompass one of the largest neurological health concerns in the world. Adolescence is a critical period for brain development where RmTBIs can substantially impact neurodevelopmental trajectories and life-long neurological health. Our current understanding of RmTBI pathophysiology suggests key roles for neuroinflammation in negatively regulating neural health and function. Microglia, the brain's resident immune population, play important roles in brain development by regulating neuronal number, and synapse formation and elimination. In response to injury, microglia activate to inflammatory phenotypes that may detract from these normal homeostatic, physiological, and developmental roles. To date, however, little is known regarding the impact of RmTBIs on microglia function during adolescent brain development. This review details key concepts surrounding RmTBI pathophysiology, adolescent brain development, and microglia dynamics in the developing brain and in response to injury, in an effort to formulate a hypothesis on how the intersection of these processes may modify long-term trajectories.

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TI Comparison between midline and lateral fluid percussion injury in mice

reveals prolonged but divergent cortical neuroinflammation

SO BRAIN RESEARCH

LA English

DT Article

DE Traumatic brain injury; Fluid percussion injury; Neuroinflammation;

Microglia

ID TRAUMATIC BRAIN-INJURY; MODEL; ACTIVATION; DISRUPTION

AB Animal models are critical for determining the mechanisms mediating traumatic brain injury-induced (TBI) neuropathology. Fluid percussion injury (FPI) is a widely used model of brain injury typically applied either midline or parasagittally (lateral). Midline FPI induces a diffuse TBI, while lateral FPI induces both focal cortical injury (ipsilateral hemisphere) and diffuse injury (contralateral hemisphere). Nonetheless, discrete differences in neuroinflammation and neuropathology between these two versions of FPI remain unclear. The purpose of this study was to compare acute (4-72 h) and subacute (7 days) neuroinflammatory responses between midline and lateral FPI. Midline FPI resulted in longer righting reflex times than lateral FPI. At acute time points, the inflammatory responses to the two different injuries were similar. For instance, there was evidence of monocytes and cytokine mRNA expression in the brain with both injuries acutely. Midline FPI had the highest proportion of brain monocytes and highest IL-1 beta/TNF alpha mRNA expression 24 h later. NanoString nCounter analysis 7 days post-injury revealed robust and prolonged expression of inflammatory-related genes in the cortex after midline FPI compared to lateral FPI; however, Iba-1 cortical immunoreactivity was increased with lateral FPI. Thus, midline and lateral FPI caused similar cortical neuroinflammatory responses acutely and mRNA expression of inflammatory genes was detectable in the brain 7 days later. The primary divergence was that inflammatory gene expression was greater and more diverse subacutely after midline FPI. These results provide novel insight to variations between midline and lateral FPI, which may recapitulate unique temporal pathogenesis.

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TI Cellular infiltration in traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Review

DE Neuroinflammation; Cellular infiltration; Traumatic brain injury;

Microglial dynamics

ID INTERLEUKIN-1 RECEPTOR ANTAGONIST; MICROGLIA/MACROPHAGE POLARIZATION

DYNAMICS; INTRACEREBRAL INFLAMMATORY RESPONSE; CORTICAL IMPACT INJURY;

CENTRAL-NERVOUS-SYSTEM; REACTIVE ASTROCYTES; NEUTROPHIL INFILTRATION;

MICROGLIAL ACTIVATION; NITRIC-OXIDE; HEAD-INJURY

AB Traumatic brain injury leads to cellular damage which in turn results in the rapid release of damage-associated molecular patterns (DAMPs) that prompt resident cells to release cytokines and chemokines. These in turn rapidly recruit neutrophils, which assist in limiting the spread of injury and removing cellular debris. Microglia continuously survey the CNS (central nervous system) compartment and identify structural abnormalities in neurons contributing to the response. After some days, when neutrophil numbers start to decline, activated microglia and astrocytes assemble at the injury site-segregating injured tissue from healthy tissue and facilitating restorative processes. Monocytes infiltrate the injury site to produce chemokines that recruit astrocytes which successively extend their processes towards monocytes during the recovery phase. In this fashion, monocytes infiltration serves to help repair the injured brain. Neurons and astrocytes also moderate brain inflammation via downregulation of cytotoxic inflammation. Depending on the severity of the brain injury, T and B cells can also be recruited to the brain pathology sites at later time points.

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TI Functional consequences of a close encounter between microglia and

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ID PLURIPOTENT STEM-CELLS; CENTRAL-NERVOUS-SYSTEM; SPINAL-CORD-INJURY;

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LANGERHANS CELLS

AB Neuroinflammation is recognized as an important factor contributing to the development and progression of several central nervous system (CNS) disorders. Upon CNS trauma or disease, parenchymal microglia highly proliferate and accumulate in and around the lesion site. In addition, blood-derived monocytes can infiltrate the inflamed CNS in response to cellular damage and/or a compromised blood-brain barrier. Both microglia and infiltrating monocytes are characterized by multiple functional states and can either display highly proinflammatory properties or promote resolution of inflammation and tissue regeneration. Despite sharing some basic immunologic functions, microglia and monocytes display many distinctive features, which ultimately define their contribution to neuropathology. Understanding how the innate immune system participates to brain disease is imperative to identify novel treatment options for CNS inflammatory disorders. In this context, existing and newly developed in vitro platforms for disease modeling are fundamental tools to investigate and modulate microglia and monocyte immune functions within a specific neuropathologic context. In this review, we first briefly summarize the current knowledge on microglia and monocyte ontogenesis, as well as their complex and interconnected contributions to the development of various CNS pathologies. Following the well-recognized concept that both microglia and monocytes can either exert neuroprotective functions or exacerbate tissue damage, we provide a comprehensive overview of cellular models currently available for in vitro study of neuroinflammatory responses. In this context, we highlight how simplified single-cell models may not always correctly recapitulate in vivo biology, hence future research should move toward novel models with higher and multicellular complexity.

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TI Traumatic brain injury in mice induces changes in the expression of the

XCL1/XCR1 and XCL1/ITGA9 axes

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DE TBI; Chemokine; XCL1; XCR1; ITGA9; Microglia; Astroglia

ID CHEMOKINE XCL1; INTEGRIN; RECEPTOR; CELLS; XCR1; ALPHA-9; FRACTALKINE;

ACTIVATION; INHIBITION; LIGANDS

AB Background Every year, millions of people suffer from various forms of traumatic brain injury (TBI), and new approaches with therapeutic potential are required. Although chemokines are known to be involved in brain injury, the importance of X-C motif chemokine ligand 1 (XCL1) and its receptors, X-C motif chemokine receptor 1 (XCR1) and alpha-9 integrin (ITGA9), in the progression of TBI remain unknown.

Methods Using RT-qPCR/Western blot/ELISA techniques, changes in the mRNA/protein levels of XCL1 and its two receptors, in brain areas at different time points were measured in a mouse model of TBI. Moreover, their cellular origin and possible changes in expression were evaluated in primary glial cell cultures.

Results Studies revealed the spatiotemporal upregulation of the mRNA expression of XCL1, XCR1 and ITGA9 in all the examined brain areas (cortex, thalamus, and hippocampus) and at most of the evaluated stages after brain injury (24 h; 4, 7 days; 2, 5 weeks), except for ITGA9 in the thalamus. Moreover, changes in XCL1 protein levels occurred in all the studied brain structures; the strongest upregulation was observed 24 h after trauma. Our in vitro experiments proved that primary murine microglial and astroglial cells expressed XCR1 and ITGA9, however they seemed not to be a main source of XCL1.

Conclusions These findings indicate that the XCL1/XCR1 and XCL1/ITGA9 axes may participate in the development of TBI. The XCL1 can be considered as one of the triggers of secondary injury, therefore XCR1 and ITGA9 may be important targets for pharmacological intervention after traumatic brain injury.

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TI The ketone ester, 3-hydroxybutyl-3-hydroxybutyrate, attenuates

neurobehavioral deficits and improves neuropathology following

controlled cortical impact in male rats

SO NUTRITIONAL NEUROSCIENCE

LA English

DT Article; Early Access

DE Astrocyte; controlled cortical impacy; contusion volume; ketone ester

supplementation; lesion volume; microglia; neuroinflammation; rat;

revised neurobehavioral severity scale; traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; BETA-HYDROXYBUTYRATE; KETOGENIC DIET; DYNAMIC

CHANGES; CELL-DEATH; RECOVERY; MICE; PROMOTES; GLUCOSE; MODEL

AB Traumatic brain injury (TBI) is a leading cause of human death and disability with no effective therapy to fully prevent long-term neurological deficits in surviving patients. Ketone ester supplementation is protective in animal models of neurodegeneration, but its efficacy against TBI pathophysiology is unknown. Here, we assessed the neuroprotective effect of the ketone monoester, 3-hydroxybutyl-3-hydroxybutyrate, (KE) in male Sprague Dawley rats (n=32). TBI was induced using the controlled cortical impact (CCI) with Sham animals not receiving the brain impact. KE was administered daily by oral gavage (0.5 ml/kg/day) and provided ad libitum at 0.3% (v/v) in the drinking water. KE supplementation started immediately after TBI and lasted for the duration of the study. Motor and sensory deficits were assessed using the Neurobehavioral Severity Scale-Revised (NSS-R) at four weeks post-injury. The NSS-R total score in CCI + KE (1.2 +/- 0.4) was significantly lower than in CCI + water (4.4 +/- 0.5). Similarly, the NSS-R motor scores in CCI + KE (0.6 +/- 0.7) were significantly lower than CCI + water (2.9 +/- 1.5). Although the NSS-R sensory score in the CCI + KE group (0.5 +/- 0.2) was significantly lower compared to CCI + water (1.8 +/- 0.4), no difference was observed between CCI + water and Sham + water (1.0 +/- 0.2) groups. The lesion volume was smaller in the CCI + KE (10 +/- 3 mm(3)) compared to CCI + water (47 +/- 11 mm(3); p < 0.001). KE significantly decreased Iba1(+) stained areas in the cortex and hippocampus, and GFAP(+) stained areas in all brain regions analyzed - prefrontal cortex, hippocampus, cortex, amygdala (p < 0.01). In summary, our results indicate that KE can protect against TBI-induced morphological and functional deficits when administered immediately after an insult.

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TI Increased Behavioral Deficits and Inflammation in a Mouse Model of

Co-Morbid Traumatic Brain Injury and Post-Traumatic Stress Disorder

SO ASN NEURO

LA English

DT Article

DE behavior; cognition; memory; astrocytes; macrophage; microglia

AB Comorbid post-traumatic stress disorder with traumatic brain injury (TBI) produce more severe affective and cognitive deficits than PTSD or TBI alone. Both PTSD and TBI produce long-lasting neuroinflammation, which may be a key underlying mechanism of the deficits observed in co-morbid TBI/PTSD. We developed a model of co-morbid TBI/PTSD by combining the closed head (CHI) model of TBI with the chronic variable stress (CVS) model of PTSD and examined multiple behavioral and neuroinflammatory outcomes. Male C57/Bl6 mice received sham treatment, CHI, CVS, CHI then CVS (CHI -> CVS) or CVS then CHI (CVS -> CHI). The CVS -> CHI group had deficits in Barnes maze or active place avoidance not seen in the other groups. The CVS -> CHI, CVS and CHI -> CVS groups displayed increased basal anxiety level, based on performance on elevated plus maze. The CVS -> CHI had impaired performance on Barnes Maze, and Active Place Avoidance. These performance deficits were strongly correlated with increased hippocampal Iba-1 level an indication of activated MP/MG. These data suggest that greater cognitive deficits in the CVS -> CHI group were due to increased inflammation. The increased deficits and neuroinflammation in the CVS -> CHI group suggest that the order by which a subject experiences TBI and PTSD is a major determinant of the outcome of brain injury in co-morbid TBI/PTSD.

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TI The CCL2/CCL7/CCL12/CCR2 pathway is substantially and persistently

upregulated in mice after traumatic brain injury, and CCL2 modulates the

complement system in microglia

SO MOLECULAR AND CELLULAR PROBES

LA English

DT Article

DE CCL2; Complement system; Lectin pathway; Microglia

ID CHEMOATTRACTANT PROTEIN-1 MCP-1; CHEMOKINE RECEPTOR CCR2;

MANNOSE-BINDING LECTIN; NEUROPATHIC PAIN; COGNITIVE IMPAIRMENTS;

EMBRYONIC-DEVELOPMENT; FRACTALKINE RECEPTOR; GENE-EXPRESSION; IN-VIVO;

DEFICIENCY

AB Traumatic brain injury (TBI) is the leading cause of death in the global population. Disturbed inflammatory processes after TBI exacerbate secondary brain injury and contribute to unfavorable outcomes. Multiple inflammatory events that accompany brain trauma, such as glial activation, chemokine release, or the initiation of the complement system cascade, have been identified as potential targets for TBI treatment. However, the participation of chemokines in the complement activation remains unknown. Our studies sought to determine the changes in the expression of the molecules involved in the CCL2/CCL7/CCL12/CCR2 pathway in the injured brain and the effect of CCL2, CCL7, and CCL12 (10, 100, and 500 ng/mL) on the classic and lectin complement pathways and inflammatory factors in microglial cell cultures. Brain injury in mice was modeled by controlled cortical impact (CCI). Our findings indicate a time-dependent upregulation of CCL2, CCL7, and CCL12 at the mRNA and protein levels within the cortex, striatum, and/or thalamus beginning 24 h after the trauma. The analysis of the expression of the receptor of the tested chemokines, CCR2, revealed its substantial upregulation within the injured brain areas mainly on the mRNA level. Using primary cortical microglial cell cultures, we observed a substantial increase in the expression of CCL2, CCL7, and CCL12 after 24 h of LPS (100 ng/mL) treatment. CCL2 stimulation of microglia increased the level of IL-1 beta mRNA but did not influence the expression of IL-18, IL-6, and IL-10. Moreover, CCL2 significantly increased the expression of Iba1, a marker of microglia activation. CCL2 and CCL12 upregulated the expression of C1qa but did not influence the expression of C1ra and C1s1 (classical pathway); moreover, CCL2 increased ficolin A expression and reduced collectin 11 expression (lectin pathway). Additionally, we observed the downregulation of pentraxin 3, a modulator of the complement cascade, after CCL2 and CCL12 treatment. We did not detect the expression of ficolin B, Mbl1, and Mbl2 in microglial cells. Our data identify CCL2 as a modulator of the classical and lectin complement pathways suggesting that CCL2 may be a promising target for pharmacological intervention after brain injury. Moreover, our study provides evidence that CCL2 and two other CCR2 ligands may play a role in the development of changes in TBI.

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TI Neuroinflammatory responses of microglia in central nervous system

trauma

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Review

DE Central nervous system; inflammation; microglia; spinal cord injury;

traumatic brain injury

ID SPINAL-CORD-INJURY; BRAIN-INJURY; INFLAMMATORY RESPONSE; MACROPHAGE

SUBSETS; CROSS-TALK; ACTIVATION; COMPLEMENT; CYTOKINES; RECOVERY; CNS

AB Although relatively few in number compared to astrocytes and neurons, microglia demonstrate multiple, varied neuroimmunological functions in the central nervous system during normal and pathological states. After injury to the brain or spinal cord, microglia express beneficial pro- and anti-inflammatory phenotypes at various stages of recovery. However, prolonged microglial activation following injury has been linked to impaired parenchymal healing and functional restoration. The nature and magnitude of microglial response to injury relates in part to peripheral immune cell invasion, extent of tissue damage, and the local microenvironment.

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AF Zhu, Ming-min

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Cao, Lu-xi

Zhang, Yi-min

TI Manual acupuncture relieves microglia-mediated neuroinflammation in a

rat model of traumatic brain injury by inhibiting the RhoA/ROCK2 pathway

SO ACUPUNCTURE IN MEDICINE

LA English

DT Article

DE manual acupuncture; microglia; neuroinflammation; RhoA; ROCK2 signaling

pathway; traumatic brain injury

AB Objective:

To investigate the regulatory mechanism of manual acupuncture (MA) on microglial polarization-mediated neuroinflammation after traumatic brain injury (TBI), focusing on the RhoA/Rho-associated coiled coil-forming protein kinase (ROCK2) pathway.

Methods:

Sprague Dawley (SD) rats were used to generate a TBI model using Feeney's freefall epidural impact method. MA was performed on half of the TBI model rats, while the others remained untreated. Acupuncture was administered at GV15, GV16, GV20, GV26, and LI4. At the end of the intervention, rat brain tissue samples were collected, and the microglial M1 polarization status was observed by immunofluorescence labeling of CD86, an M1 microglia-specific protein. RhoA/ROCK2 signaling components were detected by quantitative real-time polymerase chain reaction (qRT-PCR) and Western blotting. An enzyme-linked immunosorbent assay (ELISA) was used to detect the expression levels of inflammatory factors.

Results:

Compared with normal rats, the CD86 expression density in the untreated TBI model rats was high and showed an aggregated expression pattern. The genes and proteins of the RhoA/ROCK2 signaling pathway were highly expressed, and inflammatory factors were significantly increased. The CD86 expression density in TBI rats after MA was reduced compared to that in untreated TBI rats and showed a scattered distribution. The expression of RhoA/ROCK2 signaling pathway genes and proteins was also significantly reduced, and inflammatory factors were decreased.

Conclusion:

These results show that MA may inhibit M1 polarization of microglia by regulating the RhoA/ROCK2 signaling pathway, thereby reducing neuroinflammation in TBI.

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TI Bexarotene promotes microglia/macrophages - Specific brain - Derived

Neurotrophic factor expression and axon sprouting after traumatic brain

injury

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article

DE Traumatic brain injury; Brain derived neurotrophic factor; Axon

sprouting; Microglia/macrophages

ID WHITE-MATTER; OLIGODENDROCYTE DIFFERENTIATION; MICROGLIAL ACTIVATION;

POLARIZATION DYNAMICS; FUNCTIONAL RECOVERY; NEURONAL SURVIVAL;

NEUROPROTECTION; RAT; PROLIFERATION; REGENERATION

AB Traumatic brain injury (TBI) has been regarded as one of the leading cause of injury-related death and disability. White matter injury after TBI is characterized by axon damage and demyelination, resulting in neural network impairment and neurological deficit. Brain-derived neurotrophic factor (BDNF) can promote white matter repair. The activation of peroxisome proliferator-activated receptor gamma (PPAR gamma) has been reported to promote microglia/macrophages towards anti-inflammatory state and therefore to promote axon regeneration. Bexarotene, an agonist of retinoid X receptor (RXR), can activate RXR/PPAR gamma heterodimers. The aim of the present study was to identify the effect of bexarotene on BDNF in microglia/macrophages and axon sprouting after TBI in mice. Bexarotene was administered intraperitoneally in C57BL/6 mice undergoing controlled cortical impact (CCI). PPAR gamma dependency was determined by intraperitoneal administration of a PPAR gamma antagonist T0070907. We found that bexarotene promoted axon regeneration indicated by increased growth associated protein 43 (GAP43) expression, myelin basic protein (MBP) expression, and biotinylated dextran amine (BDA)(+) axon sprouting. Bexarotene also increased microglia/macrophages-specific brain derived neurotrophic factor (BDNF) expression after TBI. In addition, bexarotene reduced the number of pro-inflammatory microglia/macrophages while increased the number of anti-inflammatory microglia/macrophages after TBI. Moreover, bexaortene inhibited pro-inflammatory cytokine secretion. In addition, bexarotene treatment improved neurological scores and cognitive function of CCI-injured mice. These effects of bexarotene were partially abolished by T0070907. In conclusion, bexarotene promotes axon sprouting, increases microglia/macrophages-specific BDNF expression, and induces microglia/macrophages from a pro-inflammatory state towards an anti-inflammatory one after TBI at least partially in a PPAR gamma-dependent manner.

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Zhao, Peng-xiang

Pan, Shu-yi

Ma, Xue-mei

TI Hydrogen inhalation inhibits microglia activation and neuroinflammation

in a rat model of traumatic brain injury

SO BRAIN RESEARCH

LA English

DT Article

DE Traumatic brain injury; Hydrogen inhalation; Microglia activation;

Neuroinflammation; Cytokines

ID DAMAGE; CELLS

AB Traumatic brain injury (TBI) is a major cause of mortality and disability worldwide. To date, therapies to treat any forms of TBI are still limited. Recent studies have demonstrated the potential neuroprotective effects of molecular hydrogen on TBI. Although it has been demonstrated that hydrogen inhalation (HI) for about 5 hrs immediately after TBI has a beneficial effect on brain injury, the most effective intervention procedure in the treatment of TBI remains unknown. The mechanism underlying the neuroprotective effects of HI on TBI also needs to be further investigated.

Our results showed that inhalation of 4% hydrogen during the first day after TBI was the most effective hydrogen intervention procedure in the treatment of TBI. Pathological examination showed that HI could attenuate TBI-induced reactive astrocytosis and microglial activation. Nissl staining demonstrated a significant decrease in the number of nissl-stained dark neurons (N-DNs) in HI group compared to TBI group at 2 h post-TBI, and the TBI-induced neuronal loss was attenuated by HI at day 3 post-TBI. IHC staining showed that HI resulted a decrease in CD16-positive cells and a further increase in CD206-positive cells as compared to TBI group. Multiplex cytokine assay demonstrated the most profound regulatory effects induced by HI on the levels of IL-12, IFN-gamma, and GM-CSF at 24 h post-TBI, which confirmed the inhibitory effect of hydrogen on microglia activation.

We concluded that inhalation of 4% hydrogen during the first day after TBI was the most effective intervention procedure in the treatment of TBI. Our results also showed that hydrogen may exert its protective effects on TBI via inhibition of microglia activation and neuroinflammation.

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PT J

AU Mao, LL

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TI Ethyl pyruvate improves white matter remodeling in rats after traumatic

brain injury

SO CNS NEUROSCIENCE & THERAPEUTICS

LA English

DT Article; Early Access

DE ethyl pyruvate; microglia; traumatic brain injury; white matter injury

ID MODULATING MICROGLIA/MACROPHAGE POLARIZATION; MODEL; INHIBITION;

PROTECTS; DAMAGE

AB Background Severe traumatic brain injury (TBI) results in long-term neurological deficits associated with white matter injury (WMI). Ethyl pyruvate (EP) is a simple derivative of the endogenous energy substrate pyruvate with neuroprotective properties, but its role in recovery from WMI has not been explored.

Aims This study examines the effect of EP treatment on rats following TBI using behavioral tests and white matter histological analysis up to 28 days post-injury.

Materials and Methods Anaesthetised adult rats were subjected to TBI by controlled cortical impact. After surgery, EP or Ringers solution (RS) was administrated intraperitoneally at 15 min after TBI and again at 12, 24, 36, 48, and 60 h after TBI. Sensorimotor deficits were evaluated up to day 21 after TBI by four independent tests. Immunofluorescence and transmission electron microscopy (TEM) were performed to assess white matter injury. Microglia activation and related inflammatory molecules were examined up to day 14 after TBI by immunohistochemistry or real-time PCR.

Results Here, we demonstrate that EP improves sensorimotor function following TBI as well as improves white matter outcomes up to 28 d after TBI, as shown by reduced myelin loss. Furthermore, EP administration during the acute phase of TBI recovery shifted microglia polarization toward the anti-inflammatoryM2 phenotype, modulating the release of inflammatory-related factors.

Conclusion EP treatment may protect TBI-induced WMI via modulating microglia polarization toward M2.

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U2 15

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TI Combination therapy with Treg and mesenchymal stromal cells enhances

potency and attenuation of inflammation after traumatic brain injury

compared to monotherapy

SO STEM CELLS

LA English

DT Article; Early Access

DE cell therapy; mesenchymal stem cell; mesenchymal stromal cell;

microglia; neuroinflammation; regulatory T&#8201; cell; traumatic brain

injury

ID STEM-CELLS; MICROGLIA; NEUROINFLAMMATION

AB The inflammatory response after traumatic brain injury (TBI) can lead to significant secondary brain injury and chronic inflammation within the central nervous system. Cell therapies, including mesenchymal stromal cells (MSC), have led to improvements in animal models of TBI and are under investigation in human trials. One potential mechanism for the therapeutic potential of MSC is their ability to augment the endogenous response of immune suppressive regulatory T cells (Treg). We have recently shown that infusion of human cord blood Treg decreased chronic microgliosis after TBI and altered the systemic immune response in a rodent model. These cells likely use both overlapping and distinct mechanisms to modulate the immune system; therefore, combining Treg and MSC as a combination therapy may confer therapeutic benefit over either monotherapy. However, investigation of Treg + MSC combination therapy in TBI is lacking. In this study, we compared the ability MSC + Treg combination therapy, as well as MSC and Treg monotherapies, to inhibit the neuroinflammatory response to TBI in vivo and in vitro. Treg + MSC combination therapy demonstrated increased potency to reduce the neuro- and peripheral inflammatory response compared to monotherapy; furthermore, the timing of infusion proved to be a significant variable in the efficacy of both MSC monotherapy and Treg + MSC combination therapy in vivo and in vitro.

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NR 42

TC 16

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PI OXFORD

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JI Stem Cells

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TI Targeting Microglial Polarization to Improve TBI Outcomes

SO CNS & NEUROLOGICAL DISORDERS-DRUG TARGETS

LA English

DT Review

DE Traumatic brain injury; inflammation; microglia; pyroptosis;

neuroprotection; post-traumatic stress disorder (PTSD)

ID TRAUMATIC BRAIN-INJURY; INTERLEUKIN-1 RECEPTOR ANTAGONIST; HISTONE

DEACETYLASE INHIBITORS; COMPUTED-TOMOGRAPHY; CEREBROSPINAL-FLUID;

LIFETIME COSTS; UNITED-STATES; ACTIVATION; INFLAMMASOME; DEPRESSION

AB Traumatic Brain Injury (TBI) is still the worldwide leading cause of mortality and morbidity in young adults. Improved safety measures and advances in critical care have increased chances of surviving a TBI, however, numerous secondary mechanisms contribute to the injury in the weeks and months that follow TBI. The past 4 decades of research have addressed many of the metabolic impairments sufficient to mitigate mortality, however, an enduring secondary mechanism, i.e. neuroinflammation, has been intractable to current therapy. Neuroinflammation is particularly difficult to target with pharmacological agents due to lack of specificity, the blood brain barrier, and an incomplete understanding of the protective and pathologic influences of inflammation in TBI. Recent insights into TBI pathophysiology have established microglial activation as a hallmark of all types of TBI. The inflammatory response to injury is necessary and beneficial while the death of activated microglial is not. This review presents new insights on the therapeutic and maladaptive features of the immune response after TBI with an emphasis on microglial polarization, followed by a discussion of potential targets for pharmacologic and non-pharmacologic treatments. In aggregate, this review presents a rationale for guiding TBI inflammation towards neural repair and regeneration rather than secondary injury and degeneration, which we posit could improve outcomes and reduce lifelong disease burden in TBI survivors.

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TI Integration of single-cell and bulk RNA sequencing data reveals key cell

types and regulators in traumatic brain injury

SO MATHEMATICAL BIOSCIENCES AND ENGINEERING

LA English

DT Article

DE traumatic brain injury; single-cell RNA-seq; cell-cell communication;

TYROBP causal network; microglia

ID MESSENGER-RNA; ANIMAL-MODELS; INFLAMMATION; MICROGLIA;

NEUROINFLAMMATION; NETWORKS; RECEPTOR; CORTEX; TREM2

AB Traumatic brain injury (TBI) is a leading cause of disability and mortality worldwide, whose symptoms ranging from mild to severe, even life-threatening. However, specific cell types and key regulators involved in traumatic brain injury have not been well elucidated. In this study, utilizing single-cell RNA-seq (scRNA-seq) data from mice with TBI, we have successfully identified and characterized 13 cell populations including astrocytes, oligodendrocyte, newly formed oligodendrocytes, microglia, two types of endothelial cells, five types of excitatory and two types of inhibitory neurons. Differential expression analysis and gene set enrichment analysis (GSEA) revealed the upregulation of microglia and endothelial markers, along with the downregulation of markers of excitatory neurons in TBI. The cell-cell communication analysis revealed that microglia and endothelial cell might interact through the interaction of Icam1-Il2rg and C1qa-Cd93, and microglia might also communicate with each other via Icam1-Itagm. The autocrine ligand-receptor in microglia might result in activation of TYROBP causal network via Icam1-Itgam. The cell-cell contact between microglia and endothelial cell might activate integrin signaling pathways. Moreover, we also found that genes involved in microglia activation were highly downregulated in Tyrobp/Dap12-deficien network in microglia might be a candidate therapeutic target in TBI. In contrast, the excitatory neurons were involved in maintaining normal brain function, and their inactivation might cause dysfunction of nervous system in TBI patients. In conclusion, the present study has discerned major cell types such as microglia, endothelial cells and excitatory neurons, and revealed key regulator such as TYROBP, C1QA, and CD93 in TBI, which shall improve our understanding of the pathogenesis of TBI.

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TI The synapse in traumatic brain injury

SO BRAIN

LA English

DT Review

DE synaptopathy; synaptome; astrocyte; inflammation; microglia

ID OXIDATIVE STRESS; POSTSYNAPTIC DENSITY; CEREBROSPINAL-FLUID; PROTEOMIC

ANALYSIS; AXONAL INJURY; PET TRACER; REACTIVE SYNAPTOGENESIS;

ALZHEIMERS-DISEASE; DENDRITIC SPINES; CORTICAL IMPACT

AB Traumatic brain injury (TBI) is a leading cause of death and disability worldwide and is a risk factor for dementia later in life. Research into the pathophysiology of TBI has focused on the impact of injury on the neuron. However, recent advances have shown that TBI has a major impact on synapse structure and function through a combination of the immediate mechanical insult and the ensuing secondary injury processes, leading to synapse loss. In this review, we highlight the role of the synapse in TBI pathophysiology with a focus on the confluence of multiple secondary injury processes including excitotoxicity, inflammation and oxidative stress. The primary insult triggers a cascade of events in each of these secondary processes and we discuss the complex interplay that occurs at the synapse. We also examine how the synapse is impacted by traumatic axonal injury and the role it may play in the spread of tau after TBI. We propose that astrocytes play a crucial role by mediating both synapse loss and recovery. Finally, we highlight recent developments in the field including synapse molecular imaging, fluid biomarkers and therapeutics. In particular, we discuss advances in our understanding of synapse diversity and suggest that the new technology of synaptome mapping may prove useful in identifying synapses that are vulnerable or resistant to TBI.

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TI Elevated serum and urine levels of progranulin (PGRN) as a predictor of

microglia activation in the early phase of traumatic brain injury: a

further link with the development of neurodegenerative diseases

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AB Traumatic brain injury (TBI) is a frequent finding during forensic autopsies and neuropathological examinations in medico-legal practices. Despite the unprecedented attention currently focused on TBI pathogenesis, there is a need to improve its diagnostics through the use of novel biomarkers to facilitate detection, treatment, and prognosis. Recently, growth factor progranulin (PGRN) has attracted significant attention because of its neurotrophic and anti-inflammatory activities. The role of PGRN in TBI has not been widely discussed, although PGRN-related neuroinflammatory and neurodegenerative phenomena have been described. The aim of this study was to identify PGRN concentration levels in biofluids and examine PGRN and CD68 protein expression in brain tissue using immunohistochemical staining in individuals with fatal TBI in its early phase. The study was performed using cases (n = 30) of fatal head injury and control cases (n = 30) of sudden death. The serum and urine were collected within similar to 24 h after death and compared using the ELISA test, where brain specimens were stained with anti-PGRN and anti-CD68 antibodies. In our study, we observed elevated concentration levels of PGRN in the serum and urine of TBI individuals in the early phase of TBI. These changes were accompanied by increased expression of PGRN in the frontal cortex (1st-3rd layers), in which anti-CD68 immunostaining revealed disseminated cortical microglia activation. The possible implementation of performing such assays offers a novel and interesting tool for investigation and research regarding TBI diagnosis and pathogenesis. Furthermore, the above-mentioned surrogate biofluid assays may be useful in clinical prognosis and risk calculation of non-fatal cases of TBI, considering the development of neurodegenerative conditions of TBI individuals.

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TI Niche Cells Crosstalk In Neuroinflammation After Traumatic Brain Injury

SO INTERNATIONAL JOURNAL OF BIOLOGICAL SCIENCES

LA English

DT Review

DE Traumatic brain injury; Neuroinflammation; Nerve regeneration;

Extracellular vesicles; Microglia; Astrocytes; Neural stem cell (NSC)

ID NEURAL STEM/PROGENITOR CELLS; MICROGLIA EMERGE; STEM-CELL; NEURONS;

INFLAMMATION; ACTIVATION; EXPRESSION; NEUROGENESIS; PROMOTES; DEFICITS

AB Traumatic brain injury (TBI) is recognized as the disease with high morbidity and disability around world in spite of the work ongoing in neural protection. Due to heterogeneity among the patients, it's still hard to acquire satisfying achievements in clinic. Neuroinflammation, which exists since primary injury occurs, with elusive duality, appear to be of significance from recovery of injury to neurogenesis. In recent years, studied have revealed that communication in neurogenic niche is more than "cell to cell" communication, and study on NSCs represent it as central role in the progress of neural regeneration. Hence, the neuroinflammation-affecting crosstalk after TBI, and clarifying definitive role of NSCs in the course of regeneration is a promising subject for researchers, for its great potential in overcoming the frustrating status quo in clinic, promoting welfare of TBI patient.

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TI Repetitive Traumatic Brain Injury Causes Neuroinflammation before Tau

Pathology in Adolescent P301S Mice

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE concussion; tau; adolescents; traumatic brain injury; CTE; microglia

AB Repetitive closed head injury (rCHI) is commonly encountered in young athletes engaged in contact and collision sports. Traumatic brain injury (TBI) including rCHI has been reported to be an important risk factor for several tauopathies in studies of adult humans and animals. However, the link between rCHI and the progression of tau pathology in adolescents remains to be elucidated. We evaluated whether rCHI can trigger the initial acceleration of pathological tau in adolescent mice and impact the long-term outcomes post-injury. To this end, we subjected adolescent transgenic mice expressing the P301S tau mutation to mild rCHI and assessed tau hyperphosphorylation, tangle formation, markers of neuroinflammation, and behavioral deficits at 40 days post rCHI. We report that rCHI did not accelerate tau pathology and did not worsen behavioral outcomes compared to control mice. However, rCHI induced cortical and hippocampal microgliosis and corpus callosum astrocytosis in P301S mice by 40 days post-injury. In contrast, we did not find significant microgliosis or astrocytosis after rCHI in age-matched WT mice or sham-injured P301S mice. Our data suggest that neuroinflammation precedes the development of Tau pathology in this rCHI model of adolescent repetitive mild TBI.

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TI Reactive pericytes in early phase are involved in glial activation and

late-onset hypersusceptibility to pilocarpine-induced seizures in

traumatic brain injury model mice

SO JOURNAL OF PHARMACOLOGICAL SCIENCES

LA English

DT Article

DE Traumatic brain injury; Reactive pericytes; PDGFR beta; Imatinib;

Microglia

ID STATUS EPILEPTICUS; AXONAL INJURY; EPILEPSY; DECREASES

AB In this study, among neurovascular unit (NVU) cells, we focused on pericyte reactivity in mice subjected to controlled cortical impact (CCI) to understand how traumatic brain injury (TBI) causes uncoordinated crosstalk in the NVU and alters neuronal activity. Histological analyses of brain pericytes, microglia and astrocytes were performed for up to 28 days after CCI in the injured ipsilateral hippocampus. To evaluate altered neuronal activity caused by CCI, we measured seizure susceptibility to a sub-threshold dose of pilocarpine on postoperative day 7, 14, 21 and 28. Platelet-derived growth factor receptor (PDGFR) beta immunoreactivity in pericytes significantly increased from 1 h to 4 days after CCI. The expression of Iba1 and GFAP, as markers of microglia and astrocytes, respectively, increased from 4 to 28 days after CCI. The severity of seizure induced by pilocarpine gradually increased, becoming significant at 28 days after CCI. Then, we treated CCI mice with an inhibitor of PDGFR signaling, imatinib, during the postoperative day 0-4 period. Imatinib lowered seizure susceptibility to pilocarpine and suppressed microglial activation in the injured hippocampus at postoperative day 28. These findings indicate that brain pericytes with rapidly increased PDGFR beta expression may drive TBI-induced dysregulation of NVU function and brain hyperexcitability. (C) 2020 The Authors. Production and hosting by Elsevier B.V. on behalf of Japanese Pharmacological Society.

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TI Behavioral responses following repeated bilateral frontal region closed

head impacts and fear conditioning in male and female mice

SO BRAIN RESEARCH

LA English

DT Article

DE Traumatic brain injury; Concussion; Fear conditioning; Frontal cortex;

Microglia; Activity; Locomotor function; Mouse; Female; Sex factors

ID TRAUMATIC BRAIN-INJURY; INDUCED AXONAL INJURY; UNITED-STATES; MOUSE

MODEL; COGNITIVE PERFORMANCE; GENDER-DIFFERENCES; SEX-DIFFERENCES;

RISK-FACTORS; OPTIC-NERVE; MILD

AB The frontal lobes are among the most vulnerable sites in traumatic brain injuries. In the current study, a balanced 2 x 2 x 2 design (n = 18 mice/group), female and male C57Bl/6J mice received repeated bilateral frontal concussive brain injury (frCBI) and underwent fear conditioning (FC) to assess how injured mice respond to adverse conditions. Shocks received during FC impacted behavior on all subsequent tests except the tail suspension test. FC resulted in more freezing behavior in all mice that received foot shocks when evaluated in subsequent context and cue tests and induced hypoactivity in the open field (OF) and elevated zero maze (EZM). Mice that sustained frCBI learned the FC association between tone and shock. Injured mice froze less than sham controls during context and cue tests, which could indicate memory impairment, but could also suggest that frCBI resulted in hyperactivity that overrode the rodent's natural freezing response to threat, as injured mice were also more active in the OF and EZM. There were notable sex differences, where female mice exhibited more freezing behavior than male mice during FC context and cue tests. The findings suggest frCBI impaired, but did not eliminate, FC retention and resulted in an overall increase in general activity. The injury was characterized pathologically by increased inflammation (CD11b staining) in cortical regions underlying the injury site and in the optic tracts. The performance of male and female mice after injury suggested the complexity of possible sex differences for neuropsychiatric symptoms.

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TI An update on the rod microglia variant in experimental and clinical

brain injury and disease

SO BRAIN COMMUNICATIONS

LA English

DT Review; Early Access

DE rod microglia; traumatic brain injury; microglia; pathology;

inflammation

ID BIPOLAR/ROD-SHAPED MICROGLIA; ACTIVATION; MORPHOLOGY; CORTEX; FATE;

MORBIDITIES; EPILEPSY; REVEALS; CELLS

AB Contemporary microglia morphologies include ramified, activated and amoeboid, with the morphology of microglia considered highly coupled to the cellular function. Rod microglia are an additional activated microglia variant observed in the ageing, injured and diseased brain. Rod microglia were reported frequently in the early 1900s by neuropathologists in post-mortem cases of general paresis, Alzheimer's disease and encephalitis, and then remained largely ignored for almost 100 years. Recent reports have renewed interest in rod microglia, most notably after experimental traumatic brain injury. Rod microglia are formed by the narrowing of the soma and retraction of planar processes, which results in the appearance of an elongated, rod-shaped cell. Rod microglia are most commonly observed in the cortex, aligned perpendicular to the dural surface and adjacent to neuronal processes; in the hippocampus, they are aligned perpendicular to hippocampal layers. Furthermore, rod microglia form trains with one another, apical end to basal end. By replicating the process of sketching microscopic observation, rod microglia are re-defined by circumnutation around the long axis. In this update, we summarize the rod microglia variant in clinical and experimental literature and advocate for investigation into mechanisms of rod microglia origin and function.

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TI Mer regulates microglial/macrophage M1/M2 polarization and alleviates

neuroinflammation following traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Mer; Microglia; macrophage; M1; M2 polarization; Neuroinflammation; TBI

ID RECEPTOR TYROSINE KINASE; WHITE-MATTER; MICROGLIA/MACROPHAGE

POLARIZATION; SEX-DIFFERENCES; MYELOID CELLS; TAM RECEPTOR;

INFLAMMATION; MOUSE; MACROPHAGES; RESOLUTION

AB Background Traumatic brain injury (TBI) is a leading cause of death and disability worldwide. Microglial/macrophage activation and neuroinflammation are key cellular events following TBI, but the regulatory and functional mechanisms are still not well understood. Myeloid-epithelial-reproductive tyrosine kinase (Mer), a member of the Tyro-Axl-Mer (TAM) family of receptor tyrosine kinases, regulates multiple features of microglial/macrophage physiology. However, its function in regulating the innate immune response and microglial/macrophage M1/M2 polarization in TBI has not been addressed. The present study aimed to evaluate the role of Mer in regulating microglial/macrophage M1/M2 polarization and neuroinflammation following TBI. Methods The controlled cortical impact (CCI) mouse model was employed. Mer siRNA was intracerebroventricularly administered, and recombinant protein S (PS) was intravenously applied for intervention. The neurobehavioral assessments, RT-PCR, Western blot, magnetic-activated cell sorting, immunohistochemistry and confocal microscopy analysis, Nissl and Fluoro-Jade B staining, brain water content measurement, and contusion volume assessment were performed. Results Mer is upregulated and regulates microglial/macrophage M1/M2 polarization and neuroinflammation in the acute stage of TBI. Mechanistically, Mer activates the signal transducer and activator of transcription 1 (STAT1)/suppressor of cytokine signaling 1/3 (SOCS1/3) pathway. Inhibition of Mer markedly decreases microglial/macrophage M2-like polarization while increases M1-like polarization, which exacerbates the secondary brain damage and sensorimotor deficits after TBI. Recombinant PS exerts beneficial effects in TBI mice through Mer activation. Conclusions Mer is an important regulator of microglial/macrophage M1/M2 polarization and neuroinflammation, and may be considered as a potential target for therapeutic intervention in TBI.

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TI Applying a novel 3D hydrogel cell culture to investigate activation of

microglia due to rotational kinematics associated with mild traumatic

brain injury

SO JOURNAL OF THE MECHANICAL BEHAVIOR OF BIOMEDICAL MATERIALS

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DE mTBI; Glia; Hydrogel; 3D cell culture; Rotational kinematics

ID LIPOPOLYSACCHARIDE-INDUCED NEUROTOXICITY; DIFFUSE AXONAL INJURY;

NITRIC-OXIDE; POSSIBLE INVOLVEMENT; COGNITIVE DEFICITS; MURINE MODEL;

HEAD IMPACT; MOUSE MODEL; RAT MODEL; NEURONS

AB Many investigations on mild traumatic brain injury (mTBI) aim to further understand how cells in the brain react to the mechanical forces associated with the injury. While it is known that rapid head rotation is a mechanism contributing to mTBI, establishing definitive thresholds for head rotation has proved challenging. One way to advance determining mechanisms and thresholds for injury is through in vitro models. Here, an apparatus has been designed that is capable of delivering rotational forces to three-dimensional (3D) hydrogel cell cultures. Using an in vitro model, we test the hypothesis that rotational kinematics can activate microglia suspended in a 3 dimensional mixed glia environment (absent neurons). The impact apparatus was able to deliver peak angular velocities of approximately 45 rad/s, a magnitude for angular velocity that in select literature is associated with diffuse brain injury. However, no measurable glial cell reactivity was observed in response to the rotational kinematics through any of the chosen metrics (nitric oxide, pro-inflammatory cytokine release and proportion of amoeboid activated microglia). The results generated from this study suggest that rotation of the glia alone did not cause activation in future work we will investigate the effect of neuronal contributions in activating glia.

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TI Microglia depletion and cognitive functions after brain injury: From

trauma to galactic cosmic ray

SO NEUROSCIENCE LETTERS

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DT Article

DE Microglia; Traumatic brain injury; Whole-brain radiotherapy; Galactic

cosmic ray; Microglia depletion; Microglia repopulation

ID CENTRAL-NERVOUS-SYSTEM; HIPPOCAMPAL NEUROGENESIS; PROGENITOR-CELL;

RADIATION; RECEPTOR; IRRADIATION; DEFICIENCY; MECHANISMS; EXPRESSION;

HEALTH

AB Microglia are the resident immune cells of the central nervous system (CNS). In physiological conditions, microglia contribute to maintaining brain homeostasis by scanning the surrounding parenchyma and acting as scavenger cells. Following different insults to the CNS, microglia turn into a "reactive" state characterized by the production of inflammatory mediators that promote tissue repair to restore homeostasis. Brain insults such as traumatic brain injury, therapeutic brain irradiation and galactic cosmic ray exposure are associated with chronic microglia activation. Chronic microglia activation contributes to injury-related impairments in cognitive functions. Microglia depletion achieved either by pharmacological or genetic techniques represents not only a useful tool for more extensive investigations of microglia roles, but also a potential therapeutic approach to ameliorate or prevent cognitive dysfunctions following brain injury.

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TI Role of innate inflammation in traumatic brain injury

SO NEUROLOGICAL SCIENCES

LA English

DT Review; Early Access

DE Traumatic brain injury; Neuroinflammation; Microglia; Blood-brain

barrier; Cytokine; Innate inflammation

ID INTERLEUKIN-1 RECEPTOR ANTAGONIST; DOUBLE-BLIND; MICROGLIAL ACTIVATION;

MODERATE HYPOTHERMIA; FUNCTIONAL RECOVERY; HEAD-INJURY; CELL-DEATH;

PHASE-II; IN-VITRO; DAMAGE

AB Traumatic brain injury is one of the leading causes of morbidity and mortality throughout the world. Its increasing incidence, in addition to its fundamental role in the development of neurodegenerative disease, proves especially concerning. Despite extensive preclinical and clinical studies, researchers have yet to identify a safe and effective neuroprotective strategy. Following brain trauma, secondary injury from molecular, metabolic, and cellular changes causes progressive cerebral tissue damage. Chronic neuroinflammation following traumatic brain injuries is a key player in the development of secondary injury. Targeting this phenomenon for development of effective neuroprotective therapies holds promise. This strategy warrants a concrete understanding of complex neuroinflammatory mechanisms. In this review, we discuss pathophysiological mechanisms such as the innate immune response, glial activation, blood-brain barrier disruption, activation of immune mediators, as well as biological markers of traumatic brain injury. We then review existing and emerging pharmacological therapies that target neuroinflammation to improve functional outcome.

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TI Decreased Level of Exosomal miR-5121 Released from Microglia Suppresses

Neurite Outgrowth and Synapse Recovery of Neurons Following Traumatic

Brain Injury

SO NEUROTHERAPEUTICS

LA English

DT Article; Early Access

DE Exosomes; microRNA; microglia; neuron; traumatic brain injury

ID REPULSIVE GUIDANCE MOLECULE; STRETCH-INDUCED INJURY; EXTRACELLULAR

VESICLES; CORTICOSPINAL AXONS; P2X7 RECEPTOR; EXPRESSION; CELLS; MODEL;

RGMA; INFLAMMATION

AB Activated microglia can suppress neurite outgrowth and synapse recovery in the acute stage following traumatic brain injury (TBI). However, the underlying mechanism has not been clearly elucidated. Exosomes derived from microglia have been reported to play a critical role in microglia-neuron interaction in healthy and pathological brains. Here, we aimed to investigate the role of microglia-derived exosomes in regulating neurite outgrowth and synapse recovery following TBI. In our study, exosomes derived from microglia were co-cultured with stretch-injured neurons in vitro and intravenously injected into mice that underwent fluid percussion injury (FPI) by tail vein injection in vivo. The results showed that microglia-derived exosomes could be absorbed by neurons in vitro and in vivo. Moreover, exosomes derived from stretch-injured microglia decreased the protein levels of GAP43, PSD-95, GluR1, and Synaptophysin and dendritic complexity in stretch-injured neurons in vitro, and reduced GAP43+ NEUN cell percentage and apical dendritic spine density in the pericontusion region in vivo. Motor coordination was also impaired in mice treated with stretch-injured microglia-derived exosomes after FPI. A microRNA microarray showed that the level of miR-5121 was decreased most greatly in exosomes derived from stretch-injured microglia. Overexpression of miR-5121 in stretch-injured microglia-derived exosomes partly reversed the suppression of neurite outgrowth and synapse recovery of neurons both in vitro and in vivo. Moreover, motor coordination in miR-5121 overexpressed exosomes treated mice was significantly improved after FPI. Following mechanistic study demonstrated that miR-5121 might promote neurite outgrowth and synapse recovery by directly targeting RGMa. In conclusion, our finding revealed a novel exosome-mediated mechanism of microglia-neuron interaction that suppressed neurite outgrowth and synapse recovery of neurons following TBI.

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TI Transcriptional profiling of microglia in the injured brain reveals

distinct molecular features underlying neurodegeneration

SO GLIA

LA English

DT Article; Early Access

DE microglial; neurodegeneration; sex differences; transcriptome; traumatic

brain injury

ID SEX-DIFFERENCES; EXPRESSION; ACTIVATION; IDENTIFICATION; NEUROTOXICITY;

INFLAMMATION; CLEARANCE; DISEASE; MODEL; LUNG

AB Neurotrauma has been recognized as a risk factor for neurodegenerative diseases, and sex difference of the incidence and outcome of neurodegenerative diseases has long been recognized. Past studies suggest that microglia could play a versatile role in both health and disease. So far, the microglial mechanisms underlying neurodegeneration and potentially lead to sex-specific therapies are still very open. Here we applied whole transcriptome analysis of microglia acutely isolated at different timepoints after a cortical stab wound injury to gain insight into genes that might be dysregulated and transcriptionally different between males and females after cortical injury. We found that microglia displayed distinct temporal and sexual molecular signatures of transcriptome after cortical injury. Hypotheses and gene candidates that we presented in the present study could be worthy to be examined to explore the roles of microglia in neurotrauma and in sex-biased neurodegenerative diseases.

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TI Microglial Metabolism After Pediatric Traumatic Brain Injury -

Overlooked Bystanders or Active Participants?

SO FRONTIERS IN NEUROLOGY

LA English

DT Review

DE microglia; metabolism; pediatric; brain trauma; energy

ID IMPROVES FUNCTIONAL RECOVERY; GLUTAMATE-RECEPTOR 5; PENTOSE-PHOSPHATE

PATHWAY; CORTICAL IMPACT INJURY; FATTY-ACIDS; ATTENUATES

NEURODEGENERATION; LYSOPHOSPHATIDIC ACID; OXIDATIVE STRESS; QUINOLINIC

ACID; NADPH OXIDASE

AB Microglia play an integral role in brain development but are also crucial for repair and recovery after traumatic brain injury (TBI). TBI induces an intense innate immune response in the immature, developing brain that is associated with acute and chronic changes in microglial function. These changes contribute to long-lasting consequences on development, neurologic function, and behavior. Although alterations in glucose metabolism are well-described after TBI, the bulk of the data is focused on metabolic alterations in astrocytes and neurons. To date, the interplay between alterations in intracellular metabolic pathways in microglia and the innate immune response in the brain following an injury is not well-studied. In this review, we broadly discuss the microglial responses after TBI. In addition, we highlight reported metabolic alterations in microglia and macrophages, and provide perspective on how changes in glucose, fatty acid, and amino acid metabolism can influence and modulate the microglial phenotype and response to injury.

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TI A receptor-binding radiopharmaceutical for imaging of traumatic brain

injury in a rodent model: [<SUP>99m</SUP>Tc]Tc-tilmanocept

SO NUCLEAR MEDICINE AND BIOLOGY

LA English

DT Article; Early Access

DE Traumatic brain injury; Blood brain barrier; Tilmanocept; cd206;

Microglial cells; Fluorescence imaging

ID MANNOSE RECEPTOR; BARRIER PERMEABILITY; ENDOTHELIAL-CELLS; BLOOD;

DYSFUNCTION; TILMANOCEPT; DISRUPTION; ACTIVATION; CLEARANCE; BREAKDOWN

AB Introduction: Blood-brain barrier (BBB) disruption and subsequent neuro-inflammation occur following traumatic brain injury (TBI), resulting in a spectrum of human nervous system disorders. [Tc-99m]Tc-tilmanocept is a receptor-binding radiopharmaceutical FDA-approved for sentinel lymph node mapping. We hypothesize that after an intravenous (i.v.) injection, [Tc-99m]Tc-tilmanocept, will traverse a disrupted BBB and bind to CD206-bea ring microglial cells.

Methods: Age-matched mice were divided into three groups: 5-days post TBI (n = 4), and 5-days post sham (n = 4), and naive controls (n = 4). IRDye800CW-labeled [Tc-99m]Tc-tilmanocept (0.15 nmol per gram body weight) and FITC-labeled bovine scrum albumin (FITC-B5A) were injected (i.v.) into each mouse. Mice were imaged with a high-resolution gamma camera for 45 min. Immediately after imaging, the brains were perfused with fixative, excised, imaged with a fluorescence scanner, assayed for radioactivity, and prepared for histology.

Results: In vivo nuclear imaging, ex vivo fluorescence imaging, ex vivo gamma well counting, and histomicroscopy demonstrated enhanced tilmanocept uptake in the TBI region. The normalized [Tc-99m]Tc-tilmanocept uptake value from nuclear imaging and the maximum pixel intensity from fluorescence imaging of the TBI group ( 1.12 +/- 0.12 and 2288 +/- 278 a.u., respectively) were significantly (P < 0.04) higher than the sham group (0.64 0.28 and 1708 +/- 101 am., respectively) and the naive group (0.76 +/- 024 and 1643 +/- 391 a.u., respectively). The mean [Tc-99m]Tc-tilmanocept scaled uptake in the TBI brains (0.058 +/- 0.013%/g) was significantly (P < 0.010) higher than the scaled brain uptake of the sham group (0.031 +/- 0.011%/g) and higher (P 0.04) than the uptake of the naive group (0.020 +/- 0.002%/g). Fluorescence microscopy demonstrated increased uptake of the IRDye800CW-tilmanocept and ITTC-BSA in the TBI brain regions.

Conclusion: [Tc-99m]Tc-tilmanocept traverses disrupted blood-brain barrier and localizes within the injured region.

Advances in knowledge and implications for patient care: [Tc-99m]Tc-tilmanocept could serve as an imaging biomarker for TBI-associated neuroinflammation and any disease process that involves a disruption of the blood-brain barrier. (C) 2020 Elsevier Inc. All rights reserved.

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TI The Immune System's Role in the Consequences of Mild Traumatic Brain

Injury (Concussion)

SO FRONTIERS IN IMMUNOLOGY

LA English

DT Review

DE concussion; neuroimmunology; microglia; neurodegenenerative diseases;

inflammation; mild TBI

ID CLOSED-HEAD INJURY; FLUID SOLUBLE TREM2; SPINAL-CORD; MOUSE MODEL;

TRANSLOCATOR PROTEIN; ALZHEIMERS-DISEASE; 18 KDA; NEUROINFLAMMATORY

RESPONSE; SUBARACHNOID HEMORRHAGE; MACROPHAGE POLARIZATION

AB Mild traumatic brain injury (mild TBI), often referred to as concussion, is the most common form of TBI and affects millions of people each year. A history of mild TBI increases the risk of developing emotional and neurocognitive disorders later in life that can impact on day to day living. These include anxiety and depression, as well as neurodegenerative conditions such as chronic traumatic encephalopathy (CTE) and Alzheimer's disease (AD). Actions of brain resident or peripherally recruited immune cells are proposed to be key regulators across these diseases and mood disorders. Here, we will assess the impact of mild TBI on brain and patient health, and evaluate the recent evidence for immune cell involvement in its pathogenesis.

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TI Traumatic Brain Injury Causes Chronic Cortical Inflammation and Neuronal

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DT Article

DE CSF1R antagonist; microglia; neuroinflammation; neurotrauma; traumatic

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ID AXONAL INJURY; COGNITIVE IMPAIRMENT; CELL; ACTIVATION; DEPRESSION;

DRIVES; NEUROINFLAMMATION; NEURODEGENERATION; MORPHOLOGY; RECOVERY

AB Traumatic brain injury (TBI) can lead to significant neuropsychiatric problems and neurodegenerative pathologies, which develop and persist years after injury. Neuroinflammatory processes evolve over this same period. Therefore, we aimed to determine the contribution of microglia to neuropathology at acute [1 d postinjury (dpi)], subacute (7 dpi), and chronic (30 dpi) time points. Microglia were depleted with PLX5622, a CSF1R antagonist, before midline fluid percussion injury (FPI) in male mice and cortical neuropathology/inflammation was assessed using a neuropathology mRNA panel. Gene expression associated with inflammation and neuropathology were robustly increased acutely after injury (1 dpi) and the majority of this expression was microglia independent. At 7 and 30 dpi, however, microglial depletion reversed TBI-related expression of genes associated with inflammation, interferon signaling, and neuropathology. Myriad suppressed genes at subacute and chronic endpoints were attributed to neurons. To understand the relationship between microglia, neurons, and other glia, single-cell RNA sequencing was completed 7 dpi, a critical time point in the evolution from acute to chronic pathogenesis. Cortical microglia exhibited distinct TBI-associated clustering with increased type-1 interferon and neurodegenerative/damage-related genes. In cortical neurons, genes associated with dopamine signaling, long-term potentiation, calcium signaling, and synaptogenesis were suppressed. Microglial depletion reversed the majority of these neuronal alterations. Furthermore, there was reduced cortical dendritic complexity 7 dpi, reduced neuronal connectively 30 dpi, and cognitive impairment 30 dpi. All of these TBI-associated functional and behavioral impairments were prevented by microglial depletion. Collectively, these studies indicate that microglia promote persistent neuropathology and long-term functional impairments in neuronal homeostasis after TBI.

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TI Diffusion Tensor Imaging Detects Acute Pathology-Specific Changes in the

P301L Tauopathy Mouse Model Following Traumatic Brain Injury

SO FRONTIERS IN NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury; diffusion tensor imaging; transgenic;

tau-hyperphosphorylation; astrocytes; microglia; fractional anisotropy;

P301L-mutation

ID AMYLOID PRECURSOR PROTEIN; CORTICAL IMPACT INJURY; ALZHEIMERS-DISEASE;

VOXELWISE ANALYSIS; TAU PATHOLOGY; ACTIVATION; RAT; NEUROINFLAMMATION;

HIPPOCAMPUS; ASTROCYTES

AB Traumatic brain injury (TBI) has been linked with tauopathy. However, imaging methods that can non-invasively detect tau-protein abnormalities following TBI need further investigation. This study aimed to investigate the potential of diffusion tensor imaging (DTI) to detect tauopathy following TBI in P301L mutant-tau-transgenic-pR5-mice. A total of 24 9-month-old pR5 mice were randomly assigned to sham and TBI groups. Controlled cortical injuries/craniotomies were performed for TBI/sham groups followed by DTI data acquisition on days 1 and 7 post-injury. DTI data were analyzed by using voxelwise analysis and track-based spatial statistics for gray matter and white matter. Further, immunohistochemistry was performed for total-tau and phosphorylated-tau, astrocytes, and microglia. To detect the association of DTI with these pathological markers, a correlation analysis was performed between DTI and histology findings. At day 1 post-TBI, DTI revealed a widespread reduction in fractional anisotropy (FA) and axial diffusivity (AxD) in the TBI group compared to shams. On day 7, further reduction in FA, AxD, and mean diffusivity and increased radial diffusivity were observed. FA was significantly increased in the amygdala and cortex. Correlation results showed that in the ipsilateral hemisphere FA reduction was associated with increased phosphorylated-tau and glial-immunoreactivity, whereas in the contralateral regions, the FA increase was associated with increased immunostaining for astrocytes. This study is the first to exploit DTI to investigate the effect of TBI in tau-transgenic mice. We show that alterations in the DTI signal were associated with glial activity following TBI and would most likely reflect changes that co-occur with/without phosphorylated-tau. In addition, FA may be a promising measure to identify discrete pathological processes such as increased astroglia activation, tau-hyperphosphorylation or both in the brain following TBI.

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TI Gut microbial dysbiosis after traumatic brain injury modulates the

immune response and impairs neurogenesis

SO ACTA NEUROPATHOLOGICA COMMUNICATIONS

LA English

DT Article

DE Traumatic brain injury; Gut microbial dysbiosis; Antibiotics; Fear

conditioning; Microglia; Monocytes; T cells; Neurogenesis

ID CENTRAL-NERVOUS-SYSTEM; PROTECTIVE AUTOIMMUNITY; COGNITIVE DYSFUNCTION;

CA3 OUTPUT; MICROGLIA; RECEPTORS; CELLS; MODEL; NEURODEGENERATION;

HEMATOPOIESIS

AB The influence of the gut microbiota on traumatic brain injury (TBI) is presently unknown. This knowledge gap is of paramount clinical significance as TBI patients are highly susceptible to alterations in the gut microbiota by antibiotic exposure. Antibiotic-induced gut microbial dysbiosis established prior to TBI significantly worsened neuronal loss and reduced microglia activation in the injured hippocampus with concomitant changes in fear memory response. Importantly, antibiotic exposure for 1 week after TBI reduced cortical infiltration of Ly6C(high) monocytes, increased microglial pro-inflammatory markers, and decreased T lymphocyte infiltration, which persisted through 1 month post-injury. Moreover, microbial dysbiosis was associated with reduced neurogenesis in the dentate gyrus 1 week after TBI. By 3 months after injury (11 weeks after discontinuation of the antibiotics), we observed increased microglial proliferation, increased hippocampal neuronal loss, and modulation of fear memory response. These data demonstrate that antibiotic-induced gut microbial dysbiosis after TBI impacts neuroinflammation, neurogenesis, and fear memory and implicate gut microbial modulation as a potential therapeutic intervention for TBI.

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TI Downregulation of lncRNA KCNQ1OT1 relieves traumatic brain injury

induced neurological deficits via promoting "M2" microglia polarization

SO BRAIN RESEARCH BULLETIN

LA English

DT Article; Early Access

DE Traumatic brain injury; Microglia; KCNQ1OT1; ceRNA; miR-873-5p

ID LONG NONCODING RNA; EXPRESSION; PROGNOSIS

AB Background: Microglia-induced neuroinflammation is one of the main characteristics of traumatic brain injury (TBI). Presently, we aim to investigate the role of long non-coding RNA (lncRNA) KCNQ1 overlapping transcript 1 (KCNQ1OT1) in TBI-induced neurological deficits and the related mechanism.

Methods: An in-vivo TBI model was established in mice, and in-vitro experiments were carried out on BV2 microglia. Then the neurological functions, microglial activation, inflammatory cytokines, and proteins were detected.

Results: Our data indicated that KCNQ1OT1 was markedly overexpressed in the cerebral tissues of TBI mice, accompanied by a higher level of the cytokines (including IL-1 beta, IL-6, and TNF alpha). However, knocking down KCNQ1OT1 relieved neurological deficits, neuron loss, and blood-brain barrier damage. Besides, overexpressing miR-873-5p enhanced the "M2'' polarization of microglia by repressing the TRAF6-mediated p38 and NF-kappa B pathways. In contrast, downregulating KCNQ1OT1 repressed microglial neuroinflammation by attenuating the "M1'' polarization of microglia and promoting "M2'' polarization of microglia, and inactivating the p38 and NF-kappa B pathway.

Conclusions: Mechanistically, KCNQ1OT1 functioned as a competitive endogenous RNA (ceRNA) by sponging miR-873-5p, which targeted the 3' untranslated region (UTR) of TRAF6. Overall, our data confirmed that downregulating lncRNA KCNQ1OT1 exerted neuroprotective effects on TBI mice by modulating the miR-873-5p-TRAF6-p38/NF-kappa B axis.

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TI Enriching neural stem cell and anti-inflammatory glial phenotypes with

electrical stimulation after traumatic brain injury in male rats

SO JOURNAL OF NEUROSCIENCE RESEARCH

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DT Article; Early Access

DE astrocyte; electrical stimulation; microglia; neural stem cell; RRID;

AB\_10013382; RRID; AB\_10893200; RRID; AB\_1523910; RRID; AB\_2282664;

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RRID; SCR\_008520; traumatic brain injury

AB Traumatic brain injury (TBI) by an external physical impact results in compromised brain function via undesired neuronal death. Following the injury, resident and peripheral immune cells, astrocytes, and neural stem cells (NSCs) cooperatively contribute to the recovery of the neuronal function after TBI. However, excessive pro-inflammatory responses of immune cells, and the disappearance of endogenous NSCs at the injury site during the acute phase of TBI, can exacerbate TBI progression leading to incomplete healing. Therefore, positive outcomes may depend on early interventions to control the injury-associated cellular milieu in the early phase of injury. Here, we explore electrical stimulation (ES) of the injury site in a rodent model (male Sprague-Dawley rats) to investigate its overall effect on the constituent brain cell phenotype and composition during the acute phase of TBI. Our data showed that a brief ES for 1 hr on day 2 of TBI promoted anti-inflammatory phenotypes of microglia as assessed by CD206 expression and increased the population of NSCs and Nestin(+) astrocytes at 7 days post-TBI. Also, ES effectively increased the number of viable neurons when compared to the unstimulated control group. Given the salience of microglia and neural stem cells for healing after TBI, our results strongly support the potential benefit of the therapeutic use of ES during the acute phase of TBI to regulate neuroinflammation and to enhance neuroregeneration.

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U2 10

PU WILEY

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TI The second phase of brain trauma can be controlled by nutraceuticals

that suppress DAMP-mediated microglial activation

SO EXPERT REVIEW OF NEUROTHERAPEUTICS

LA English

DT Review; Early Access

DE Brain trauma; neuronal death; damp-mediated microglial activation;

peroxynitrite; nutraceuticals

ID NF-KAPPA-B; TEA POLYPHENOL EPIGALLOCATECHIN-3-GALLATE; NADPH OXIDASE;

SIGNALING PATHWAY; HEME OXYGENASE-1; PROTEIN-KINASE; FERULIC ACID;

INFLAMMATORY RESPONSE; SPIRULINA-PLATENSIS; FUNCTIONAL RECOVERY

AB Introduction

A delayed second wave of brain trauma is mediated in large part by microglia that are activated to a pro-inflammatory M1 phenotype by DAMP proteins released by dying neurons. These microglia can promote apoptosis or necrosis in neighboring neurons by producing a range of pro-inflammatory cytokines and the deadly oxidant peroxynitrite. This second wave could therefore be mitigated with agents that blunt the post-traumatic M1 activation of microglia and that preferentially promote a pro-healing M2 phenotype.

Areas covered

The literature on nutraceuticals that might have clinical potential in this regard.

Expert opinion

The chief signaling pathway whereby DAMPs promote M1 microglial activation involves activation of toll-like receptor 4 (TLR4), NADPH oxidase, NF-kappaB, and the stress activated kinases JNK and p38. The green tea catechin EGCG can suppress TLR4 expression. Phycocyanobilin can inhibit NOX2-dependent NADPH oxidase, ferulate and melatonin can oppose pro-inflammatory signal modulation by NADPH oxidase-derived oxidants. Long-chain omega-3 fatty acids, the soy isoflavone genistein, the AMPK activator berberine, glucosamine, and ketone bodies can down-regulate NF-kappaB activation. Vitamin D activity can oppose JNK/p38 activation. A sophisticated program of nutraceutical supplementation may have important potential for mitigating the second phase of neuronal death and aiding subsequent healing.

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TI Functions and Mechanisms of the Voltage-Gated Proton Channel Hv1 in

Brain and Spinal Cord Injury

SO FRONTIERS IN CELLULAR NEUROSCIENCE

LA English

DT Review

DE voltage-gated proton channel Hv1; NOX2; acidification; ROS; microglia;

traumatic brain injury; spinal cord injury; stroke

AB The voltage-gated proton channel Hv1 is a newly discovered ion channel that is highly conserved among species. It is known that Hv1 is not only expressed in peripheral immune cells but also one of the major ion channels expressed in tissue-resident microglia of the central nervous systems (CNS). One key role for Hv1 is its interaction with NADPH oxidase 2 (NOX2) to regulate reactive oxygen species (ROS) and cytosolic pH. Emerging data suggest that excessive ROS production increases and requires proton currents through Hv1 in the injured CNS, and manipulations that ablate Hv1 expression or induce loss of function may provide neuroprotection in CNS injury models including stroke, traumatic brain injury, and spinal cord injury. Recent data demonstrating microglial Hv1-mediated signaling in the pathophysiology of the CNS injury further supports the idea that Hv1 channel may function as a key mechanism in posttraumatic neuroinflammation and neurodegeneration. In this review, we summarize the main findings of Hv1, including its expression pattern, cellular mechanism, role in aging, and animal models of CNS injury and disease pathology. We also discuss the potential of Hv1 as a therapeutic target for CNS injury.

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TI Buprenorphine alters microglia and astrocytes acutely following diffuse

traumatic brain injury

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DT Article

ID SUSTAINED-RELEASE BUPRENORPHINE; INTRACRANIAL-PRESSURE; OPIOID RECEPTOR;

INCISIONAL PAIN; AXONAL INJURY; ACTIVATION; MODEL; RATS;

PHARMACOKINETICS; ANALGESIA

AB Traumatic brain injury (TBI) is a common phenomenon, accounting for significant cost and adverse health effects. While there is information about focal pathologies following TBI, knowledge of more diffuse processes is lacking, particularly regarding how analgesics affect this pathology. As buprenorphine is the most commonly used analgesic in experimental TBI models, this study investigated the acute effects of the opioid analgesic buprenorphine (Bup-SR-Lab) on diffuse neuronal/glial pathology, neuroinflammation, cell damage, and systemic physiology. We utilized a model of central fluid percussion injury (CFPI) in adult male rats treated with a single subcutaneous bolus of Bup-SR-Lab or saline 15 min post-injury. Microscopic assessments were performed at 1 day post-injury. Cell impermeable dextran was infused intraventricularly prior to sacrifice to assess neuronal membrane disruption. Axonal injury was assessed by investigating labeling of the anterogradely transported amyloid precursor protein. Neuroinflammation was assessed by analyzing Iba-1+microglial and GFAP+astrocyte histological/morphological features as well as cytokine levels in both regions of interest (ROIs). Myelin pathology was assessed by evaluating the expression of myelin basic protein (MBP) and the propensity of MBP+myelin debris. Acute physiologic data showed no difference between groups except for reduction in weight loss following cFPI in Bup treated animals compared to saline. There were no discernable differences in axonal injury or membrane disruption between treatment groups. Cytokine levels were consistent between Bup and saline treated animals, however, microglia and astrocytes revealed region specific histological changes at 1d following Bup treatment. Myelin integrity and overall MBP expression showed no differences between Bup and saline treated animals, but there were significant regional differences in MBP expression between the cortex and thalamus. These data suggest effects of Bup treatment on weight following CFPI and potential regional specificity of Bup-associated microglial and astrocyte alterations, but very little change in other acute pathology at 1-day post-injury. Overall, this preliminary study indicates that use of Bup-SR-Lab in preclinical work does have effects on acute glial pathology, however, longer term studies will be needed to assess potential effects of Bup treatment on more chronic pathological progressions.

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TI Design and Evaluation of an In Vitro Mild Traumatic Brain Injury

Modeling System Using 3D Printed Mini Impact Device on the 3D Cultured

Human iPSC Derived Neural Progenitor Cells

SO ADVANCED HEALTHCARE MATERIALS

LA English

DT Article; Early Access

DE 3D printing; iPSC derived neural progenitor cells; microglia;

neuroinflammation; repetitive mild traumatic brain injury

ID LONG-TERM CONSEQUENCES; REACTIVE ASTROCYTES; MICROGLIA ACTIVATION;

COGNITIVE IMPAIRMENT; POTENTIAL MECHANISMS; CEREBRAL ORGANOIDS;

ANIMAL-MODELS; MOUSE MODEL; CONCUSSION; INFLAMMATION

AB Despite significant progress in understanding the disease mechanism of traumatic brain injury (TBI), promising preclinical therapeutics have seldom been translated into successful clinical outcomes, partially because the model animals have physiological and functional differences in the central nervous system (CNS) compared to humans. Human relevant models are thus urgently required. Here, an in vitro mild TBI (mTBI) modeling system is reported based on 3D cultured human induced pluripotent stem cells (iPSC) derived neural progenitor cells (iPSC-NPCs) to evaluate consequences of single and repetitive mTBI using a 3D printed mini weight-drop impact device. Computational simulation is performed to understand the single/cumulative effects of weight-drop impact on the NPC differentiated neurospheres. Experimental results reveal that neurospheres show reactive astrogliosis and glial scar formation after repetitive (10 hits) mild impacts, while no astrocyte activation is found after one or two mild impacts. A 3D co-culture model of human microglia cells with neurospheres is further developed. It is found that astrocyte response is promoted even after two mild impacts, possibly caused by the chronic neuroinflammation after microglia activation. The in vitro mTBI modeling system recapitulates several hallmarks of the brain impact injury and might serve as a good platform for future drug screening.

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TI PET Imaging of Peripheral Benzodiazepine Receptor Standard Uptake Value

Increases After Controlled Cortical Impact, a Rodent Model of Traumatic

Brain Injury

SO ASN NEURO

LA English

DT Article

DE TBI; microglia; inflammation; PET; CT; peripheral benzodiazapine

receptor

ID ACTIVATED MICROGLIAL/MACROPHAGE RESPONSE; TRANSLOCATOR PROTEIN TSPO;

MICROGLIAL ACTIVATION; INFLAMMATION; BINDING; NEUROINFLAMMATION;

RECRUITMENT; ASTROCYTES; EXPRESSION; AFFINITY

AB Traumatic brain injury (TBI) is a chronic, life threatening injury for which few effective interventions are available. Evidence in animal models suggests un-checked immune activation may contribute to the pathophysiology. Changes in regional density of active brain microglia can be quantified in vivo with positron emission topography (PET) with the relatively selective radiotracer, peripheral benzodiazepine receptor 28 (11 C-PBR28). Phenotypic assessment (activated vs resting) can subsequently be assessed (ex vivo) using morphological techniques. To elucidate the mechanistic contribution of immune cells in due to TBI, we employed a hybrid approach involving both in vivo (11 C-PBR28 PET) and ex vivo (morphology) to elucidate the role of immune cells in a controlled cortical impact (CCI), a rodent model for TBI. Density of activated brain microglia/macrophages was quantified 120 hours after injury using the standardized uptake value (SUV) approach. Ex vivo morphological analysis from specific brain regions using IBA-1 antibodies differentiated ramified (resting) from amoeboid (activated) immune cells. Additional immunostaining of PBRs facilitated co-localization of PBRs with IBA-1 staining to further validate PET data. Injured animals displayed greater PBR28suv when compared to sham animals. Immunohistochemistry demonstrated elevated density of amoeboid microglia/macrophages in the ipsilateral dentate gyrus, corpus callosum, thalami and injury penumbra of injured animals compared to sham animals. PBR co-stained with amoeboid microglia/macrophages in the injury penumbra and not with astrocytes. These data suggest the technologies evaluated may serve as bio-signatures of neuroinflammation following severe brain injury in small animals, potentially enabling in vivo tracking of neuroinflammation following TBI and cellular-based therapies.

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TI Diverse changes in microglia morphology and axonal pathology during the

course of 1 year after mild traumatic brain injury in pigs

SO BRAIN PATHOLOGY

LA English

DT Article; Early Access

DE axonal pathology; concussion; microglia; mild traumatic brain injury;

skeletal analysis

ID INFLAMMATORY RESPONSE; CELLS; NEURODEGENERATION; NEUROPROTECTION;

MACROPHAGES; SYNAPSES; NEURONS; MEMORY; MOUSE

AB Over 2.8 million people experience mild traumatic brain injury (TBI) in the United States each year, which may lead to long-term neurological dysfunction. The mechanical forces that are caused by TBI propagate through the brain to produce diffuse axonal injury (DAI) and trigger secondary neuroinflammatory cascades. The cascades may persist from acute to chronic time points after injury, altering the homeostasis of the brain. However, the relationship between the hallmark axonal pathology of diffuse TBI and potential changes in glial cell activation or morphology have not been established in a clinically relevant large animal model at chronic time points. In this study, we assessed the tissue from pigs subjected to rapid head rotation in the coronal plane to generate mild TBI. Neuropathological assessments for axonal pathology, microglial morphological changes, and astrocyte reactivity were conducted in specimens out to 1-year post-injury. We detected an increase in overall amyloid precursor protein pathology, as well as periventricular white matter and fimbria/fornix pathology after a single mild TBI. We did not detect the changes in corpus callosum integrity or astrocyte reactivity. However, detailed microglial skeletal analysis revealed changes in morphology, most notably increases in the number of microglial branches, junctions, and endpoints. These subtle changes were most evident in periventricular white matter and certain hippocampal subfields, and were observed out to 1-year post-injury in some cases. These ongoing morphological alterations suggest persistent change in neuroimmune homeostasis. Additional studies are needed to characterize the underlying molecular and neurophysiological alterations, as well as potential contributions to neurological deficits.

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TI Protective effect of acute splenic irradiation in rats with traumatic

brain injury

SO NEUROREPORT

LA English

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DE microglia; traumatic brain injury; Tuftsin; X-rays

AB Objective

To explore the protective effect of acute splenic irradiation against traumatic brain injury (TBI) in rats.

Methods

A rat model of TBI was established according to Feeney's method. Splenic irradiation was performed by the reverse intensity-modulated radiation therapy (IMRT) source-axis distance (SAD) irradiation technique. Rat brain tissue samples were collected, the water content of the rat brain tissue was determined and the abundance of microglia was detected by immunofluorescence. Spleens were collected to measure the spleen index. Lung, liver, small intestine and kidney tissues were taken for hematoxylin and eosin staining to observe whether there was radiation-induced pathological damage. Peripheral blood was collected to detect tuftsin and the inflammatory factors IL-6 and IL-10.

Results

Compared with the nonirradiated TBI rat group, the 4-h spleen irradiation TBI rat group showed (1) increased behavioral scores at 3 days after TBI (P < 0.05), (2) reduced water content of the ipsilateral hemisphere at 3 days after TBI, (3) reduced spleen index at 3 and 7 days after TBI, (4) reduced number of microglia cells infiltrating around the lesion at 7 days after TBI, (5) reduced IL-6 levels at 3 days after TBI, (6) increased IL-10 levels at 3 and 5days after TBI and (7) Compared with the nonirradiated TBI rat group, the 8-h spleen irradiation TBI rat group showed reduced tuftsin levels at 3 and 7days after TBI.

Conclusions

Acute splenic irradiation had a protective effect in rats with TBI.

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TI TLR4 pathway impairs synaptic number and cerebrovascular functions

through astrocyte activation following traumatic brain injury

SO BRITISH JOURNAL OF PHARMACOLOGY

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DT Article; Early Access

DE astrocytes; BBB breakdown; microglia; infiltrated monocytes; synaptic

remodelling; TLR4 antagonism; traumatic brain injury

ID MICROGLIA; TRANSCRIPTOME; PLASTICITY; PHENOTYPE; INTEGRITY; RECOVERY;

DATABASE; NEURONS; CORTEX; DAMAGE

AB Background and Purpose Activation of astrocytes contributes to synaptic remodelling, tissue repair and neuronal survival following traumatic brain injury (TBI). The mechanisms by which these cells interact to resident/infiltrated inflammatory cells to rewire neuronal networks and repair brain functions remain poorly understood. Here, we explored how TLR4-induced astrocyte activation modified synapses and cerebrovascular integrity following TBI.

Experimental Approach To determine how functional astrocyte alterations induced by activation of TLR4 pathway in inflammatory cells regulate synapses and neurovascular integrity after TBI, we used pharmacology, genetic approaches, live calcium imaging, immunofluorescence, flow cytometry, blood-brain barrier (BBB) integrity assessment and molecular and behavioural methods.

Key Results Shortly after a TBI, there is a recruitment of excitable and reactive astrocytes mediated by TLR4 pathway activation with detrimental effects on post-synaptic density-95 (PSD-95)/vesicular glutamate transporter 1 (VGLUT1) synaptic puncta, BBB integrity and neurological outcome. Pharmacological blockage of the TLR4 pathway with resatorvid (TAK-242) partially reversed many of the observed effects. Synapses and BBB recovery after resatorvid administration were not observed in IP(3)R2(-/-) mice, indicating that effects of TLR4 inhibition depend on the subsequent astrocyte activation. In addition, TBI increased the astrocytic-protein thrombospondin-1 necessary to induce a synaptic recovery in a sub-acute phase.

Conclusions and Implications Our data demonstrate that TLR4-mediated signalling, most probably through microglia and/or infiltrated monocyte-astrocyte communication, plays a crucial role in the TBI pathophysiology and that its inhibition prevents synaptic loss and BBB damage accelerating tissue recovery/repair, which might represent a therapeutic potential in CNS injuries and disorders.

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TI Effect of Hyperbaric Oxygen Therapy on Polarization Phenotype of Rat

Microglia After Traumatic Brain Injury

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DE hyperbaric oxygen; traumatic brain injury; microglia; polarization

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ID IMPAIRMENT; EXPRESSION; ISCHEMIA; SURVIVAL; IMPACT; CELLS

AB Background: The neurological defect caused by secondary damage following traumatic brain injury (TBI) is considered critical for the management of TBI. Microglia (MG) are a resident brain macrophage that could differentiate into M1 type or M2 type in response to injury and repair. It is known that the MG transition from M1 phenotype to anti-inflammatory M2 phenotype might reduce secondary injury of TBI. So, a TBI animal model was established and we compared biomarkers of M1 and M2MG between the controls and experimental animals receiving hyperbaric oxygen therapy (HBOT). This study aimed to explore whether HBOT was an effective method to improve neural functional recovery via promoting the polarization of MG into M2 after TBI.

Methods: The rats were randomly divided into four groups: SH (Sham-operated), SH + HBO (hyperbaric oxygen), TBI, and TBI + HBO. Each group included 42 rats, and each of these were divided into the following groups: 1, 6, 12, 24, 72 h, 7, and 14 days. The expression of M1 biomarker inducible nitric oxide synthase (iNOS), M2 biomarker arginase 1 (Arg1), associated cytokine tumor necrosis factor-alpha (TNF-alpha), and transforming growth factor-beta 1 (TGF-beta 1) was evaluated after the observation time.

Results: TBI significantly increased the expression levels of M1 marker iNOS and M2 markers Arg1 at different time points. The increased expression of iNOS was suppressed, while the expression level of Arg1 was enhanced by HBOT. Moreover, HBOT suppressed the pro-inflammatory TNF-alpha secreted by M1, and promoting the anti-inflammatory TGF-1 beta.

Conclusions: In the present study, HBOT showed the effects on shift of M1 toward M2 phenotype with increased expression of M2 biomarkers and decreased expression of M1 biomarkers in the early stage after TBI.

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TI Microglia as therapeutic targets after neurological injury: strategy for

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SO EXPERT OPINION ON THERAPEUTIC TARGETS

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INCREASED EXPRESSION; P2Y(12) RECEPTOR; IMMUNE-RESPONSE

AB Introduction Microglia is the resident tissue macrophages of the central nervous system. Prolonged microglial activation often occurs after traumatic brain injury and is associated with deteriorating neurocognitive outcomes. Resolution of microglial activation is associated with limited tissue loss and improved neurocognitive outcomes. Limiting the prolonged pro-inflammatory response and the associated secondary tissue injury provides the rationale and scientific premise for considering microglia as a therapeutic target. Areas Covered In this review, we discuss markers of microglial activation, such as immunophenotype and microglial response to injury, including cytokine/chemokine release, free radical formation, morphology, phagocytosis, and metabolic shifts. We compare the origin and role in neuroinflammation of microglia and monocytes/macrophages. We review potential therapeutic targets to shift microglial polarization. Finally, we review the effect of cell therapy on microglia. Expert Opinion Dysregulated microglial activation after neurologic injury, such as traumatic brain injury, can worsen tissue damage and functional outcomes. There are potential targets in microglia to attenuate this activation, such as proteins and molecules that regulate microglia polarization. Cellular therapeutics that limit, but do not eliminate, the inflammatory response have improved outcomes in animal models by reducing pro-inflammatory microglial activation via secondary signaling. These findings have been replicated in early phase clinical trials.

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TI Cordycepin confers long-term neuroprotection via inhibiting neutrophil

infiltration and neuroinflammation after traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE White matter injury; Neutrophil; Microglia; macrophage; Blood-brain

barrier; Traumatic brain injury

ID WHITE-MATTER; BARRIER; PROTECTS; DAMAGE; MICE; INFLAMMATION; DYNAMICS;

MODELS; STROKE

AB BackgroundThe secondary injury caused by traumatic brain injury (TBI), especially white matter injury (WMI), is highly sensitive to neuroinflammation, which further leads to unfavored long-term outcomes. Although the cross-talk between the three active events, immune cell infiltration, BBB breakdown, and proinflammatory microglial/macrophage polarization, plays a role in the vicious cycle, its mechanisms are not fully understood. It has been reported that cordycepin, an extract from Cordyceps militaris, can inhibit TBI-induced neuroinflammation although the long-term effects of cordycepin remain unknown. Here, we report our investigation of cordycepin's long-term neuroprotective function and its underlying immunological mechanism.MethodsTBI mice model was established with a controlled cortical impact (CCI) method. Cordycepin was intraperitoneally administered twice daily for a week. Neurological outcomes were assessed by behavioral tests, including grid walking test, cylinder test, wire hang test, and rotarod test. Immunofluorescence staining, transmission electron microscopy, and electrophysiology recording were employed to assess histological and functional lesions. Quantitative-PCR and flow cytometry were used to detect neuroinflammation. The tracers of Sulfo-NHS-biotin and Evans blue were assessed for the blood-brain barrier (BBB) leakage. Western blot and gelatin zymography were used to analyze protein activity or expression. Neutrophil depletion in vivo was performed via using Ly6G antibody intraperitoneal injection.ResultsCordycepin administration ameliorated long-term neurological deficits and reduced neuronal tissue loss in TBI mice. Meanwhile, the long-term integrity of white matter was also preserved, which was revealed in multiple dimensions, such as morphology, histology, ultrastructure, and electrical conductivity. Cordycepin administration inhibited microglia/macrophage pro-inflammatory polarization and promoted anti-inflammatory polarization after TBI. BBB breach was attenuated by cordycepin administration at 3 days after TBI. Cordycepin suppressed the activities of MMP-2 and MMP-9 and the neutrophil infiltration at 3 days after TBI. Moreover, neutrophil depletion provided a cordycepin-like effect, and cordycepin administration united with neutrophil depletion did not show a benefit of superposition.ConclusionsThe long-term neuroprotective function of cordycepin via suppressing neutrophil infiltration after TBI, thereby preserving BBB integrity and changing microglia/macrophage polarization. These findings provide significant clinical potentials to improve the quality of life for TBI patients.

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TI JM-20 Treatment After Mild Traumatic Brain Injury Reduces Glial Cell

Pro-inflammatory Signaling and Behavioral and Cognitive Deficits by

Increasing Neurotrophin Expression

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article; Early Access

DE JM-20; Neuroprotection; Neuroinflammation; Multi-target; Astrocytes;

Microglia

ID NERVE GROWTH-FACTOR; NECROSIS-FACTOR-ALPHA; GENE-EXPRESSION; NEURONAL

LOSS; NITRIC-OXIDE; GDNF; NEUROINFLAMMATION; INVOLVEMENT; IMPAIRMENTS;

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AB Traumatic brain injury (TBI) is considered a public health problem and is often related to motor and cognitive disabilities, besides behavioral and emotional changes that may remain for the rest of the subject's life. Resident astrocytes and microglia are the first cell types to start the inflammatory cascades following TBI. It is widely known that continuous or excessive neuroinflammation may trigger many neuropathologies. Despite the large numbers of TBI cases, there is no effective pharmacological treatment available. This study aimed to investigate the effects of the new hybrid molecule 3-ethoxycarbonyl-2-methyl-4-(2-nitrophenyl)-4,11-dihydro1H-pyrido[2,3-b][1,5]benzodiazepine (JM-20) on TBI outcomes. Male Wistar rats were submitted to a weight drop model of mild TBI and treated with a single dose of JM-20 (8 mg/kg). Twenty-four hours after TBI, JM-20-treated animals showed improvements on locomotor and exploratory activities, and short-term memory deficits induced by TBI improved as well. Brain edema was present in TBI animals and the JM-20 treatment was able to prevent this change. JM-20 was also able to attenuate neuroinflammation cascades by preventing glial cells-microglia and astrocytes-from exacerbated activation, consequently reducing pro-inflammatory cytokine levels (TNF-alpha and IL-1 beta). BDNF mRNA level was decreased 24 h after TBI because of neuroinflammation cascades; however, JM-20 restored the levels. JM-20 also increased GDNF and NGF levels. These results support the JM-20 neuroprotective role to treat mild TBI by reducing the initial damage and limiting long-term secondary degeneration after TBI.

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NR 92

TC 4

Z9 5

U1 0

U2 3

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J9 MOL NEUROBIOL

JI Mol. Neurobiol.

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WC Neurosciences

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ER

PT J

AU Basit, RH

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AF Basit, Raja Haseeb

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TI In vitro model of traumatic brain injury to screen neuro-regenerative

biomaterials

SO MATERIALS SCIENCE & ENGINEERING C-MATERIALS FOR BIOLOGICAL APPLICATIONS

LA English

DT Article; Early Access

DE Microglia; Scarring; Immune cells; Astrocyte; Surgical materials

ID PLATFORM; REVEALS; REPAIR

AB Penetrating traumatic brain injury (pTBI) causes serious neurological deficits with no clinical regenerative therapies currently available. Tissue engineering strategies using biomaterial-based 'structural bridges' offer high potential to promote neural regeneration post-injury. This includes surgical grade materials which can be repurposed as biological scaffolds to overcome challenges associated with long approval processes and scaleup for human application. However, high throughput, pathomimetic models of pTBI are lacking for the developmental testing of such neuro-materials, representing a bottleneck in this rapidly emergent field. We have established a high throughput and facile culture model containing the major neural cell types which govern biomaterial handling in the central nervous system. We show that induction of traumatic injuries was feasible in the model, with post-injury implantation of a surgical grade biomaterial. Cellular imaging in lesions was achievable using standard epifluorescence microscopy methods. Key pathological features of pTBI were evident in vitro namely immune cell infiltration of lesions/biomaterial, with responses characteristic of cell scarring, namely hypertrophic astrocytes with GFAP upregulation. Based on our observations, we consider the high throughput, inexpensive and facile pTBI model can be used to study biomaterial 'implantation' and evaluate neural cell-biomaterial responses. The model is highly versatile to test a range of laboratory and clinical grade materials for neural regeneration.

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NR 30

TC 3

Z9 3

U1 8

U2 16

PU ELSEVIER

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JI Mater. Sci. Eng. C-Mater. Biol. Appl.

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ER

PT J

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TI HIF-1α aggravated traumatic brain injury by NLRP3 inflammasome-mediated

pyroptosis and activation of microglia

SO JOURNAL OF CHEMICAL NEUROANATOMY

LA English

DT Article; Early Access

DE Traumatic brain injury; HIF-1 alpha; NLRP3 inflammasome; Pyroptosis;

Microglia

ID BEHAVIORAL DEFICITS; NEUROPROTECTION; IMPACT; EDEMA; MODEL

AB Hypoxia inducible factor 1 alpha (HIF-1 alpha) is involved in regulating the biological functions of neuronal death after traumatic brain injury (TBI), and attaches importance in the inflammatory response, but its potential mechanism is still unknown. Our study aimed to explore the regulatory mechanism between HIF-1 alpha and NLRP3 inflammasome after TBI. Male mice underwent controlled cortical impact (CCI) or sham-operated procedures. Brain water content and blood-brain barrier permeability were measured at the indicated time after TBI. The behavioral performance, ELISA, immunofluorescence, and western blot analysis were used to determine whether HIF-1 alpha specifically targeted TBI-induced pymptosis. We discovered that TBI-induced brain injury caused by external mechanical forces is characterized by edema and blood-brain barrier disorder, and the release of IL-1 beta, IL-18, and LDH and upregulation of HIF-1 alpha expression, reaching the peak on the third day post-TBI. In addition, HIF-1 alpha accumulated NLRP3 inflammasome-mediated pymptosis and activation of microglia. The protein expressions of NLRP3, GSDMD, GSDMD-N, pro-caspase 1, and cleaved caspase 1 were markedly increased in the injured cortex, which were restored to normal levels by the interference of HIF-1 alpha. The inactivation of HIF-1 alpha conferred neumprotection and alleviated brain injury after TBI. HIF-1 alpha was implicated in TBI-induced brain injury, aggravated NLRP3 inflammasome -mediated pymptosis, and the activation of microglia, which provided a potential target for treating TBI.

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NR 41

TC 31

Z9 33

U1 2

U2 13

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J9 J CHEM NEUROANAT

JI J. Chem. Neuroanat.

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WC Biochemistry & Molecular Biology; Neurosciences

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ER

PT J

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TI Visual deficits after traumatic brain injury

SO HISTOLOGY AND HISTOPATHOLOGY

LA English

DT Review

DE Retina; TBI; Blast injury; Microglia; Muller; Vision; Chronic traumatic

encephalopathy; Traumatic optic neuropathy; Mesenchymal stem cells; RGC

ID RETINAL GANGLION-CELLS; MOUSE MODEL; BLAST INJURY; DAMAGE; PREVENTION;

OVERPRESSURE; THERAPY

AB Traumatic brain injury (TBI) is frequently described as any head injury ceasing the brain's normal function. Anatomically, developmentally, and physiologically, the eye is deemed as an extension of the brain. Vision in TBI is underrepresented, and the number of active clinical trials in this field are sparse. Frequently, visual problems are overlooked at the time of TBI, often resulting in progressive vision loss, lengthening, and impairing rehabilitation. TBI can be either penetrative or non-penetrative, associated with degeneration of neurons, apoptotic cell death, inflammation, microglial activation, hemorrhage associated with vascular dysfunction; however, precise animal modeling that mimics the extensive visual deficits of TBI pathology remain elusive. Recent works in both the diagnostics and therapeutics fields are starting to make substantial progress in the right direction. Discussion of current advancements in TBI animal models and the recent pathophysiological findings related to the neuro-glia-vascular unit (NVU) will help elucidate novel targets for potential lines of therapeutics. Only over the past decade have newer pharmaceutical and stem cell-based treatments begun to come to light. The potency for these new lines of TBI specific curatives will be discussed along with the review of current blast-induced TBI models, providing potential directions for future research.

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preparation of the manuscript. Competing interests. RG is a co-founder

and hold equity in Cell Care Therapeutics Inc., whose interest is in the

use of adipose-derived stromal cells in visual disorders. None of the

other authors declare any financial conflicts.

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TC 6

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U1 0

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PU F HERNANDEZ

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TI Traumatic brain injury results in unique microglial and astrocyte

transcriptomes enriched for type I interferon response

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Traumatic brain injury; Microglia; Astrocytes; Type I interferon

ID UP-REGULATION; NEUROINFLAMMATION; NEURODEGENERATION; INFLAMMATION;

MEMORY

AB Background Traumatic brain injury (TBI) is a leading cause of death and disability that lacks neuroprotective therapies. Following a TBI, secondary injury response pathways are activated and contribute to ongoing neurodegeneration. Microglia and astrocytes are critical neuroimmune modulators with early and persistent reactivity following a TBI. Although histologic glial reactivity is well established, a precise understanding of microglia and astrocyte function following trauma remains unknown. Methods Adult male C57BL/6J mice underwent either fluid percussion or sham injury. RNA sequencing of concurrently isolated microglia and astrocytes was conducted 7 days post-injury to evaluate cell-type-specific transcriptional responses to TBI. Dual in situ hybridization and immunofluorescence were used to validate the TBI-induced gene expression changes in microglia and astrocytes and to identify spatial orientation of cells expressing these genes. Comparative analysis was performed between our glial transcriptomes and those from prior reports in mild TBI and other neurologic diseases to determine if severe TBI induces unique states of microglial and astrocyte activation. Results Our findings revealed sustained, lineage-specific transcriptional changes in both microglia and astrocytes, with microglia showing a greater transcriptional response than astrocytes at this subacute time point. Microglia and astrocytes showed overlapping enrichment for genes related to type I interferon signaling and MHC class I antigen presentation. The microglia and astrocyte transcriptional response to severe TBI was distinct from prior reports in mild TBI and other neurodegenerative and neuroinflammatory diseases. Conclusion Concurrent lineage-specific analysis revealed novel TBI-specific transcriptional changes; these findings highlight the importance of cell-type-specific analysis of glial reactivity following TBI and may assist with the identification of novel, targeted therapies.

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TI Acute TBK1/IKK-ε Inhibition Enhances the Generation of

Disease-Associated Microglia-Like Phenotype Upon Cortical Stab-Wound

Injury

SO FRONTIERS IN AGING NEUROSCIENCE

LA English

DT Article

DE TBK1; microglia; traumatic brain injury; amlexanox; neuroinflammation;

stab wound injury

ID TRAUMATIC BRAIN-INJURY; IKK-EPSILON; TBK1; EPIDEMIOLOGY; NEURONS;

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AB Traumatic brain injury has a poorer prognosis in elderly patients, possibly because of the enhanced inflammatory response characteristic of advanced age, known as "inflammaging." Recently, reduced activation of the TANK-Binding-Kinase 1 (Tbk1) pathway has been linked to age-associated neurodegeneration and neuroinflammation. Here we investigated how the blockade of Tbk1 and of the closely related IKK-epsilon by the small molecule Amlexanox could modify the microglial and immune response to cortical stab-wound injury in mice. We demonstrated that Tbk1/IKK-epsilon inhibition resulted in a massive expansion of microglial cells characterized by the TMEM119(+)/CD11c(+) phenotype, expressing high levels of CD68 and CD317, and with the upregulation of Cst7a, Prgn and Ccl4 and the decrease in the expression levels of Tmem119 itself and P2yr12, thus a profile close to Disease-Associated Microglia (DAM, a subset of reactive microglia abundant in Alzheimer's Disease and other neurodegenerative conditions). Furthermore, Tbk1/IKK-epsilon inhibition increased the infiltration of CD3(+) lymphocytes, CD169(+) macrophages and CD11c(+)/CD169(+) cells. The enhanced immune response was associated with increased expression of Il-33, Ifn-g, Il-17, and Il-19. This upsurge in the response to the stab wound was associated with the expanded astroglial scars and increased deposition of chondroitin-sulfate proteoglycans at 7 days post injury. Thus, Tbk1/IKK-epsilon blockade results in a massive expansion of microglial cells with a phenotype resembling DAM and with the substantial enhancement of neuroinflammatory responses. In this context, the induction of DAM is associated with a detrimental outcome such as larger injury-related glial scars. Thus, the Tbk1/IKK-epsilon pathway is critical to repress neuroinflammation upon stab-wound injury and Tbk1/IKK-epsilon inhibitors may provide an innovative approach to investigate the consequences of DAM induction.

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TI Intranasal delivery of interleukin-4 attenuates chronic cognitive

deficits via beneficial microglial responses in experimental traumatic

brain injury

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article; Early Access

DE Cognitive function; microglia polarization; long-term potentiation; PPAR

gamma; DTI

ID LONG-TERM POTENTIATION; AMYLOID PRECURSOR PROTEIN; MICROGLIA/MACROPHAGE

POLARIZATION; SPATIAL MEMORY; NEURONAL LOSS; WHITE-MATTER; SPINAL-CORD;

ROSIGLITAZONE; ACTIVATION; IL-4

AB Traumatic brain injury (TBI) is commonly followed by long-term cognitive deficits that severely impact the quality of life in survivors. Recent studies suggest that microglial/macrophage (Mi/M phi) polarization could have multidimensional impacts on post-TBI neurological outcomes. Here, we report that repetitive intranasal delivery of interleukin-4 (IL-4) nanoparticles for 4 weeks after controlled cortical impact improved hippocampus-dependent spatial and non-spatial cognitive functions in adult C57BL6 mice, as assessed by a battery of neurobehavioral tests for up to 5 weeks after TBI. IL-4-elicited enhancement of cognitive functions was associated with improvements in the integrity of the hippocampus at the functional (e.g., long-term potentiation) and structural levels (CA3 neuronal loss, diffusion tensor imaging of white matter tracts, etc.). Mechanistically, IL-4 increased the expression of PPAR gamma and arginase-1 within Mi/M phi, thereby driving microglia toward a global inflammation-resolving phenotype. Notably, IL-4 failed to shift microglial phenotype after TBI in Mi/M phi-specific PPAR gamma knockout (mKO) mice, indicating an obligatory role for PPAR gamma in IL-4-induced Mi/M phi polarization. Accordingly, post-TBI treatment with IL-4 failed to improve hippocampal integrity or cognitive functions in PPAR gamma mKO mice. These results demonstrate that administration of exogenous IL-4 nanoparticles stimulates PPAR gamma-dependent beneficial Mi/M phi responses, and improves hippocampal function after TBI.

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TI Astrocyte-derived CCL7 promotes microglia-mediated inflammation

following traumatic brain injury

SO INTERNATIONAL IMMUNOPHARMACOLOGY

LA English

DT Article; Early Access

DE Traumatic brain injury; CCL7; Inflammation; Microglia; Astrocytes

ID CONTRIBUTES; INHIBITION; EXPRESSION; RATS

AB Microglia are immune cells of the central nervous system that mediate neuroinflammation. It is widely known that microglia-mediated inflammation in the brain contribute to the widespread tissue damage and neurological deficits in traumatic brain injury (TBI). However, the mechanisms responsible for this inflammatory response remain elusive. Here, we investigated the role of astrocyte-derived chemokine (C-C motif) ligand 7 (CCL7) in microglial-controlled inflammation following TBI. Our results demonstrated that astrocyte-derived CCL7 induced microglial activation and the release of proinflammatory mediators in the cortex and serum of rats that underwent experimental TBI. Furthermore, CCL7 knockout improved microglia-controlled inflammation, brain morphology and neurological dysfunction following TBI. In vitro, CCL7-siRNA attenuated the LPS-induced expression of pro-inflammatory markers in the co-culture of microglia and astrocytes. Collectively, our findings uncover an important role for astrocyte-derived CCL7 in promoting microglia-mediated inflammation after TBI and suggests CCL7 could serve as a potential therapeutic strategy for attenuating TBI by inhibiting microglial activation.

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TI Intramuscular insulin-like growth factor-1 gene therapy modulates

reactive microglia after traumatic brain injury

SO BRAIN RESEARCH BULLETIN

LA English

DT Article; Early Access

DE IGF-1; Neuroinflammation; Neuroprotection; Traumatic brain injury;

Astrocytes; Microglia

ID INCREASES HIPPOCAMPAL NEUROGENESIS; ANIMAL-MODELS; IGF-1; RATS;

NEUROPROTECTION; TARGETS; MEMORY; DAMAGE; MOUSE

AB Reactive gliosis is a key feature and an important pathophysiological mechanism underlying chronic neurodegeneration following traumatic brain injury (TBI). In this study, we have explored the effects of intramuscular IGF-1 gene therapy on reactive gliosis and functional outcome after an injury of the cerebral cortex. Young adult male rats were intramuscularly injected with a recombinant adenoviral construct harboring the cDNA of human IGF-1 (RAd-IGF1), with a control vector expressing green fluorescent protein (RAd-GFP) or PBS as control. Three weeks after the intramuscular injections of adenoviral vectors, animals were subjected to a unilateral penetrating brain injury. The data revealed that RAd-IGF1 gene therapy significantly increased serum IGF1 levels and improved working memory performance after one week of TBI as compared to PBS or RAd-GFP lesioned animals. At the same time, when we analyzed the effects of therapy on glial scar formation, the treatment with RAd-IGF1 did not modify the number of glial fibrillary acidic protein (GFAP) positive cells, but we observed a decrease in vimentin immunoreactive astrocytes at 7 days post-lesion in the injured hemisphere compared to RAd-GFP group. Moreover, IGF-1 gene therapy reduced the number of Iba1+ cells with reactive phenotype and the number of MHCII + cells in the injured hemisphere. These results suggest that intramuscular IGF-1 gene therapy may represent a new approach to prevent traumatic brain injury outcomes in rats.

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TI Long-term cognitive deficits after traumatic brain injury associated

with microglia activation

SO CLINICAL IMMUNOLOGY

LA English

DT Article; Early Access

DE Traumatic brain injury; Microglia; Cognition; Spatial memory;

Infiltrating macrophages; Chronic inflammation

ID REVEALS; MILD; NEUROINFLAMMATION; INFLAMMATION; APOPTOSIS; SEVERITY;

MODERATE; SUBSETS; MEMORY; MICE

AB Traumatic Brain Injury (TBI) is the most prevalent of all head injuries. Microglia play an essential role in homeostasis and diseases of the central nervous system. We hypothesize that microglia may play a beneficial or detrimental role in TBI depending on their state of activation and duration. In this study, we evaluated whether TBI results in a spatiotemporal change in microglia phenotype and whether it affects sensory-motor or learning and memory functions in male C57BL/6 mice. We used a panel of neurological and behavioral tests and a multi-color flow cytometry-based data analysis followed by unsupervised clustering to evaluate isolated microglia from injured brain tissue. We characterized several microglial phenotypes and their association with cognitive deficits. TBI results in a spatiotemporal increase in activated microglia that correlated negatively with spatial learning and memory at 35 days post-injury. These observations could define therapeutic windows and accelerate translational research to improve patient outcomes.

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TI Doxycycline alleviates acute traumatic brain injury by suppressing

neuroinflammation and apoptosis in a mouse model

SO JOURNAL OF NEUROIMMUNOLOGY

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DT Article; Early Access

DE Traumatic brain injury; Neuroinflammation; Microglia; Doxycycline

ID RECRUITMENT; FLUID; NEUROPROTECTION; EXPRESSION; BIOMARKERS; REDUCTION;

MEDIATORS; HEALTH; DAMAGE

AB Traumatic brain injury (TBI) is one of the significant causes of death among young people worldwide. Doxycycline (DOX), an antibiotic with anti-inflammatory effects, has not been used as a therapeutic agent to modify the inflammatory response after the traumatic brain injury. In this study, intraperitoneal administration of DOX reduced significantly the acute inflammatory markers like IL-6 and CD3, microglial migration to the damaged area marked with Iba-1, and neuronal apoptosis assessed with TUNEL assay at 72 h after the trauma. The low dose, 10 mg/kg of DOX had a dominant anti-inflammatory effect; while the high dose, 100 mg/kg of DOX, was more effective in decreasing neuronal apoptosis. In early hours after the head trauma, use of a low dose (10 mg/ kg) of DOX for decreasing the acute form of inflammation followed by a high dose (100 mg/kg) for the antiapoptotic effects particularly in severe head traumas, would be a promising approach to alleviate the brain injury.

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TI Post-traumatic Neuroinflammation: Relevance to Pediatrics

SO PEDIATRIC NEUROLOGY

LA English

DT Article; Early Access

DE Fractal morphology analysis; Cosyntropin; Chemokines; Cytokines;

Microglia; Traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; ANIMAL-MODELS; MICROGLIA; INFLAMMATION;

NEUROPROTECTION; RECEPTORS; TURNOVER; NEURODEGENERATION; INTERLEUKIN-10;

MELANOCORTINS

AB Both detrimental and beneficial effects of post-traumatic neuroinflammation have become a major research focus as they offer the potential for immediate as well as delayed targeted reparative therapies. Understanding the complex interactions of central and peripheral immunocompetent cells as well as their mediators on brain injury and recovery is complicated by the temporal, regional, and developmental differences in their response to injuries. Microglia, the brain-resident macrophages, have become central in these investigations as they serve a major surveillance function, have the ability to react swiftly to injury, recruit various cellular and chemical mediators, and monitor the reparative/degenerative processes. In this review we describe selected aspects of this burgeoning literature, describing the critical role of cytokines and chemokines, microglia, advances in neuroimaging, genetics and fractal morphology analysis, our research efforts in this area, and selected aspects of pediatric post-traumatic neuroinflammation. (c) 2021 Elsevier Inc. All rights reserved.

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TI Novel microglia-mediated mechanisms underlying synaptic loss and

cognitive impairment after traumatic brain injury

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article; Early Access

DE Traumatic brain injury; Microglia; Neuroinflammation; Learning and

memory

ID SEX-DIFFERENCES; MEMORY; INFLAMMATION; DEFICITS; MODELS

AB Traumatic brain injury (TBI) is one of the leading causes of long-term neurological disability in the world. Currently, there are no therapeutics for treating the deleterious consequences of brain trauma; this is in part due to a lack of complete understanding of cellular processes that underlie TBI-related pathologies. Following TBI, microglia, the brain resident immune cells, turn into a "reactive" state characterized by the production of inflammatory mediators that contribute to the development of cognitive deficits. Utilizing multimodal, state-of-the-art techniques that widely span from ultrastructural analysis to optogenetic interrogation of circuit function, we investigated the reactive microglia phenotype one week after injury when learning and memory deficits are also measured. Microglia displayed increased: (i) phagocytic activity in vivo, (ii) synaptic engulfment, (iii) increased neuronal contact, including with dendrites and somata (termed 'satellite microglia'). Functionally, satellite microglia might impact somatic inhibition as demonstrated by the associated reduction in inhibitory synaptic drive. Cumulatively, here we demonstrate novel microglia-mediated mechanisms that may contribute to synaptic loss and cognitive impairment after traumatic brain injury.

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TI Traumatic Brain Injury Induces cGAS Activation and Type I Interferon

Signaling in Aged Mice

SO FRONTIERS IN IMMUNOLOGY

LA English

DT Article

DE type I interferons; traumatic brain injury; aging; neuroinflammation;

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ID GENE-EXPRESSION; IFN-BETA; CNS; RESPONSES; DRIVES; NEUROINFLAMMATION;

NEURODEGENERATION; INDUCTION; MICROGLIA; PROGRESSION

AB Aging adversely affects inflammatory processes in the brain, which has important implications in the progression of neurodegenerative disease. Following traumatic brain injury (TBI), aged animals exhibit worsened neurological function and exacerbated microglial-associated neuroinflammation. Type I Interferons (IFN-I) contribute to the development of TBI neuropathology. Further, the Cyclic GMP-AMP Synthase (cGAS) and Stimulator of Interferon Genes (STING) pathway, a key inducer of IFN-I responses, has been implicated in neuroinflammatory activity in several age-related neurodegenerative diseases. Here, we set out to investigate the effects of TBI on cGAS/STING activation, IFN-I signaling and neuroinflammation in young and aged C57Bl/6 male mice. Using a controlled cortical impact model, we evaluated transcriptomic changes in the injured cortex at 24 hours post-injury, and confirmed activation of key neuroinflammatory pathways in biochemical studies. TBI induced changes were highly enriched for transcripts that were involved in inflammatory responses to stress and host defense. Deeper analysis revealed that TBI increased expression of IFN-I related genes (e.g. Ifnb1, Irf7, Ifi204, Isg15) and IFN-I signaling in the injured cortex of aged compared to young mice. There was also a significant age-related increase in the activation of the DNA-recognition pathway, cGAS, which is a key mechanism to propagate IFN-I responses. Finally, enhanced IFN-I signaling in the aged TBI brain was confirmed by increased phosphorylation of STAT1, an important IFN-I effector molecule. This age-related activation of cGAS and IFN-I signaling may prove to be a mechanistic link between microglial-associated neuroinflammation and neurodegeneration in the aged TBI brain.

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TI Neonatal microglia and proteinase inhibitors-treated adult microglia

improve traumatic brain injury in rats by resolving the

neuroinflammation

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DE adult microglia; neonatal microglia; protease inhibitor; traumatic brain

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ID PATHOPHYSIOLOGY; ACTIVATION; EXPRESSION; PHENOTYPE; CELLS

AB Microglia participate in the regulation of neuroinflammation caused by traumatic brain injury (TBI). This research aimed to explore the repair effects of intracranial injection of neonatal microglia or protease-treated adult microglia on TBI in rat model. Lateral fluid percussion injury was used to establish rat brain injury model. E64 and serpinA3N were employed for the treatment of adult microglia. Cleaved caspase-3 level was analyzed through immunoblotting assay. Enzyme-linked immunosorbent assay was employed to analyze cytokine and chemokine levels. Astrocytosis and microgliosis were shown by immunofluorescence. The cognitive function of rats was analyzed by water maze. The injection of neonatal microglia inhibited cell apoptosis, reduced astrocytosis and microgliosis, decreased the level of chemokines and cytokines in cortex and ipsilateral hippocampus, and improved cognitive function of TBI rat model. The transplantation of peptidase inhibitors-treated adult microglia also inhibited cell apoptosis, reduced astrocytosis and microgliosis, and improved cognitive function of rats with TBI. The transplantation of either neonatal microglia or peptidase inhibitors-treated adult microglia significantly inhibited the pathogenesis of TBI in rat model, while untreated adult microglia showed no significant effect.

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Liu, Bin

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TI Traumatic Brain Injury Accelerates the Onset of Cognitive Dysfunction

and Aggravates Alzheimer's-Like Pathology in the Hippocampus by Altering

the Phenotype of Microglia in the APP/PS1 Mouse Model

SO FRONTIERS IN NEUROLOGY

LA English

DT Article

DE traumatic brain injury; Alzheimer's disease; A beta plaque pathology;

neuroinflammation; microglia

ID OBJECT RECOGNITION; DISEASE; EXPRESSION; RECEPTOR; EPIDEMIOLOGY;

PHAGOCYTOSIS; POPULATION; RATS

AB An increasing number of studies have suggested that traumatic brain injury (TBI) is associated with some neurodegenerative diseases, including Alzheimer's disease (AD). Various aspects of the mechanism of TBI-induced AD have been elucidated. However, there are also studies opposing the view that TBI is one of the causes of AD. In the present study, we demonstrated that TBI exacerbated the disruption of hippocampal-dependent learning and memory, worsened the reductions in neuronal cell density and synapse formation, and aggravated the deposition of A beta plaques in the hippocampi of APP/PS1 mice. We also found that TBI rapidly activated microglia in the central nervous system (CNS) and that this effect lasted for at least for 3 weeks. Furthermore, TBI boosted A beta-related microglia-mediated neuroinflammation in the hippocampi of APP/PS1 mice and the transformation of microglia toward the proinflammatory phenotype. Therefore, our experiments suggest that TBI accelerates the onset of cognitive dysfunction and Alzheimer-like pathology in the APP/PS1 mouse model, at least partly by altering microglial reactions and polarization.

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TI Metformin reduces neuroinflammation and improves cognitive functions

after traumatic brain injury

SO NEUROSCIENCE RESEARCH

LA English

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DE Metformin; TBI; Neuroinflammation; Microglia; Par1; MARK

ID ACTIVATED PROTEIN-KINASE; SYNAPTIC PLASTICITY; KAPPA-B; PHOSPHORYLATION;

MEMORY; LKB1; NEUROGENESIS; EPIDEMIOLOGY; ASTROCYTES; BEHAVIOR

AB Within the brain, traumatic brain injury (TBI) alters synaptic plasticity and increases neuroinflammation and neuronal death. Yet, there lacks effective TBI treatments providing pleiotropic beneficial effects on these diverse cellular processes necessary for functional recovery. Here, we show the diabetes drug, metformin, significantly improves cognitive functions after controlled cortical impact (CCI) injury in mice, showing improved spatial learning and nest building. Furthermore, injured animals treated with metformin exhibit increased ramification of microglia processes, indicating reduced neuroinflammation. Finally, metformin treatment in vitro increased neuronal activation of partitioning defective 1 (Par1), a family of Ser/Thr kinases playing a key role in synaptic plasticity and neuroinflammation. These results suggest metformin is a promising therapeutic agent for targeting multiple cellular processes necessary for functional TBI recovery. (c) 2021 Elsevier B.V. and Japan Neuroscience Society. All rights reserved.

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TI Rapid morphologic changes to microglial cells and upregulation of mixed

microglial activation state markers induced by P2X7 receptor stimulation

and increased intraocular pressure

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Microglial M1; M2 activation; Neuroinflammation; Sholl analysis;

Chemoattraction migration; ATP release; Intraocular pressure; Iba1;

Glaucoma; Traumatic brain injury

ID RETINAL GANGLION-CELLS; CENTRAL-NERVOUS-SYSTEM; P2X(7) RECEPTOR; MOUSE

MODEL; SUSTAINED ELEVATION; EXTRACELLULAR ATP; AXONAL DAMAGE; RELEASE;

RAT; ANTAGONIST

AB Background The identification of endogenous signals that lead to microglial activation is a key step in understanding neuroinflammatory cascades. As ATP release accompanies mechanical strain to neural tissue, and as the P2X7 receptor for ATP is expressed on microglial cells, we examined the morphological and molecular consequences of P2X7 receptor stimulation in vivo and in vitro and investigated the contribution of the P2X7 receptor in a model of increased intraocular pressure (IOP). Methods In vivo experiments involved intravitreal injections and both transient and sustained elevation of IOP. In vitro experiments were performed on isolated mouse retinal and brain microglial cells. Morphological changes were quantified in vivo using Sholl analysis. Expression of mRNA for M1- and M2-like genes was determined with qPCR. The luciferin/luciferase assay quantified retinal ATP release while fura-2 indicated cytoplasmic calcium. Microglial migration was monitored with a Boyden chamber. Results Sholl analysis of Iba1-stained cells showed retraction of microglial ramifications 1 day after injection of P2X7 receptor agonist BzATP into mouse retinae. Mean branch length of ramifications also decreased, while cell body size and expression of Nos2, Tnfa, Arg1, and Chil3 mRNA increased. BzATP induced similar morphological changes in ex vivo tissue isolated from Cx3CR1(+/GFP) mice, suggesting recruitment of external cells was unnecessary. Immunohistochemistry suggested primary microglial cultures expressed the P2X7 receptor, while functional expression was demonstrated with Ca2+ elevation by BzATP and block by specific antagonist A839977. BzATP induced process retraction and cell body enlargement within minutes in isolated microglial cells and increased Nos2 and Arg1. While ATP increased microglial migration, this required the P2Y12 receptor and not P2X7 receptor. Transient elevation of IOP led to microglial process retraction, cell body enlargement, and gene upregulation paralleling changes observed with BzATP injection, in addition to retinal ATP release. Pressure-dependent changes were reduced in P2X7(-/-) mice. Death of retinal ganglion cells accompanied increased IOP in C57Bl/6J, but not P2X7(-/-) mice, and neuronal loss showed some association with microglial activation. Conclusions P2X7 receptor stimulation induced rapid morphological activation of microglial cells, including process retraction and cell body enlargement, and upregulation of markers linked to both M1- and M2-type activation. Parallel responses accompanied IOP elevation, suggesting ATP release and P2X7 receptor stimulation influence the early microglial response to increased pressure.

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TI CCR2 deficiency alters activation of microglia subsets in traumatic

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AB In traumatic brain injury (TBI), a diversity of brain resident and peripherally derived myeloid cells have the potential to worsen damage and/or to assist in healing. We define the heterogeneity of microglia and macrophage phenotypes during TBI in wild-type (WT) mice and Ccr2(-/-) mice, which lack macrophage influx following TBI and are resistant to brain damage. We use unbiased single-cell RNA sequencing methods to uncover 25 microglia, monocyte/macrophage, and dendritic cell subsets in acute TBI and normal brains. We find alterations in transcriptional profiles of microglia subsets in Ccr2(-/-) TBI mice compared to WT TBI mice indicating that infiltrating monocytes/macrophages influence microglia activation to promote a type I IFN response. Preclinical pharmacological blockade of hCCR2 after injury reduces expression of IFN-responsive gene, Irf7, and improves outcomes. These data extend our understanding of myeloid cell diversity and crosstalk in brain trauma and identify therapeutic targets in myeloid subsets.

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TI Conditional Deletion of EphA4 on Cx3cr1-Expressing Microglia Fails to

Influence Histopathological Outcome and Blood Brain Barrier Disruption

Following Brain Injury

SO FRONTIERS IN MOLECULAR NEUROSCIENCE

LA English

DT Article

DE neuroinflammation; TMEM119; peripheral monocytes; Eph signaling; innate

immune; traumatic brain injury

ID UP-REGULATION; GENERATION; ACTIVATION; EXPRESSION; EPHRIN-B2; EPHB2

AB Erythropoietin-producing human hepatocellular receptors play a major role in central nervous system injury. Preclinical and clinical studies revealed the upregulation of erythropoietin-producing human hepatocellular A4 (EphA4) receptors in the brain after acute traumatic brain injury. We have previously reported that Cx3cr1-expressing cells in the peri-lesion show high levels of EphA4 after the induction of controlled cortical impact (CCI) injury in mice. Cx3cr1 is a fractalkine receptor expressed on both resident microglia and peripheral-derived macrophages. The current study aimed to determine the role of microglial-specific EphA4 in CCI-induced damage. We used Cx3cr1(CreER/+) knock-in/knock-out mice, which express EYFP in Cx3cr1-positive cells to establish microglia, EphA4-deficient mice following 1-month tamoxifen injection. Consistent with our previous findings, induction of CCI in wild-type (WT) Cx3cr1(CreER/+)EphA4(+/+) mice increased EphA4 expression on EYFP-positive cells in the peri-lesion. To distinguish between peripheral-derived macrophages and resident microglia, we exploited GFP bone marrow-chimeric mice and found that CCI injury increased EphA4 expression in microglia (TMEM119+GFP-) using immunohistochemistry. Using Cx3cr1(CreER/+)EphA4 (f/f) (KO) mice, we observed that the EphA4 mRNA transcript was undetected in microglia but remained present in whole blood when compared to WT. Finally, we found no difference in lesion volume or blood-brain barrier (BBB) disruption between WT and KO mice at 3 dpi. Our data demonstrate a nonessential role of microglial EphA4 in the acute histopathological outcome in response to CCI.

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TC 6

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TI Mild Traumatic Brain Injury Contributes to the Development of Delayed

Neuroinflammation

SO NEUROIMMUNOMODULATION

LA English

DT Article; Early Access

DE Mild traumatic brain injury; Microglia; IL1 beta; IL6; CD86;

Endocannabinoid system; Cannabinoid receptor type 1

ID CANNABINOID RECEPTORS; ACTIVATION; MICROGLIA; DEMENTIA; SURVIVAL

AB Introduction: In recent years, according to the literature, the problem of mild traumatic brain injury (mTBI) has become more and more urgent. Compared to moderate to severe craniocerebral trauma, mTBI occurs in a far greater number of people. The delayed sequelae caused by a single mTBI or multiple mTBIs are a significant public health problem. Methods: A weight-drop model was used for the formation of mTBI. A metal rod weighing 337 g with a blunt tip of 3 mm diameter was uplifted at 8 cm height and held by a lever. The trauma was created by lowering the lever and the rod and free-dropping onto the rat skull. In the cerebral cortex of experimental animals, we analyzed the level of microglial activity (Iba-1-positive system) and the expression of pro-inflammatory markers (IL1 beta, IL6, and CD86). Also, the expression level of the endocannabinoid system receptor (cannabinoid receptor type 1 [CB1]) was assessed in brain samples. Results: Experiments have shown that mTBI increases (1) the amount of microglia (iba-1) activated by the pro-inflammatory pathway (CD86); (2) the level of pro-inflammatory cytokines IL1 beta and IL6; and (3) CB1R activity. Conclusion: Overall, the results of this study indicate that mTBI induces a sustained neuroinflammatory response. (c) 2021 S. Karger AG, Base

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NR 41

TC 3

Z9 4

U1 0

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TI Downregulation of phosphoglycerate mutase 5 improves microglial

inflammasome activation after traumatic brain injury

SO CELL DEATH DISCOVERY

LA English

DT Article

ID CELL-DEATH; PHOSPHATASE; NECROSIS

AB Traumatic brain injury (TBI) is considered as the most common cause of disability and death, and therefore an effective intervention of cascade pathology of secondary brain injury promptly can be a potential therapeutic direction for TBI prognosis. Further study of the physiological mechanism of TBI is urgent and important. Phosphoglycerate mutase 5 (Pgam5), a mitochondrial protein, mediate mitochondrial homeostasis, cellular senescence, and necroptosis. This study evaluated the effects of Pgam5 on neurological deficits and neuroinflammation of controlled cortical impact-induced TBI mouse model in vivo and LPS + ATP-induced microglia model in vitro. Pgam5 was overexpressed post-TBI. Pgam5 depletion reduced pyroptosis-related molecules and improved microglia activation, neuron damage, tissue lesion, and neurological dysfunctions in TBI mice. RNA-seq analysis and molecular biology experiments demonstrated that Pgam5 might regulate inflammatory responses by affecting the post-translational modification and protein expression of related genes, including Nlrp3, caspase1, Gsdmd, and Il-1 beta. In microglia, Pgam5-sh abrogated LPS + ATP-induced Il-1 beta secretion through Asc oligomerization-mediated caspase-1 activation, which was independent of Rip3. The data demonstrate the critical role Pgam5 plays in nerve injury in the progression of TBI, which regulates Asc polymerization and subsequently caspase1 activation, and thus reveals a fundamental mechanism linking microglial inflammasome activation to Asc/caspase1-generated Il-1 beta-mediated neuroinflammation. Thus, our data indicate Pgam5 worsens physiological and neurological outcomes post-TBI, which may be a potential therapeutic target to improve neuroinflammation after TBI.

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PU SPRINGERNATURE

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J9 CELL DEATH DISCOV

JI Cell Death Discov.

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TI Phillyrin Prevents Neuroinflammation-Induced Blood-Brain Barrier Damage

Following Traumatic Brain Injury <i>via</i> Altering Microglial

Polarization

SO FRONTIERS IN PHARMACOLOGY

LA English

DT Article

DE traumatic brain injury; phillyrin; microglia; PPAR gamma;

neuroinflammation; blood-brain barrier

ID ACTIVATED RECEPTOR-GAMMA; NEUROLOGICAL DEFICITS; COGNITIVE DEFICITS;

OXIDATIVE STRESS; NEURONAL LOSS; M2 MICROGLIA; INFLAMMATION; APOPTOSIS;

FORSYTHIASIDE; PHARMACOLOGY

AB Background: Phillyrin (Phi) is the main polyphenolic compound found in Forsythia suspensa. Recent studies have revealed that Phi has potent antioxidative and anti-inflammatory effects. However, whether Phi could relieve blood-brain barrier (BBB) damage following traumatic brain injury (TBI) remains unknown.

Materials and Methods: Lipopolysaccharide (LPS) was used to activate primary microglia, which were then treated with different doses of Phi or the peroxisome proliferator-activated receptor-gamma (PPAR gamma) antagonist (GW9662). CCK-8 assay was used for evaluating cell viability, and the cytokines (including IL-1 beta, IL-6, TNF alpha, IL-4, IL-10, and TGF beta), microglial phenotypic markers (iNOS, COX2, and CD86 for "M1" polarization; Arg1, Ym1, and CD206 for "M2" polarization), PPAR gamma, and NF-kappa B were determined by RT-PCR, Western blot, or cellular immunofluorescence. Primary cultured mouse brain microvascular endothelial cells (BMECs) were stimulated by the condition medium (CM) from microglia. The cell viability, angiogenesis, and tight junction of BMECs were determined via CCK-8 assay, tube formation assay, and Western blot (for detecting MMP3, MMP9, ZO1, claudin-5, and occludin). Furthermore, the mouse TBI model was constructed and treated with Phi and/or GW9662. The BBB integrity was evaluated by H&E staining, Evans blue staining, and tissue immunofluorescence.

Results: Phi markedly restrained the pro-inflammatory ("M1" state) cytokines and promoted anti-inflammatory ("M2" polarization) cytokines in LPS-mediated microglia. Phi mitigated "M1" polarization and promoted "M2" polarization of microglia via enhancing PPAR gamma and inhibiting the NF-kappa B pathway. The PPAR gamma antagonist GW9662 significantly repressed Phi-mediated anti-inflammatory effects. Meanwhile, Phi enhanced the viability, tube formation ability, and cell junction of BMECs. In the TBI mouse model, Phi promoted "M2" polarization, whereas it repressed the "M1" polarization of microglia. In addition, Phi reduced TBI-mediated BBB damage. However, the protective effects of Phi were reversed mainly by GW9662 treatment.

Conclusion: Phi prevents BBB damage via inhibiting the neuroinflammation of microglia through the PPAR gamma/NF-kappa B pathway, which provides a potential therapeutic drug against TBI.

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TI Traumatic Brain Injury: Mechanisms of Glial Response

SO FRONTIERS IN PHYSIOLOGY

LA English

DT Review

DE traumatic brain injury; glia; astrocytes; microglia; oligodendrocytes

ID NF-KAPPA-B; GLUTAMATE TRANSPORTER GLT-1; AMYLOID PRECURSOR PROTEIN;

DIFFUSE AXONAL INJURY; WHITE-MATTER INJURY; CLOSED-HEAD-INJURY; REACTIVE

ASTROCYTES; MICROGLIAL ACTIVATION; INFLAMMASOME ACTIVATION;

PERICONTUSIONAL CORTEX

AB Traumatic brain injury (TBI) is a heterogeneous disorder that involves brain damage due to external forces. TBI is the main factor of death and morbidity in young males with a high incidence worldwide. TBI causes central nervous system (CNS) damage under a variety of mechanisms, including synaptic dysfunction, protein aggregation, mitochondrial dysfunction, oxidative stress, and neuroinflammation. Glial cells comprise most cells in CNS, which are mediators in the brain's response to TBI. In the CNS are present astrocytes, microglia, oligodendrocytes, and polydendrocytes (NG2 cells). Astrocytes play critical roles in brain's ion and water homeostasis, energy metabolism, blood-brain barrier, and immune response. In response to TBI, astrocytes change their morphology and protein expression. Microglia are the primary immune cells in the CNS with phagocytic activity. After TBI, microglia also change their morphology and release both pro and anti-inflammatory mediators. Oligodendrocytes are the myelin producers of the CNS, promoting axonal support. TBI causes oligodendrocyte apoptosis, demyelination, and axonal transport disruption. There are also various interactions between these glial cells and neurons in response to TBI that contribute to the pathophysiology of TBI. In this review, we summarize several glial hallmarks relevant for understanding the brain injury and neuronal damage under TBI conditions.</p>

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TI Raloxifene Modulates Microglia and Rescues Visual Deficits and Pathology

After Impact Traumatic Brain Injury

SO FRONTIERS IN NEUROSCIENCE

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DT Article

DE traumatic brain injury; visual deficits; microglia; CB2 receptors;

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ID RETINAL GANGLION-CELLS; CANNABINOID CB2 RECEPTOR; OPTIC-NERVE; MOUSE

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AB Mild traumatic brain injury (TBI) involves widespread axonal injury and activation of microglia, which initiates secondary processes that worsen the TBI outcome. The upregulation of cannabinoid type-2 receptors (CB2) when microglia become activated allows CB2-binding drugs to selectively target microglia. CB2 inverse agonists modulate activated microglia by shifting them away from the harmful pro-inflammatory M1 state toward the helpful reparative M2 state and thus can stem secondary injury cascades. We previously found that treatment with the CB2 inverse agonist SMM-189 after mild TBI in mice produced by focal cranial blast rescues visual deficits and the optic nerve axon loss that would otherwise result. We have further shown that raloxifene, which is Food and Drug Administration (FDA)-approved as an estrogen receptor modulator to treat osteoporosis, but also possesses CB2 inverse agonism, yields similar benefit in this TBI model through its modulation of microglia. As many different traumatic events produce TBI in humans, it is widely acknowledged that diverse animal models must be used in evaluating possible therapies. Here we examine the consequences of TBI created by blunt impact to the mouse head for visual function and associated pathologies and assess raloxifene benefit. We found that mice subjected to impact TBI exhibited decreases in contrast sensitivity and the B-wave of the electroretinogram, increases in light aversion and resting pupil diameter, and optic nerve axon loss, which were rescued by daily injection of raloxifene at 5 or 10 mg/ml for 2 weeks. Raloxifene treatment was associated with reduced M1 activation and/or enhanced M2 activation in retina, optic nerve, and optic tract after impact TBI. Our results suggest that the higher raloxifene dose, in particular, may be therapeutic for the optic nerve by enhancing the phagocytosis of axonal debris that would otherwise promote inflammation, thereby salvaging less damaged axons. Our current work, together with our prior studies, shows that microglial activation drives secondary injury processes after both impact and cranial blast TBI and raloxifene mitigates microglial activation and visual system injury in both cases. The results thus provide a strong basis for phase 2 human clinical trials evaluating raloxifene as a TBI therapy.

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TI Selective activation of cannabinoid receptor-2 reduces white matter

injury via PERK signaling in a rat model of traumatic brain injury

SO EXPERIMENTAL NEUROLOGY

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DE Traumatic brain injury; Cannabinoid receptor-2; White matter injury;

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ID MICROGLIAL ACTIVATION; MICROGLIA/MACROPHAGE POLARIZATION; INHIBITION;

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IMPAIRMENT

AB Background and purpose: Traumatic brain injury (TBI) destroys white matter, and this destruction is aggravated by secondary neuroinflammatory reactions. Although white matter injury (WMI) is strongly correlated with poor neurological function, understanding of white matter integrity maintenance is limited, and no available therapies can effectively protect white matter. One candidate approach that may fulfill this goal is cannabinoid receptor 2 (CB2) agonist treatment. Here, we confirmed that a selective CB2 agonist, JWH133, protected white matter after TBI. Methods: The motor evoked potentials (MEPs), open field test, and Morris water maze test were used to assess neurobehavioral outcomes. Brain tissue loss, WM damage, Endoplasmic reticulum stress (ER stress), microglia responses were evaluated after TBI. The functional integrity of WM was measured by diffusion tensor imaging (DTI) and transmission electron microscopy (TEM). Primary microglia and oligodendrocyte cocultures were used for additional mechanistic studies. Results: JWH133 increased myelin basic protein (MBP) and neurofilament heavy chain (NF200) levels and anatomic preservation of myelinated axons revealed by DTI and TEM. JWH133 also increased the numbers of oligodendrocyte precursor cells and mature oligodendrocytes. Furthermore, JWH133 drove microglial polarization toward the protective M2 phenotype and modulated the redistribution of microglia in the striatum. Further investigation of the underlying mechanism revealed that JWH133 downregulated phosphorylation of the protein kinase R (PKR)-like endoplasmic reticulum (ER) kinase (PERK) signaling pathway and its downstream signals eukaryotic translation initiation factor 2 alpha (eIF2 alpha), activating transcription factor 4 (ATF4) and Growth arrest and DNA damage-inducible protein (GADD34); this downregulation was followed by p-Protein kinase B(pAkt) upregulation. In primary cocultures of microglia and oligodendrocytes, JWH133 decreased phosphorylated PERK expression in microglia stimulated with tunicamycin and facilitated oligodendrocyte survival. These data reveal that JWH133 ultimately alleviates WMI and improves neurological behavior following TBI. However, these effects were prevented by SR144528, a selective CB2 antagonist. Conclusions: This work illustrates the PERK-mediated interaction between microglia and oligodendrocytes. In addition, the results are consistent with recent findings that microglial polarization switching accelerates WMI, highlighting a previously unexplored role for CB2 agonists. Thus, CB2 agonists are potential therapeutic agents for TBI and other neurological conditions involving white matter destruction.

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TI 17β-Estradiol Abrogates Oxidative Stress and Neuroinflammation after

Cortical Stab Wound Injury

SO ANTIOXIDANTS

LA English

DT Article

DE traumatic brain injury; 17 beta-estradiol; neuroprotection; oxidative

stress; neuroinflammation; astrocytosis; microglial polarization;

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ID TRAUMATIC BRAIN-INJURY; ACTIVATED PROTEIN-KINASE; MICROGLIA/MACROPHAGE

POLARIZATION DYNAMICS; NEUROTROPHIC FACTOR; MICROGLIAL ACTIVATION;

INFLAMMATORY RESPONSE; NLRP3 INFLAMMASOME; ESTROGEN; EXPRESSION; PATHWAY

AB Disruptions in brain energy metabolism, oxidative damage, and neuroinflammation are commonly seen in traumatic brain injury (TBI). Microglial activation is the hallmark of neuroinflammation. After brain injury, microglia also act as a double-edged sword with distinctive phenotypic changes. Therefore, therapeutic applications to potentiate microglia towards pro-inflammatory response following brain injury have become the focus of attention in recent years. Here, in the current study, we investigated the hypothesis that 17 beta-estradiol could rescue the mouse brain against apoptotic cell death and neurodegeneration by suppressing deleterious proinflammatory response probably by abrogating metabolic stress and oxidative damage after brain injury. Male C57BL/6N mice were used to establish a cortical stab wound injury (SWI) model. Immediately after brain injury, the mice were treated with 17 beta-estradiol (10 mg/kg, once every day via i.p. injection) for one week. Immunoblotting and immunohistochemical analysis was performed to examine the cortical and hippocampal brain regions. For the evaluation of reactive oxygen species (ROS), reduced glutathione (GSH), and oxidized glutathione (GSSG), we used specific kits. Our findings revealed that 17 beta-estradiol treatment significantly alleviated SWI-induced energy dyshomeostasis and oxidative stress by increasing the activity of phospho-AMPK (Thr172) and by regulating the expression of an antioxidant gene (Nrf2) and cytoprotective enzymes (HO-1 and GSH) to mitigate ROS. Importantly, 17 beta-estradiol treatment downregulated gliosis and proinflammatory markers (iNOS and CD64) while significantly augmenting an anti-inflammatory response as evidenced by the robust expression of TGF-beta and IGF-1 after brain injury. The treatment with 17 beta-estradiol also reduced inflammatory mediators (Tnf-alpha, IL-1 beta, and COX-2) in the injured mouse. Moreover, 17 beta-estradiol administration rescued p53-associated apoptotic cell death in the SWI model by regulating the expression of Bcl-2 family proteins (Bax and Bcl-2) and caspase-3 activation. Finally, SWI + 17 beta-estradiol-treated mice illustrated reduced brain lesion volume and enhanced neurotrophic effect and the expression of synaptic proteins. These findings suggest that 17 beta-estradiol is an effective therapy against the brain secondary injury-induced pathological cascade following trauma, although further studies may be conducted to explore the exact mechanisms.

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TI Differential Expression Patterns of TDP-43 in Single Moderate versus

Repetitive Mild Traumatic Brain Injury in Mice

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE mice; microglia; NF-kappa B; postsynaptic density protein 95; traumatic

brain injury; transactive response DNA-binding protein 43

ID FACTOR-KAPPA-B; AMYOTROPHIC-LATERAL-SCLEROSIS; PROTEIN EXPRESSION;

OXIDATIVE STRESS; MOUSE MODEL; HEAD-INJURY; BLOOD-FLOW; RAT MODEL;

CASPASE 3; A-BETA

AB Traumatic brain injury (TBI) is a disabling disorder and a major cause of death and disability in the world. Both single and repetitive traumas affect the brain acutely but can also lead to chronic neurodegenerative changes. Clinical studies have shown some dissimilarities in transactive response DNA binding protein 43 (TDP-43) expression patterns following single versus repetitive TBI. We explored the acute cortical post-traumatic changes of TDP-43 using the lateral fluid percussion injury (LFPI) model of single moderate TBI in adult male mice and investigated the association of TDP-43 with post-traumatic neuroinflammation and synaptic plasticity. In the ipsilateral cortices of animals following LFPI, we found changes in the cytoplasmic and nuclear levels of TDP-43 and the decreased expression of postsynaptic protein 95 within the first 3 d post-injury. Subacute pathological changes of TDP-43 in the hippocampi of animals following LFPI and in mice exposed to repetitive mild TBI (rmTBI) were studied. Changes in the hippocampal TDP-43 expression patterns at 14 d following different brain trauma procedures showed pathological alterations only after single moderate, but not following rmTBI. Hippocampal LFPI-induced TDP-43 pathology was not accompanied by the microglial reaction, contrary to the findings after rmTBI, suggesting that different types of brain trauma may cause diverse pathophysiological changes in the brain, specifically related to the TDP-43 protein as well as to the microglial reaction. Taken together, our findings may contribute to a better understanding of the pathophysiological events following brain trauma.

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TI Secondary thalamic neuroinflammation after focal cortical stroke and

traumatic injury mirrors corticothalamic functional connectivity

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LA English

DT Article; Early Access

DE astrocytes; microglia; neuroinflammation; nucleus reticularis thalami;

stroke; thalamus; traumatic brain injury

ID HIGHER-ORDER; RETICULAR NUCLEUS; RETROGRADE DEGENERATION; MICROGLIAL

ACTIVATION; COGNITIVE IMPAIRMENT; MEDIODORSAL THALAMUS; PREFRONTAL

CORTEX; BRAIN-INJURY; ORGANIZATION; NEURONS

AB While cortical injuries, such as traumatic brain injury (TBI) and neocortical stroke, acutely disrupt the neocortex, most of their consequent disabilities reflect secondary injuries that develop over time. Thalamic neuroinflammation has been proposed to be a biomarker of cortical injury and of the long-term cognitive and neurological deficits that follow. However, the extent to which thalamic neuroinflammation depends on the type of cortical injury or its location remains unknown. Using two mouse models of focal neocortical injury that do not directly damage subcortical structures-controlled cortical impact and photothrombotic ischemic stroke-we found that chronic neuroinflammation in the thalamic region mirrors the functional connections with the injured cortex, and that sensory corticothalamic regions may be more likely to sustain long-term damage than nonsensory circuits. Currently, heterogeneous clinical outcomes complicate treatment. Understanding how thalamic inflammation depends on the injury site can aid in predicting features of subsequent deficits and lead to more effective, customized therapies.

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TI Tailored Therapeutic Doses of Dexmedetomidine in Evolving

Neuroinflammation after Traumatic Brain Injury

SO NEUROCRITICAL CARE

LA English

DT Article; Early Access

DE Traumatic brain injury; Neuroinflammation; Inflammasome; NLRP3;

Microglia; T cells; Dexmedetomidine

ID ISCHEMIA-REPERFUSION INJURY; NF-KAPPA-B; NLRP3 INFLAMMASOME;

HEAD-INJURY; T-CELLS; NEUROPROTECTION; ACTIVATION; EXPRESSION; PATHWAY;

RECRUITMENT

AB Background Understanding the secondary damage mechanisms of traumatic brain injury (TBI) is essential for developing new therapeutic approaches. Neuroinflammation has a pivotal role in secondary brain injury after TBI. Activation of NLRP3 inflammasome complexes results in the secretion of proinflammatory mediators and, in addition, later in the response, microglial activation and migration of the peripheral immune cells into the injured brain are observed. Therefore, these components involved in the inflammatory process are becoming a new treatment target in TBI. Dexmedetomidine (Dex) is an effective drug, widely used over the past few years in neurocritical care units and during surgical operations for sedation and analgesia, and has anti-inflammatory effects, which are shown in in vivo studies. The aim of this original research is to discuss the anti-inflammatory effects of different Dex doses over time in TBI. Methods Brain injury was performed by using a weight-drop model. Half an hour after the trauma, intraperitoneal saline was injected into the control groups and 40 and 200 mu g/kg of Dex were given to the drug groups. Neurological evaluations were performed with the modified Neurological Severity Score before being killed. Then, the mice were killed on the first or the third day after TBI and histopathologic (hematoxylin-eosin) and immunofluorescent (Iba1, NLRP3, interleukin-1 beta, and CD3) findings of the brain tissues were examined. Nonparametric data were analyzed by using the Kruskal-Wallis test for multiple comparisons, and the Mann-Whitney U-test was done for comparing two groups. The results are presented as mean +/- standard error of mean. Results The results showed that low doses of Dex suppress NLRP3 and interleukin-1 beta in both terms. Additionally, high doses of Dex cause a remarkable decrease in the migration and motility of microglial cells and T cells in the late phase following TBI. Interestingly, the immune cells were influenced by only high-dose Dex in the late phase of TBI and it also improves neurologic outcome in the same period. Conclusions In the mice head trauma model, different doses of Dex attenuate neuroinflammation by suppressing distinct components of the neuroinflammatory process in a different timecourse that contributes to neurologic recovery. These results suggest that Dex may be an appropriate choice for sedation and analgesia in patients with TBI.

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TI Minocycline fails to treat chronic traumatic brain injury-induced

impulsivity and attention deficits

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article; Early Access

DE Controlled cortical impact; Executive function; Microglia; Five choice

serial reaction time task

ID MICROGLIAL ACTIVATION; UNITED-STATES; HEAD-INJURY; RATS;

NEUROINFLAMMATION; NEURODEGENERATION; INFLAMMATION; RECOVERY; MODERATE

AB Traumatic brain injury (TBI) impacts millions worldwide and can cause lasting psychiatric symptoms. Chronic neuroinflammation is a characteristic of post-injury pathology and is also associated with psychiatric conditions such as ADHD and bipolar disorder. Therefore, the current study sought to determine whether TBI-induced impulsivity and inattention could be treated using minocycline, an antibiotic with anti-inflammatory properties. Rats were trained on the five-choice serial reaction time task (5CSRT), a measure of motor impulsivity and attention. After behavior was stable on the 5CSRT, rats received either a bilateral frontal TBI or sham procedure. Minocycline was given at either an early (1 h post-injury) or chronic (9 weeks post-injury) timepoint. Minocycline was delivered every 12 h for 5 days (45 mg/kg, i.p.). Behavioral testing on the 5CSRT began again after one week of recovery and continued for 12 more weeks, then rats were transcardially perfused. Impulsivity and inattention were both substantially increased following TBI. Minocycline had no therapeutic effects at either the early or late time points. TBI rats had increased lesion volume, but minocycline did not attenuate lesion size. Additionally, microglia count measured by IBA-1(+) cells was only increased acutely after TBI, and minocycline did not differentially change the number of microglia in TBI rats. Despite this, minocycline had clear effects on the gut microbiome. Based on the results of this study, minocycline may have limited efficacy for post-injury psychiatric-like symptoms.

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TI The Novel Monoacylglycerol Lipase Inhibitor MJN110 Suppresses

Neuroinflammation, Normalizes Synaptic Composition and Improves

Behavioral Performance in the Repetitive Traumatic Brain Injury Mouse

Model

SO CELLS

LA English

DT Article

DE monoacylglycerol lipase inhibitor; 2-arachidonyl glycerol; glutamate

receptors; GABA receptors; microglia; neuronal cell death; hippocampus;

repetitive mTBI

ID AMPA RECEPTORS; NMDA RECEPTOR; FUNCTIONAL RECOVERY; EXPRESSION; SUBUNIT;

GABA; NEURODEGENERATION; BARRIER; NEURONS; MEMORY

AB Modulation of the endocannabinoid system has emerged as an effective approach for the treatment of many neurodegenerative and neuropsychological diseases. However, the underlying mechanisms are still uncertain. Using a repetitive mild traumatic brain injury (mTBI) mouse model, we found that there was an impairment in locomotor function and working memory within two weeks post-injury, and that treatment with MJN110, a novel inhibitor of the principal 2-arachidononyl glycerol (2-AG) hydrolytic enzyme monoacylglycerol lipase dose-dependently ameliorated those behavioral changes. Spatial learning and memory deficits examined by Morris water maze between three and four weeks post-TBI were also reversed in the drug treated animals. Administration of MJN110 selectively elevated the levels of 2-AG and reduced the production of arachidonic acid (AA) and prostaglandin E-2 (PGE(2)) in the TBI mouse brain. The increased production of proinflammatory cytokines, accumulation of astrocytes and microglia in the TBI mouse ipsilateral cerebral cortex and hippocampus were significantly reduced by MJN110 treatment. Neuronal cell death was also attenuated in the drug treated animals. MJN110 treatment normalized the expression of the NMDA receptor subunits NR2A and NR2B, the AMPA receptor subunits GluR1 and GluR2, and the GABA(A) receptor subunits alpha 1, beta 2,3 and gamma 2, which were all reduced at 1, 2 and 4 weeks post-injury. The reduced inflammatory response and restored glutamate and GABA receptor expression likely contribute to the improved motor function, learning and memory in the MJN110 treated animals. The therapeutic effects of MJN110 were partially mediated by activation of CB1 and CB2 cannabinoid receptors and were eliminated when it was co-administered with DO34, a novel inhibitor of the 2-AG biosynthetic enzymes. Our results suggest that augmentation of the endogenous levels of 2-AG can be therapeutically useful in the treatment of TBI by suppressing neuroinflammation and maintaining the balance between excitatory and inhibitory neurotransmission.

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TI Neuroinflammatory Cytokine Response, Neuronal Death, and Microglial

Proliferation in the Hippocampus of Rats During the Early Period After

Lateral Fluid Percussion-Induced Traumatic Injury of the Neocortex

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article; Early Access

DE Traumatic brain injury; Hippocampus; Neuroinflammation; Microglia;

Neurodegeneration; Distant damage; Corticosterone; IL1b; TNF; IL6

ID BRAIN-INJURY; VENTRAL HIPPOCAMPUS; OXIDATIVE STRESS; PROTEIN-LEVELS;

SEVERITY; MODEL

AB Time course of changes in neuroinflammatory processes in the dorsal and ventral hippocampus was studied during the early period after lateral fluid percussion-induced neocortical traumatic brain injury (TBI) in the ipsilateral and contralateral hemispheres. In the ipsilateral hippocampus, neuroinflammation (increase in expression of pro-inflammatory cytokines) was evident from day 1 after TBI and ceased by day 14, while in the contralateral hippocampus, it was mainly limited to the dorsal part on day 1. TBI induced an increase in hippocampal corticosterone level on day 3 bilaterally and an accumulation of Il1b on day 1 in the ipsilateral hippocampus. Activation of microglia was observed from day 7 in different hippocampal areas of both hemispheres. Neuronal cell loss was detected in the ipsilateral dentate gyrus on day 3 and extended to the contralateral hippocampus by day 7 after TBI. The data suggest that TBI results in distant hippocampal damage (delayed neurodegeneration in the dentate gyrus and microglia proliferation in both the ipsilateral and contralateral hippocampus), the time course of this damage being different from that of the neuroinflammatory response.

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TI Temporal patterns of microglial activation in white matter following

experimental mild traumatic brain injury: a systematic literature review

SO ACTA NEUROPATHOLOGICA COMMUNICATIONS

LA English

DT Review

DE Microglia; Activation; White matter; Timecourses; Mild traumatic brain

injury; Iba1; Corpus callosum

ID AXONAL INJURY; MOUSE MODEL; MICROGLIA/MACROPHAGE POLARIZATION; DIFFUSION

TENSOR; TIME-POINTS; CELLS; INFLAMMATION; MICE; INTERLEUKIN-1; DEFICITS

AB Mild traumatic brain injuries (mTBIs) are a prevalent form of injury that can result in persistent neurological impairments. Microglial activation has become increasingly recognized as a key process regulating the pathology of white matter in a wide range of brain injury and disease contexts. As white matter damage is known to be a major contributor to the impairments that follow mTBI, microglia have rightfully become a common target of investigation for the development of mTBI therapies and biomarkers. Recent work has demonstrated that the efficacy of microglial manipulation as a therapeutic intervention following injury or disease is highly time-sensitive, emphasizing the importance of advancing our understanding of the dynamics of post-mTBI microglial activation from onset to resolution. Current reporting of microglial activation in experimental studies of mTBI is non-standardized, which has limited our ability to identify concrete patterns of post-mTBI microglial activation over time. In this review, we examine preclinical studies of mTBI that report on microglial activation in white matter regions to summarize our current understanding of these patterns. Specifically, we summarize timecourses of post-mTBI microglial activation in white matter regions of the brain, identify factors that influence this activation, examine the temporal relationship between microglial activation and other post-mTBI assessments, and compare the relative sensitivities of various methods for detecting microglial activation. While the lack of replicated experimental conditions has limited the extent of conclusions that can confidently be drawn, we find that microglia are activated over a wide range of timecourses following mTBI and that microglial activation is a long-lasting outcome of mTBI that may resolve after most typical post-mTBI assessments, with the exception of those measuring oligodendrocyte lineage cell integrity. We identify several understudied parameters of post-mTBI microglial activation in white matter, such as the inclusion of female subjects. This review summarizes our current understanding of the progression of microglial activation in white matter structures following experimental mTBI and offers suggestions for important future research directions.

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TI Animal model of repeated low-level blast traumatic brain injury displays

acute and chronic neurobehavioral and neuropathological changes

SO EXPERIMENTAL NEUROLOGY

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DT Article; Early Access

DE Anxiety; Astrocytes; Blast traumatic brain injury; Behavioral changes;

Depression; Motor incoordination; Microglia; Neuroinflammation;

Oxidative stress; Occupational low level blast

ID DISORDER-RELATED TRAITS; MOUSE MODEL; RAT MODEL; EXPOSURE; SINGLE;

CONSEQUENCES; MECHANISMS; DEFICITS

AB Blast-induced neurotrauma (BINT) is not only a signature injury to soldiers in combat field and training facilities but may also a growing concern in civilian population due to recent increases in the use of improvised explosives by insurgent groups. Unlike moderate or severe BINT, repeated low-level blast (rLLB) is different in its etiology as well as pathology. Due to the constant use of heavy weaponry as part of combat readiness, rLLB usually occurs in service members undergoing training as part of combat readiness. rLLB does not display overt pathological symptoms; however, earlier studies report chronic neurocognitive changes such as altered mood, irritability, and aggressive behavior, all of which may be caused by subtle neuropathological manifestations. Current animal models of rLLB for investigation of neurobehavioral and neuropathological alterations have not been adequate and do not sufficiently represent rLLB conditions. Here, we developed a rat model of rLLB by applying controlled low-level blast pressures (<10 psi) repeated successively five times to mimic the pressures experienced by service members. Using this model, we assessed anxiety-like symptoms, motor coordination, and short-term memory as a function of time. We also examined levels of superoxide-producing enzyme NADPH oxidase, microglial activa-tion, and reactive astrocytosis as factors likely contributing to these neurobehavioral changes. Animals exposed to rLLB displayed acute and chronic anxiety-like symptoms, motor and short-term memory impairments. These changes were paralleled by increased microglial activation and reactive astrocytosis. Conversely, animals exposed to a single low-level blast did not display significant changes. Collectively, this study demonstrates that, unlike a single low-level blast, rLLB exerts a cumulative impact on different brain regions and produces chronic neuropathological changes in so doing, may be responsible for neurobehavioral alterations.

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AB Neuroinflammatory processes mediated by microglial activation and subsequent neuronal damage are the hallmarks of traumatic brain injury (TBI). As an inhibitor of the macrophage-inducible C-type lectin (Mincle)/spleen tyrosine kinase (Syk) signaling pathway, BAY61-3606 (BAY) has previously demonstrated anti-inflammatory effects on some pathological processes, such as acute kidney injury, by suppressing the inflammatory macrophage response. In the present study, the potential effects of BAY on microglial phenotype and neuroinflammation after TBI were investigated. BAY (3 mg/kg) was first administered into mice by intraperitoneal injection after TBI induction in vivo and microglia were also treated with BAY (2 mu M) in vitro. The levels of inflammatory factors in microglia were assessed using reverse transcription-quantitative PCR and ELISA. Cortical neuron, myelin sheath, astrocyte and cerebrovascular endothelial cell markers were detected using immunofluorescence. The levels of components of the Mincle/Syk/NF-kappa B signaling pathway [Mincle, phosphorylated (p)-Syk and NF-kappa B], in addition to proteins associated with inflammation (ASC, caspase-1, TNF-alpha, IL-1 beta and IL-6), apoptosis (Bax and Bim) and tight junctions (Claudin-5), were measured via western blotting and ELISA. Migration and chemotaxis of microglial cells were evaluated using Transwell and agarose spot assays. Neurological functions of the mice were determined in vivo using the modified neurological severity scoring system and a Morris water maze. The results of the present study revealed that the expression levels of proteins in the Mincle/Syk/NF-kappa B signaling pathway (including Mincle, p-Syk and p-NF-kappa B), inflammatory cytokines (TNF-alpha, IL-1 beta and IL-6), proteins involved in inflammation (ASC and caspase-1), apoptotic markers (Bax and Bim) and the tight junction protein Claudin-5 were significantly altered post-TBI. BAY treatment reversed these effects in both the cerebral cortex extract-induced cell model and the controlled cortical impact mouse model. BAY was also revealed to suppress activation of the microglial proinflammatory phenotype and microglial migration. In addition, BAY effectively attenuated TBI-induced neurovascular unit damage and neurological function deficits. Taken together, these findings provided evidence that BAY may inhibit the Mincle/Syk/NF-kappa B signaling pathway in microglia; this in turn could attenuate microglia-mediated neuroinflammation and improve neurological deficits following TBI.

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TI A Levee to the Flood: Pre-injury Neuroinflammation and Immune Stress

Influence Traumatic Brain Injury Outcome

SO FRONTIERS IN AGING NEUROSCIENCE

LA English

DT Review

DE traumatic brain injury; inflammation; microglia; mitochondria;

Alzheimer's disease

ID AMYLOID PRECURSOR PROTEIN; ALZHEIMERS-DISEASE; MICROGLIAL ACTIVATION;

A-BETA; OXIDATIVE STRESS; MITOCHONDRIAL FISSION; CELL-DEATH; TNF-ALPHA;

INFLAMMATORY RESPONSE; CLUSTERIN EXPRESSION

AB Increasing evidence demonstrates that aging influences the brain's response to traumatic brain injury (TBI), setting the stage for neurodegenerative pathology like Alzheimer's disease (AD). This topic is often dominated by discussions of post-injury aging and inflammation, which can diminish the consideration of those same factors before TBI. In fact, pre-TBI aging and inflammation may be just as critical in mediating outcomes. For example, elderly individuals suffer from the highest rates of TBI of all severities. Additionally, pre-injury immune challenges or stressors may alter pathology and outcome independent of age. The inflammatory response to TBI is malleable and influenced by previous, coincident, and subsequent immune insults. Therefore, pre-existing conditions that elicit or include an inflammatory response could substantially influence the brain's ability to respond to traumatic injury and ultimately affect chronic outcome. The purpose of this review is to detail how age-related cellular and molecular changes, as well as genetic risk variants for AD affect the neuroinflammatory response to TBI. First, we will review the sources and pathology of neuroinflammation following TBI. Then, we will highlight the significance of age-related, endogenous sources of inflammation, including changes in cytokine expression, reactive oxygen species processing, and mitochondrial function. Heightened focus is placed on the mitochondria as an integral link between inflammation and various genetic risk factors for AD. Together, this review will compile current clinical and experimental research to highlight how pre-existing inflammatory changes associated with infection and stress, aging, and genetic risk factors can alter response to TBI.

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TI Age-At-Injury Influences the Glial Response to Traumatic Brain Injury in

the Cortex of Male Juvenile Rats

SO FRONTIERS IN NEUROLOGY

LA English

DT Article

DE concussion; pediatric; juvenile; inflammation; microglia; astrocyte;

aging

ID MICROGLIAL ACTIVATION; FUNCTIONAL PLASTICITY; MULTILEVEL ANALYSIS;

EFFECT SIZE; R PACKAGE; INFLAMMATION; VULNERABILITY; REGRESSION;

MORPHOLOGY; INCREASES

AB Few translational studies have examined how age-at-injury affects the glial response to traumatic brain injury (TBI). We hypothesized that rats injured at post-natal day (PND) 17 would exhibit a greater glial response, that would persist into early adulthood, compared to rats injured at PND35. PND17 and PND35 rats (n = 75) received a mild to moderate midline fluid percussion injury or sham surgery. In three cortical regions [peri-injury, primary somatosensory barrel field (S1BF), perirhinal], we investigated the glial response relative to age-at-injury (PND17 or PND35), time post-injury (2 hours, 1 day, 7 days, 25 days, or 43 days), and post-natal age, such that rats injured at PND17 or PND35 were compared at the same post-natal-age (e.g., PND17 + 25D post-injury = PND42; PND35 + 7D post-injury = PND42). We measured lba1 positive microglia cells (area, perimeter) and quantified their activation status using skeletal analysis (branch length/cell, mean processes/cell, cell abundance). GFAP expression was examined using immunohistochemistry and pixel analysis. Data were analyzed using Bayesian multivariate multi-level models. Independent of age-at-injury, TBI activated microglia (shorter branches, fewer processes) in the S1BF and perirhinal cortex with more microglia in all regions compared to uninjured shams. TBI-induced microglial activation (shorter branches) was sustained in the S1BF into early adulthood (PND60). Overall, PND17 injured rats had more microglial activation in the perirhinal cortex than PND35 injured rats. Activation was not confounded by age-dependent cell size changes, and microglial cell body sizes were similar between PND17 and PND35 rats. There were no differences in astrocyte GFAP expression. Increased microglial activation in PND17 brain-injured rats suggests that TBI upregulates the glial response at discrete stages of development. Age-at-injury and aging with an injury are translationally important because experiencing a TBI at an early age may trigger an exaggerated glial response.

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TI Immune dynamics in the CNS and its barriers during homeostasis and

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SO IMMUNOLOGICAL REVIEWS

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DT Review; Early Access

DE Alzheimer's; brain; dura; glymphatics; infection; intravital;

lymphatics; meninges; microglia; monocytes; neuroimmunology;

neutrophils; sinuses; stroke; traumatic brain injury; two-photon

microscopy; vasculature; virus

ID TRAUMATIC BRAIN-INJURY; CD8(+) T-CELLS; CHOROID-PLEXUS; INTERSTITIAL

FLUID; VIRAL-INFECTIONS; MICROGLIA; SYSTEM; CLEARANCE; INFLAMMATION;

IMPAIRMENT

AB The central nervous system (CNS) has historically been viewed as an immunologically privileged site, but recent studies have uncovered a vast landscape of immune cells that reside primarily along its borders. While microglia are largely responsible for surveying the parenchyma, CNS barrier sites are inhabited by a plethora of different innate and adaptive immune cells that participate in everything from the defense against microbes to the maintenance of neural function. Static and dynamic imaging studies have revolutionized the field of neuroimmunology by providing detailed maps of CNS immune cells as well as information about how these cells move, organize, and interact during steady-state and inflammatory conditions. These studies have also redefined our understanding of neural-immune interactions at a cellular level and reshaped our conceptual view of immune privilege in this specialized compartment. This review will focus on insights gained using imaging techniques in the field of neuroimmunology, with an emphasis on anatomy and CNS immune dynamics during homeostasis, infectious diseases, injuries, and aging.

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TI Inhibition of Exosome Release Alleviates Cognitive Impairment After

Repetitive Mild Traumatic Brain Injury

SO FRONTIERS IN CELLULAR NEUROSCIENCE

LA English

DT Article

DE repetitive mild traumatic brain injury; chronic traumatic

encephalopathy; exosome; microglia; cytokine; neuropathological protein;

cognition; GW4869

ID OXIDATIVE STRESS; INFLAMMATION; NEUROPROTECTION; PATHOGENESIS;

SECRETION; MODEL

AB BackgroundRepetitive mild traumatic brain injury (rmTBI) is closely associated with chronic traumatic encephalopathy (CTE). Neuroinflammation and neuropathological protein accumulation are key links to CTE progression. Exosomes play important roles in neuroinflammation and neuropathological protein accumulation and spread. Here, we explored the role of brain-derived exosomes (BDEs) in mice with rmTBI and how the inhibition of BDE release contributes to neuroprotection. MethodsGW4869 was used to inhibit exosome release, and behavioural tests, PET/CT and western blotting were conducted to explore the impact of this inhibition from different perspectives. We further evaluated cytokine expression by Luminex and microglial activation by immunofluorescence in mice with rmTBI after exosome release inhibition. ResultsInhibition of BDE release reversed cognitive impairment in mice with rmTBI, enhanced glucose uptake and decreased neuropathological protein expression. Inhibition of BDE release also changed cytokine production trends and enhanced microglial proliferation. ConclusionIn this study, we found that BDEs are key factor in cognitive impairment in mice with rmTBI and that microglia are the main target of BDEs. Thus, inhibition of exosome release may be a new strategy for improving CTE prognoses.

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TI Protein Expression of the Microglial Marker Tmem119 Decreases in

Association With Morphological Changes and Location in a Mouse Model of

Traumatic Brain Injury

SO FRONTIERS IN CELLULAR NEUROSCIENCE

LA English

DT Article

DE microglia; macrophage; traumatic brain injury; Tmem119; complement C1q;

neuroinflammation

ID CENTRAL-NERVOUS-SYSTEM; CELLS; HEALTH; MACROPHAGES; INDUCTION

AB The activation of microglia and the infiltration of macrophages are hallmarks of neuroinflammation after acute brain injuries, including traumatic brain injury (TBI). The two myeloid populations share many features in the post-injury inflammatory response, thus, being antigenically indistinguishable. Recently Tmem119, a type I transmembrane protein specifically expressed by microglia under physiological conditions, was proposed as a tool to differentiate resident microglia from blood-borne macrophages, not expressing it. However, the validity of Tmem119 as a specific marker of resident microglia in the context of acute brain injury, where microglia are activated and macrophages are recruited, needs validation. Our purpose was to investigate Tmem119 expression and distribution in relation to the morphology of brain myeloid cells present in the injured area after TBI. Mice underwent sham surgery or TBI by controlled cortical impact (CCI). Brains from sham-operated, or TBI mice, were analyzed by in situ hybridization to identify the cells expressing Tmem119, and by Western blot and quantitative immunofluorescence to measure Tmem119 protein levels in the entire brain regions and single cells. The morphology of Iba1+ myeloid cells was analyzed at different times (4 and 7 days after TBI) and several distances from the contused edge in order to associate Tmem119 expression with morphological evolution of active microglia. In situ hybridization indicated an increased Tmem119 RNA along with increased microglial complement C1q activation in the contused area and surrounding regions. On the contrary, the biochemical evaluation showed a drop in Tmem119 protein levels in the same areas. The Tmem119 immunoreactivity decreased in Iba1+ myeloid cells found in the contused cortex at both time points, with the cells showing the hypertrophic ameboid morphology having no Tmem119 expression. The Tmem119 was present on ramifications of resident microglia and its presence was decreased as a consequence of microglial activation in cortical areas close to contusion. Based on the data, we conclude that the decrease of Tmem119 in reactive microglia may depend on the process of microglial activation, which involves the retracting of their branchings to acquire an ameboid shape. The Tmem119 immunoreactivity decreases in reactive microglia to similar levels than the blood-borne macrophages, thus, failing to discriminate the two myeloid populations after TBI.

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TI Hippocampal Activated Microglia May Contribute to Blood-Brain Barrier

Impairment and Cognitive Dysfunction in Post-Traumatic Stress

Disorder-Like Rats

SO JOURNAL OF MOLECULAR NEUROSCIENCE

LA English

DT Article; Early Access

DE Post-traumatic stress disorder; Single prolonged stress; Blood-brain

barrier; Cognition dysfunction

ID INTEGRITY

AB Post-traumatic stress disorder (PTSD)-associated cognitive dysfunction significantly disturbs patients' quality of life and will to live. However, its underlying mechanism is as yet unknown. Recent researches indicate that blood-brain barrier (BBB) breakdown is responsible for early cognitive dysfunction. Microglia might participate in remodeling of BBB-associated tight junction and regulating BBB integrity. Nevertheless, it is unclear whether microglia activation and BBB injury involve in PTSD-associated cognitive dysfunction. Hence, we established an animal model of PTSD, single prolonged stress (SPS), and investigated permeability changes in the hippocampus and further explored the effects of microglia on BBB remodeling. The Y maze was used to assess the changes of cognitive function. The sodium fluorescein (NaFlu) assay and western blotting analysis were employed to detect BBB integrity changes. Minocycline was administered to inhibit microglial activation. Immunofluorescence stains were used to assess the activation states in microglia. The results showed that SPS-exposed rats exhibited poorer cognitive performance, higher passage of NaFlu, and lower expression of tight junction proteins (occludin and claudin 5) in the hippocampus on the day after SPS, but no difference on the 7th day. Inhibition of microglial activation by minocycline attenuated poor cognitive performance and BBB impairment including the extravasation of NaFlu and protein levels of the tight junction. Taken together, the present study indicates that BBB impairment may underlie the shared pathological basis of PTSD and cognitive dysfunction. Microglial activation may involve in BBB remodeling at the early stage of SPS.

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TI P2X4 Inhibition reduces microglia inflammation and apoptosis by NLRP3

and improves nervous system defects in rat brain trauma model

SO JOURNAL OF CLINICAL NEUROSCIENCE

LA English

DT Article; Early Access

DE NLRP3; Inflammation; Apoptosis; Neuroprotection; Traumatic brain injury

ID CELL-DEATH; BCL-2; ACTIVATION; RECEPTORS; LEUKEMIA; INJURY; BAX

AB The purinergic receptor P2X4 is an adenosine triphosphate (ATP)-gated cation channel, which plays an essential role in regulating various biological activities in the organism. This study was designed to investigate the potential role and mechanism of P2X4 in the traumatic brain injury (TBI) rat model. Real-time PCR, Western blot, immunofluorescence, apoptosis, brain water content and neurological score analysis were evaluated. We found that the expression level of P2X4 surrounding the injured area of the brain in the TBI rat model increased significantly after 48 h. Following the P2X4 selective antagonist 5-BDBD treatment, the neurological damage after TBI was significantly improved and brain edema was reduced. The inhibition of P2X4 effectively reduced the inflammation and apoptosis of microglia, and NLRP3 may be involved in this process. Our results indicate that inhibition of P2X4 may be a potential therapeutic approach for TBI by reducing the occurrence of inflammation and apoptosis of microglia, alleviating brain edema, and improving neurological deficits.

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TI Microglia and Neuroinflammation: Crucial Pathological Mechanisms in

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DT Review

DE microglia; neuroinflammation; traumatic brain injury; neurodegeneration;

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ID CENTRAL-NERVOUS-SYSTEM; LIVER X RECEPTORS; NADPH OXIDASE 2;

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DAMAGE

AB Traumatic brain injury (TBI) is one of the most common diseases in the central nervous system (CNS) with high mortality and morbidity. Patients with TBI usually suffer many sequelae in the life time post injury, including neurodegenerative disorders such as Alzheimer's disease (AD) and Parkinson's disease (PD). However, the pathological mechanisms connecting these two processes have not yet been fully elucidated. It is important to further investigate the pathophysiological mechanisms underlying TBI and TBI-induced neurodegeneration, which will promote the development of precise treatment target for these notorious neurodegenerative consequences after TBI. A growing body of evidence shows that neuroinflammation is a pivotal pathological process underlying chronic neurodegeneration following TBI. Microglia, as the immune cells in the CNS, play crucial roles in neuroinflammation and many other CNS diseases. Of interest, microglial activation and functional alteration has been proposed as key mediators in the evolution of chronic neurodegenerative pathology following TBI. Here, we review the updated studies involving phenotypical and functional alterations of microglia in neurodegeneration after injury, survey key molecules regulating the activities and functional responses of microglia in TBI pathology, and explore their potential implications to chronic neurodegeneration after injury. The work will give us a comprehensive understanding of mechanisms driving TBI-related neurodegeneration and offer novel ideas of developing corresponding prevention and treatment strategies for this disease.

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TI Hydroxychloroquine attenuates neuroinflammation following traumatic

brain injury by regulating the TLR4/NF-κB signaling pathway

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Hydroxychloroquine; TBI; Neuroinflammation; Microglia activation;

TLR4/NF-kappa B

ID INHIBITION; AUTOPHAGY; ACID; MICE; INFLAMMATION; CHLOROQUINE;

DYSFUNCTION; DEFICIENT; MECHANISM; PROTEIN

AB Background: After traumatic brain injury (TBI), an acute, robust inflammatory cascade occurs that is characterized by the activation of resident cells such as microglia, the migration and recruitment of peripheral immune cells and the release of inflammatory mediators that induce secondary cell death and impede neurological recovery. In addition, neuroinflammation can alter blood-brain barrier (BBB) permeability. Controlling inflammatory responses is considered a promising therapeutic approach for TBI. Hydroxychloroquine (HCQ) has already been used clinically for decades, and it is still widely used to treat various autoimmune diseases. However, the effects of HCQ on inflammation and the potential mechanism after TBI remain to be defined. The aim of the current study was to elucidate whether HCQ could improve the neurological recovery of mice post-TBI by inhibiting the inflammatory response via the TLR4/NF-kappa B signaling pathway.

Methods: C57BL/6 mice were subjected to controlled cortical impact (CCI) and randomly divided into groups that received intraperitoneal HCQ or vehicle daily after TBI. TAK-242 (3.0 mg/kg), an exogenous TLR4 antagonist, was injected intraperitoneally 1 h before TBI. Behavioral assessments were performed on days 1 and 3 post-TBI, and the gene expression levels of inflammatory cytokines were analyzed by qRT-PCR. The presence of infiltrated immune cells was examined by flow cytometry and immunostaining. In addition, BBB permeability, tight junction expression and brain edema were investigated.

Results: HCQ administration significantly ameliorated TBI-induced neurological deficits. HCQ alleviated neuroinflammation, the activation and accumulation of microglia and immune cell infiltration in the brain, attenuated BBB disruption and brain edema, and upregulated tight junction expression. Combined administration of HCQ and TAK-242 did not enhance the neuroprotective effects of HCQ.

Conclusions: HCQ reduced proinflammatory cytokine expression, and the underlying mechanism may involve suppressing the TLR4/NF-kappa B signaling pathway, suggesting that HCQ is a potential therapeutic agent for TBI treatment.

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TI Phosphatidylethanolamine Deficiency and Triglyceride Overload in

Perilesional Cortex Contribute to Non-Goal-Directed Hyperactivity after

Traumatic Brain Injury in Mice

SO BIOMEDICINES

LA English

DT Article

DE traumatic brain injury; cortical impact; triglycerides; microglia;

phosphatidylethanolamines; hyperactivity

ID NEURONOPATHIC GAUCHER-DISEASE; ALPHA-SYNUCLEIN; ACCUMULATION;

INFLAMMATION; METABOLISM; INCREASES; AUTOPHAGY; GLUCOCEREBROSIDASE;

GLUCOSYLCERAMIDE; BIOSYNTHESIS

AB Traumatic brain injury (TBI) is often complicated by long-lasting disabilities, including headache, fatigue, insomnia, hyperactivity, and cognitive deficits. In a previous study in mice, we showed that persistent non-goal-directed hyperactivity is a characteristic post-TBI behavior that was associated with low levels of endocannabinoids in the perilesional cortex. We now analyzed lipidome patterns in the brain and plasma in TBI versus sham mice in association with key behavioral parameters and endocannabinoids. Lipidome profiles in the plasma and subcortical ipsilateral and contralateral brain were astonishingly equal in sham and TBI mice, but the ipsilateral perilesional cortex revealed a strong increase in neutral lipids represented by 30 species of triacylglycerols (TGs) of different chain lengths and saturation. The accumulation of TG was localized predominantly to perilesional border cells as revealed by Oil Red O staining. In addition, hexosylceramides (HexCer) and phosphatidylethanolamines (PE and ether-linked PE-O) were reduced. They are precursors of gangliosides and endocannabinoids, respectively. High TG, low HexCer, and low PE/PE-O showed a linear association with non-goal-directed nighttime hyperactivity but not with the loss of avoidance memory. The analyses suggest that TG overload and HexCer and PE deficiencies contributed to behavioral dimensions of post-TBI psychopathology.

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TI Progranulin deficiency promotes persistent neuroinflammation and causes

regional pathology in the hippocampus following traumatic brain injury

SO GLIA

LA English

DT Article; Early Access

DE astrocyte; hippocampus; microglia; neuroinflammation; progranulin;

traumatic brain injury

ID FRONTOTEMPORAL DEMENTIA; ACTIVATED MICROGLIA; SHORT-TERM; ASTROCYTES;

CELLS; MUTATIONS; PRECURSOR; NEURODEGENERATION; INFLAMMATION; PHENOTYPE

AB Traumatic brain injury (TBI) can be progressive and can lead to the development of a long-term complication termed chronic traumatic encephalopathy. The mechanisms underlying the progressive changes are still unknown; however, studies have suggested that microglia-mediated neuroinflammation in response to TBI may play a fundamental role. This study aimed to determine whether progranulin (PGRN), a major modulator of microglial activity, plays a role in the progressive damage following TBI. PGRN-deficient and wild-type mice were subjected to controlled cortical impact and were observed neuropathologically after 3 days, 7 days, and 5 months. Compared to sham and wild-type mice, the PGRN-deficient mice showed overall stronger microgliosis and astrocytosis. The astrocytosis involved broader areas than the microgliosis and was more prominent in the basal ganglia, hippocampus, and internal capsule in PGRN-deficient mice. Ongoing neuronal death was uniquely observed in the hippocampal CA3 region of PGRN-deficient mice at 5 months after TBI, accompanying the regional chronic microgliosis and astrocytosis involving the CA3 commissural pathway. In addition, there was M1 microglial polarization in the pericontusional area with activated TLR4/MyD88/NF-kappa B signaling; however, the hippocampus showed only mild M1 polarization 7 days after TBI. Lastly, Morris water maze tests showed PGRN-deficient mice had poorer spatial learning and memory 5 months after TBI than wild-type or sham mice. The data indicated the PGRN deficiency caused TBI progression by promoting persistent microgliosis with microglial polarization and astrocytosis, as well as regional pathology in the hippocampus. The study suggests that PGRN should be evaluated as a potential therapy for TBI.

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TI Time dependent analysis of rat microglial surface markers in traumatic

brain injury reveals dynamics of distinct cell subpopulations

SO SCIENTIFIC REPORTS

LA English

DT Article

ID MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS; ALZHEIMERS-DISEASE;

IDENTIFICATION; ACTIVATION; EXPRESSION; RECEPTORS; MECHANISM

AB Traumatic brain injury (TBI) results in a cascade of cellular responses, which produce neuroinflammation, partly due to the activation of microglia. Accurate identification of microglial populations is key to understanding therapeutic approaches that modify microglial responses to TBI and improve long-term outcome measures. Notably, previous studies often utilized an outdated convention to describe microglial phenotypes. We conducted a temporal analysis of the response to controlled cortical impact (CCI) in rat microglia between ipsilateral and contralateral hemispheres across seven time points, identified microglia through expression of activation markers including CD45, CD11b/c, and p2y12 receptor and evaluated their activation state using additional markers of CD32, CD86, RT1B, CD200R, and CD163. We identified unique sub-populations of microglial cells that express individual or combination of activation markers across time points. We further portrayed how the size of these sub-populations changes through time, corresponding to stages in TBI response. We described longitudinal changes in microglial population after CCI in two different locations using activation markers, showing clear separation into cellular sub-populations that feature different temporal patterns of markers after injury. These changes may aid in understanding the symptomatic progression following TBI and help define microglial subpopulations beyond the outdated M1/M2 paradigm.

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TI Partial Ablation of Astrocytes Exacerbates Cerebral Infiltration of

Monocytes and Neuronal Loss After Brain Stab Injury in Mice

SO CELLULAR AND MOLECULAR NEUROBIOLOGY

LA English

DT Article; Early Access

DE Traumatic brain injury; Stab injury; Astrocytes; Microglia;

Neuroinflammation; Monocytes

ID MICROGLIAL ACTIVATION; REACTIVE ASTROCYTES; INFLAMMATION;

NEURODEGENERATION; PROLIFERATION; HETEROGENEITY; DEGENERATION;

RECRUITMENT; RECEPTOR; SYSTEM

AB In traumatic brain injury (TBI), mechanical injury results in instantaneous tissue damages accompanied by subsequent pro-inflammatory cascades composed of microgliosis and astrogliosis. However, the interactive roles between microglia and astrocytes during the pathogenesis of TBI remain unclear and sometimes debatable. In this study, we used a forebrain stab injury mouse model to investigate the pathological role of reactive astrocytes in cellular and molecular changes of inflammatory response following TBI. In the ipsilateral hemisphere of stab-injured brain, monocyte infiltration and neuronal loss, as well as increased elevated astrogliosis, microglia activation and inflammatory cytokines were observed. To verify the role of reactive astrocytes in TBI, local and partial ablation of astrocytes was achieved by stereotactic injection of diphtheria toxin in the forebrain of Aldh1l1-CreER(T2)::Ai9::iDTR transgenic mice which expressed diphtheria toxin receptor (DTR) in astrocytes after tamoxifen induction. This strategy achieved about 20% of astrocytes reduction at the stab site as validated by immunofluorescence co-staining of GFAP with tdTomato-positive astrocytes. Interestingly, reduction of astrocytes showed increased microglia activation and monocyte infiltration, accompanied with increased severity in stab injury-induced neuronal loss when compared with DTR-/- mice, together with elevation of inflammatory chemokines such as CCL2, CCL5 and CXCL10 in astrogliosis-reduced mice. Collectively, our data verified the interactive role of astrocytes as an immune modulator in suppressing inflammatory responses in the injured brain.

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TI Functional and transcriptional profiling of microglial activation during

the chronic phase of TBI identifies an age-related driver of poor

outcome in old mice

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Traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; HISTONE H3 ACETYLATION; NEURONAL CELL-DEATH;

SPINAL-CORD-INJURY; MOUSE MODEL; NEUROPATHIC PAIN; TREHALOSE;

NEURODEGENERATION; AUTOPHAGY; INFLAMMATION

AB Elderly patients with traumatic brain injury (TBI) have greater mortality and poorer outcomes than younger individuals. The extent to which old age alters long-term recovery and chronic microglial activation after TBI is unknown, and evidence for therapeutic efficacy in aged mice is sorely lacking. The present study sought to identify potential inflammatory mechanisms underlying age-related outcomes late after TBI. Controlled cortical impact was used to induce moderate TBI in young and old male C57BL/6 mice. At 12 weeks post-injury, aged mice exhibited higher mortality, poorer functional outcomes, larger lesion volumes, and increased microglial activation. Transcriptomic analysis identified age- and TBI-specific gene changes consistent with a disease-associated microglial signature in the chronically injured brain, including those involved with complement, phagocytosis, and autophagy pathways. Dysregulation of phagocytic and autophagic function in microglia was accompanied by increased neuroinflammation in old mice. As proof-of-principle that these pathways have functional importance, we administered an autophagic enhancer, trehalose, in drinking water continuously for 8 weeks after TBI. Old mice treated with trehalose showed enhanced functional recovery and reduced microglial activation late after TBI compared to the sucrose control group. Our data indicate that microglia undergo chronic changes in autophagic regulation with both normal aging and TBI that are associated with poorer functional outcome. Enhancing autophagy may therefore be a promising clinical therapeutic strategy for TBI, especially in older patients.

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TI ACT001 attenuates microglia-mediated neuroinflammation after traumatic

brain injury via inhibiting AKT/NFκB/NLRP3 pathway

SO CELL COMMUNICATION AND SIGNALING

LA English

DT Article

DE ACT001; Traumatic brain injury; Microglia cells; Neuroinflammation; AKT;

NF kappa B; NLRP3 inflammasome

ID NF-KAPPA-B; SESQUITERPENE LACTONES; THERAPEUTIC TARGET; NLRP3

INFLAMMASOME; CELL-DEATH; ACTIVATION; EXPRESSION; PATHOPHYSIOLOGY;

MICHELIOLIDE; MODULATION

AB Background: Microglia-mediated neuroinflammatory response following traumatic brain injury (TBI) is considered as a vital secondary injury factor, which drives trauma-induced neurodegeneration and is lack of efficient treatment. ACT001, a sesquiterpene lactone derivative, is reportedly involved in alleviation of inflammatory response. However, little is known regarding its function in regulating innate immune response of central nervous system (CNS) after TBI. This study aimed to investigate the role and underlying mechanism of ACT001 in TBI.

Methods: Controlled cortical impact (CCI) models were used to establish model of TBI. Cresyl violet staining, evans blue extravasation, neurobehavioral function assessments, immunofluorescence and transmission electron microscopy were used to evaluate therapeutic effects of ACT001 in vivo. Microglial depletion was induced by administering mice with colony stimulating factor 1 receptor (CSF1R) inhibitor, PLX5622. Cell-cell interaction models were established as co-culture system to simulate TBI conditions in vitro. Cytotoxic effect of ACT001 on cell viability was assessed by cell counting kit-8 and activation of microglia cells were induced by Lipopolysaccharides (LPS). Pro-inflammatory cytokines expression was determined by Real-time PCR and nitric oxide production. Apoptotic cells were detected by TUNEL and flow cytometry assays. Tube formation was performed to evaluate cellular angiogenic ability. ELISA and western blot experiments were used to determine proteins expression. Pull-down assay was used to analyze proteins that bound ACT001.

Results: ACT001 relieved the extent of blood-brain barrier integrity damage and alleviated motor function deficits after TBI via reducing trauma-induced activation of microglia cells. Delayed depletion of microglia with PLX5622 hindered therapeutic effect of ACT001. Furthermore, ACT001 alleviated LPS-induced activation in mouse and rat primary microglia cells. Besides, ACT001 was effective in suppressing LPS-induced pro-inflammatory cytokines production in BV2 cells, resulting in reduction of neuronal apoptosis in HT22 cells and improvement of tube formation in bEnd.3 cells. Mechanism by which ACT001 functioned was related to AKT/NF kappa B/NLRP3 pathway. ACT001 restrained NF kappa B nuclear translocation in microglia cells through inhibiting AKT phosphorylation, resulting in decrease of NLRP3 inflammasome activation, and finally down-regulated microglial neuroinflammatory response.

Conclusions: Our study indicated that ACT001 played critical role in microglia-mediated neuroinflammatory response and might be a novel potential chemotherapeutic drug for TBI.

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TI Age-relevant <i>in vitro</i> models may lead to improved translational

research for traumatic brain injury

SO CURRENT OPINION IN BIOMEDICAL ENGINEERING

LA English

DT Article; Early Access

DE Traumatic brain injury; Astrocytes; Microglia; Oligodendrocytes;

Magnetic-activated cell sorting

ID MICROGLIA; PROMOTES

AB Traumatic brain injury (TBI) is a major health problem affecting both children and adults. Although TBI studies have been focused on neurons, glial cells play an important role in neuropathology following injury. As the consequences of TBI are age-dependent, it is essential that in vitro and in vivo models are fully representative of clinical outcomes. Traditionally, in vitro models that focused on TBI-induced glial cell dysfunction use primary cells isolated from neonatal rodents, or cell lines. These models are widely used to elucidate molecular pathways affected by the injury; however, they fail to account for age-related differences. As glial characteristics are known to change during maturation, it is important to explore new age-relevant in vitro models leading to improved translation research and advancements in therapeutic strategies for TBI.

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TI Sirtuin 1 alleviates microglia-induced inflammation by modulating the

PGC-1a/Nrf2 pathway after traumatic brain injury in male rats

SO BRAIN RESEARCH BULLETIN

LA English

DT Article; Early Access

DE SIRT1; Neuronal apoptosis; PGC-1a; Nrf2; Traumatic brain injury

ID NEUROINFLAMMATION; ACTIVATION

AB Microglial activation and the subsequent inflammatory response play important roles in the central nervous system after traumatic brain injury (TBI). Activation of the PGC-1 alpha pathway is responsible for microglial acti-vation after TBI. Our previous study demonstrated that SIRT1 alleviates neuroinflammation-induced apoptosis after TBI, and activation of the PGC-1 alpha/Nrf2 pathway extenuates TBI-induced neuronal apoptosis. However, no study has investigated whether SIRT1 can affect the PGC-1 alpha/Nrf2 pathway to induce microglial excitation and the subsequent neuroinflammatory response. Microglial activation and the levels of pro-inflammatory factors, namely, tumor necrosis factor (TNF-alpha), interleukin-1 beta (IL-1 beta), and interleukin-6 (IL-6) were assessed to evaluate the neuroinflammatory response after TBI. To examine the effects of SIRT1, immunohistochemical staining and western blot analysis were used to observe the nuclear translocation and secretion of PGC-1 alpha, as well as the activation of the PGC-1 alpha/Nrf2 pathway. Treatment with the SIRT1 inhibitor sirtinol promoted microglial acti-vation and pro-inflammatory factor expression (TNF-alpha, IL-6, and IL-1 beta) and inhibited PGC-1 alpha and Nrf2 nuclear translocation and secretion after TBI, while treatment with the SIRT1 activator A3 had the opposite effects. The results of this study suggest that microglial activation, the subsequent neuroinflammatory response, and the PGC-1 alpha/Nrf2 pathway play essential roles in secondary injury after TBI. These results indicate that SIRT1 protects neurons after TBI by inhibiting microglial activation and the subsequent inflammatory response, possibly by activating the PGC-1 alpha/Nrf2 pathway.

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TC 5

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PU PERGAMON-ELSEVIER SCIENCE LTD

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PT J

AU Arora, P

Singh, K

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AF Arora, Palkin

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TI Temporal profile of serum metabolites and inflammation following closed

head injury in rats is associated with HPA axis hyperactivity

SO METABOLOMICS

LA English

DT Article

DE Traumatic brain injury; Serum metabolomics; Microglia; Corticosterone;

Inflammation

ID TRAUMATIC BRAIN-INJURY; BRANCHED-CHAIN AMINO; NEUROENDOCRINE;

INTERLEUKIN-10; ACTIVATION; EXPRESSION; MICROGLIA; RESPONSES; OUTCOMES

AB Introduction Closed head injury (CHI) causes neurological disability along with systemic alterations that can activate neuroendocrine response through hypothalamic-pituitary-adrenal (HPA) axis activation. A dysregulated HPA axis function can lead to relocation of energy substrates and alteration in metabolic pathways and inflammation at the systemic level.

Objectives Assessment of time-dependent changes in serum metabolites and inflammation after both mild and moderate CHI. Along with this, serum corticosterone levels and hypothalamic microglial response were observed.

Methods Rats underwent mild and moderate weight-drop injury and their serum and hypothalamus were assessed at acute, sub-acute and chronic timepoints. Changes in serum metabolomics were determined using high resolution NMR spectroscopy. Serum inflammatory cytokine, corticosterone levels and hypothalamic microglia were assessed at all timepoints.

Results Metabolites including lactate, choline and branched chain amino acids were found as the classifiers that helped distinguish between control and injured rats during acute, sub-acute and chronic timepoints. While, increased alpha aglucose: beta glucose and TMAO: choline ratios after acute and sub-acute timepoints of mild injury differentiated from moderate injured rats. The injured rats also showed distinct inflammatory profile where IL-1 beta and TNF-alpha levels were upregulated in moderate injured rats while IL-10 levels were downregulated in mild injured rats. Furthermore, injury specific alterations in serum metabolic and immunologic profile were found to be associated with hyperactive HPA axis, with consistent increase in serum corticosterone concentration post injury. The hypothalamic microglia showed a characteristic activated de-ramified cellular morphology in both mild and moderate injured rats.

Conclusion The study suggests that HPA axis hyperactivity along with hypothalamic microglial activation led to temporal changes in the systemic metabolism and inflammation. These time dependent changes in the metabolite profile of rats can further strengthen the knowledge of diagnostic markers and help distinguish injury related outcomes after TBI.

[GRAPHICS]

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U2 3

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J9 METABOLOMICS

JI Metabolomics

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TI Ketogenic Diet Modulates Neuroinflammation via Metabolites from

<i>Lactobacillus reuteri</i> After Repetitive Mild Traumatic Brain

Injury in Adolescent Mice

SO CELLULAR AND MOLECULAR NEUROBIOLOGY

LA English

DT Article; Early Access

DE Microglia; Astrogliosis; Aryl hydrocarbon receptor; Toll-like receptor;

beta-Hydroxybutyrate

ID ARYL-HYDROCARBON RECEPTOR; GUT MICROBIOTA; TRYPTOPHAN-METABOLISM;

SPATIAL WORKING; MOUSE MODEL; INFLAMMATION; ACTIVATION; MAZE

AB Repetitive mild traumatic brain injury (rmTBI) is associated with a range of neural changes which is characterized by axonal injury and neuroinflammation. Ketogenic diet (KD) is regarded as a potential therapy for facilitating recovery after moderate-severe traumatic brain injury (TBI). However, its effect on rmTBI has not been fully studied. In this study, we evaluated the anti-neuroinflammation effects of KD after rmTBI in adolescent mice and explored the potential mechanisms. Experimentally, specific pathogen-free (SPF) adolescent male C57BL/6 mice received a sham surgery or repetitive mild controlled cortical impacts consecutively for 7 days. The uninjured mice received the standard diet, and the mice with rmTBI were fed either the standard diet or KD for 7 days. One week later, all mice were subjected to behavioral tests and experimental analysis. Results suggest that KD significantly increased blood beta-hydroxybutyrate (beta-HB) levels and improved neurological function. KD also reduced white matter damage, microgliosis, and astrogliosis induced by rmTBI. Aryl hydrocarbon receptor (AHR) signaling pathway, which was mediated by indole-3-acetic acid (3-IAA) from Lactobacillus reuteri (L. reuteri) in gut and activated in microglia and astrocytes after rmTBI, was inhibited by KD. The expression level of the toll-like receptor 4 (TLR4)/myeloid differentiation primary response 88 (MyD88) in inflammatory cells, which mediates the NF-kappa B pathway, was also attenuated by KD. Taken together, our results indicated that KD can promote recovery following rmTBI in adolescent mice. KD may modulate neuroinflammation by altering L. reuteri in gut and its metabolites. The inhibition of indole/AHR pathway and the downregulation of TLR4/MyD88 may play a role in the beneficial effect of KD against neuroinflammation in rmTBI mice.

[GRAPHICS]

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TI Fecal Microbiota Transplantation Derived from Alzheimer's Disease Mice

Worsens Brain Trauma Outcomes in Wild-Type Controls

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE microbiome; traumatic brain injury; Alzheimer's disease; fecal

microbiota transplant; neuroinflammation; microglia; astrocytes;

dysbiosis; Muribaculum; Lactobacillus johnsonii

ID AMYLOID PROTEIN DEPOSITION; GUT MICROBIOTA; HEAD-INJURY; IMMUNE

ACTIVATION; MOUSE MODEL; NEUROINFLAMMATION; DYSBIOSIS; DEMENTIA; RISK;

PATHOGENESIS

AB Traumatic brain injury (TBI) causes neuroinflammation and neurodegeneration, both of which increase the risk and accelerate the progression of Alzheimer's disease (AD). The gut microbiome is an essential modulator of the immune system, impacting the brain. AD has been related with reduced diversity and alterations in the community composition of the gut microbiota. This study aimed to determine whether the gut microbiota from AD mice exacerbates neurological deficits after TBI in control mice. We prepared fecal microbiota transplants from 18 to 24 month old 3xTg-AD (FMT-AD) and from healthy control (FMT-young) mice. FMTs were administered orally to young control C57BL/6 (wild-type, WT) mice after they underwent controlled cortical impact (CCI) injury, as a model of TBI. Then, we characterized the microbiota composition of the fecal samples by full-length 16S rRNA gene sequencing analysis. We collected the blood, brain, and gut tissues for protein and immunohistochemical analysis. Our results showed that FMT-AD administration stimulates a higher relative abundance of the genus Muribaculum and a decrease in Lactobacillus johnsonii compared to FMT-young in WT mice. Furthermore, WT mice exhibited larger lesion, increased activated microglia/macrophages, and reduced motor recovery after FMT-AD compared to FMT-young one day after TBI. In summary, we observed gut microbiota from AD mice to have a detrimental effect and aggravate the neuroinflammatory response and neurological outcomes after TBI in young WT mice.

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TI Activation of Sigma-1 Receptor Alleviates ER-Associated Cell Death and

Microglia Activation in Traumatically Injured Mice

SO JOURNAL OF CLINICAL MEDICINE

LA English

DT Article

DE traumatic brain injury; endoplasmic reticulum stress; apoptosis;

pyroptosis; microglia activation; cerebrovascular function; sigma-1

receptor; PRE-084; BD-1047

ID MOUSE MODEL; BRAIN-INJURY; STRESS; AGONIST; NEUROINFLAMMATION;

MACROPHAGE; PYROPTOSIS; APOPTOSIS; IMMUNITY; DEFICITS

AB Background: Endoplasmic reticulum (ER) stress and unfolded protein response (UPR) is associated with neuroinflammation and subsequent cell death following traumatic brain injury (TBI). The sigma-1 receptor (Sig-1R) acts as a dynamic pluripotent modulator of fundamental cellular processes at the mitochondria-associated membranes (MAMs). The activation of Sig-1R is neuroprotective in a variety of central nervous system diseases, but its impact on ER stress induced by traumatic brain injury is not known. This study investigated the role of Sig-1R in regulating the ER stress-mediated microglial activation and programmed cell death (apoptosis and pyroptosis) induced by TBI. Methods: Ten human brain tissues were obtained from The Tianjin Medical University General Hospital. Four normal brain tissues were obtained from patients who underwent surgery for cerebral vascular malformation, through which peripheral brain tissues were isolated. Six severe TBI tissues were from patients with brain injury caused by accidents. None of the patients had any other known neurological disorders. Mice with Sig-1R deletion using CRISPR technology were subjected to controlled cortical impact-induced injury. In parallel, wild type C57BL/6J mice were analyzed for outcomes after they were exposed to TBI and received the Sig-1R agonist PRE-084 (10 mg/kg daily for three days) either alone or in combination with the Sig-1R antagonist BD-1047 (10 mg/kg). Results: The expression of Sig-1R and the 78 kDa glucose-regulated protein, a known UPR marker, were significantly elevated in the injured cerebral tissues from TBI patients and mice subjected to TBI. PRE-084 improved neurological function, restored the cerebral cortical perfusion, and ameliorated and brain edema in C57BL/6J mice subjected to TBI by reducing endoplasmic reticulum stress-mediated apoptosis, pyroptosis, and microglia activation. The effect of PRE-084 was abolished in mice receiving Sig-1R antagonist BD-1047. Conclusions: ER stress and UPR were upregulated in TBI patients and mice subjected to TBI. Sig-1R activation by the exogenous activator PRE-084 attenuated microglial cells activation, reduced ER stress-associated programmed cell death, and restored cerebrovascular and neurological function in TBI mice.

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TI A Calixarene Assembly Strategy of Combined Anti-Neuroinflammation and

Drug Delivery Functions for Traumatic Brain Injury Therapy

SO MOLECULES

LA English

DT Article

DE calixarenes; self-assembly; neuroinflammation; microglia; traumatic

brain injury

ID MECHANISMS; MODEL

AB Excessive inflammatory reaction aggravates brain injury and hinders the recovery of neural function in nervous system diseases. Microglia, as the major players of neuroinflammation, control the progress of the disease. There is an urgent need for effective non-invasive therapy to treat neuroinflammation mediated by microglia. However, the lack of specificity of anti-inflammatory agents and insufficient drug dose penetrating into the brain lesion area are the main problems. Here, we evaluated a series of calixarenes and found that among them the self-assembling architecture of amphiphilic sulfonatocalix[8]arene (SC8A12C) had the most potent ability to suppress neuroinflammation in vitro and in vivo. Moreover, SC8A12C assemblies were internalized into microglia through macropinocytosis. In addition, after applying the SC8A12C assemblies to the exposed brain tissue, we observed that SC8A12C assemblies penetrated into the brain parenchyma and eliminated the inflammatory factor storm, thereby restoring neurobiological functions in a mouse model of traumatic brain injury.

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NR 50

TC 4

Z9 4

U1 5

U2 32

PU MDPI

PI BASEL

PA ST ALBAN-ANLAGE 66, CH-4052 BASEL, SWITZERLAND

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J9 MOLECULES

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AU Li, XL

Wang, B

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AF Li, Xiang-Long

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Chen, Li-Gang

You, Jian

TI HOXA11-AS aggravates microglia-induced neuroinflammation after traumatic

brain injury

SO NEURAL REGENERATION RESEARCH

LA English

DT Article

DE astrocyte; competitive endogenous RNA; HOXA11-AS; microglia; midkine;

miR-124-3p; neuroinflammation; traumatic brain injury

ID LONG NONCODING RNA; HUMAN CANCER; INFLAMMATION; CONTRIBUTES; MIDKINE;

PROLIFERATION; DYSFUNCTION; PROGRESSION; REPAIR; GLIOMA

AB Long noncoding RNAs (lncRNAs) participate in many pathophysiological processes after traumatic brain injury by mediating neuroinflammation and apoptosis. Homeobox A11 antisense RNA (HOXA11-AS) is a member of the lncRNA family that has been reported to participate in many inflammatory reactions; however, its role in traumatic brain injury remains unclear. In this study, we established rat models of traumatic brain injury using a weight-drop hitting device and injected LV-HOXA11-AS into the right lateral ventricle 2 weeks before modeling. The results revealed that overexpression of HOXA11-AS aggravated neurological deficits in traumatic brain injury rats, increased brain edema and apoptosis, promoted the secretion of proinflammatory factors interleukin-1 beta, interleukin-6, and tumor necrosis factor alpha, and promoted the activation of astrocytes and microglia. Microglia were treated with 100 ng/mL lipopolysaccharide for 24 hours to establish in vitro cell models, and then transfected with pcDNA-HOXA11-AS, miR-124-3p mimic, or sh-MDK. The results revealed that HOXA11-AS inhibited miR-124-3p expression and boosted MDK expression and TLR4-nuclear factor-kappa B pathway activation. Furthermore, lipopolysaccharide enhanced potent microglia-induced inflammatory responses in astrocytes. Forced overexpression of miR-124-3p or downregulating MDK repressed microglial activation and the inflammatory response of astrocytes. However, the miR-124-3p-mediated anti-inflammatory effects were reversed by HOXA11-AS. These findings suggest that HOXA11-AS can aggravate neuroinflammation after traumatic brain injury by modulating the miR-124-3p-MDK axis.

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SC Cell Biology; Neurosciences & Neurology

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AU Revi, N

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AF Revi, Neeraja

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Rengan, Aravind Kumar

TI A study on the role of eugenol encapsulated liposomes in facilitating

neuron-microglia mediated wound recovery

SO MATERIALIA

LA English

DT Article; Early Access

DE Neurons; Microglia; Traumatic brain injury; Neuroinflammation; Eugenol

ID TRAUMATIC BRAIN-INJURY; CLINICAL-TRIALS

AB Activated microglia helps in resolving injury and supports wound healing. However, prolonged activation of microglia can lead to the aggravation of wound. Objective: The present study investigates neuronal injury-induced inflammatory response in microglia and how it is influenced by the treatment with an anti-inflammatory formulation, Eugenol encapsulated liposomes. Method: We introduced injury in an in vitro model of neuronal and microglia origin cell lines using scratch assay and evaluated wound closure rates with respect to treatment. On similar lines, both cell lines were treated in a combinatorial model using Eugenol encapsulated liposomes and investigated the effect of secreted factors in Conditioned Media (CM) on Reactive Oxygen Species (ROS) production, cell viability etc. We also evaluated the polarization properties of microglia in case of an injury by evaluating the amount of nitrite release. Further, in a co-culture model, we introduced neuronal injury in the vicinity of microglia. We then evaluated the inflammatory response of microglia after incubating the injured cells with the formulation for an hour. Results: Individual wounds, CM and co-culture models of neuronal injury treated with Eugenol encapsulated liposomes indicated a decrease in the production of ROS and pro-inflammatory microglia, with the latter displaying an enhanced difference (50-75% with respect to control). The markers produced by microglia cells with respect to an inflammatory condition followed by treatment with Eugenol encapsulated liposomes were investigated using ELISA and Western blotting. There was a reduction in the amount of pro-inflammatory markers TNF-alpha, IL-1 beta and an increase in the amount of anti-inflammatory markers IL-10 and IL-4. In control and treatment groups, cardiac rhythm was found to be in the basal levels (100-150 beats per minute). Viability of the embryos were also unaffected due to the treatment in individual group of 20 embryos each. Further, uptake of the formulation in embryos 2,3 & 4 days post fertilization (dpf) were analyzed. Co-encapsulating tracker dye, Nile red within the formulation indicated encapsulated tracker dye crossing organ barriers with respect to free dye. Conclusion: Neuronal injury causes microglial activation. Prolonged microglial activation hinders wound closure and can lead to secondary trauma. The present study was conducted using an in vitro scratch assay wound model. Treating the site of injury with an anti-inflammatory formulation, Eugenol loaded liposomes, converts pro-inflammatory microglia to an anti-inflammatory state and increases the rate of wound closure.

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PT J

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TI Comprehensive RNA Expression Analysis Revealed Biological Functions of

Key Gene Sets and Identified Disease-Associated Cell Types Involved in

Rat Traumatic Brain Injury

SO JOURNAL OF CLINICAL MEDICINE

LA English

DT Article

DE traumatic brain injury; WGCNA; inflammatory response; microglia;

perivascular macrophages

ID PERIVASCULAR MACROPHAGES; CEREBRAL-CORTEX; INFLAMMATION; MICROGLIA;

NEUROINFLAMMATION; NEUROPROTECTION; MECHANISMS; MONOCYTES; DEMENTIA;

MODELS

AB Traumatic brain injury (TBI) is a worldwide public health concern without major therapeutic breakthroughs over the past decades. Developing effective treatment options and improving the prognosis of TBI depends on a better understanding of the mechanisms underlying TBI. This study performed a comprehensive analysis of 15 RNA expression datasets of rat TBIs from the GEO database. By integrating the results from the various analyses, this study investigated the biological processes, pathways, and cell types associated with TBI and explored the activity of these cells during various TBI phases. The results showed the response to cytokine, inflammatory response, bacteria-associated response, metabolic and biosynthetic processes, and pathways of neurodegeneration to be involved in the pathogenesis of TBI. The cellular abundance of microglia, perivascular macrophages (PM), and neurons were found to differ after TBI and at different times postinjury. In conclusion, immune- and inflammation-related pathways, as well as pathways of neurodegeneration, are closely related to TBI. Microglia, PM, and neurons are thought to play roles in TBI with different activities that vary by phase of TBI.

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TI Fluvoxamine Confers Neuroprotection via Inhibiting Infiltration of

Peripheral Leukocytes and M1 Polarization of Microglia/Macrophages in a

Mouse Model of Traumatic Brain Injury

SO JOURNAL OF NEUROTRAUMA

LA English

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DE fluvoxamine; immune cells infiltration; microglial; macrophage

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ID NEUROINFLAMMATION; ACTIVATION

AB Neuroinflammation is an important mediator of secondary injury pathogenesis that exerts dual beneficial and detrimental effects on pathophysiology of the central nervous system (CNS) after traumatic brain injury (TBI). Fluvoxamine is a serotonin selective reuptake inhibitor (SSRI) and has been reported to have the anti-inflammatory properties. However, the mechanisms and therapeutic effects of fluvoxamine in neuroinflammation after TBI have not be defined. In this study, we showed that fluvoxamine inhibited peripheral immune cell infiltration and glia activation at 3 days in mice subjected to TBI. Fluvoxamine treatment promoted microglial/macrophage phenotypic transformation from pro-inflammatory M1-phenotype to anti-inflammatory M2-phenotype in in vivo and in vitro experiments. In addition, fluvoxamine treatment attenuated neuronal apoptosis, blood-brain barrier (BBB) disruption, cerebrovascular damage, and post-traumatic edema formation, thereby improving neurological function of mice subjected to TBI. These findings support the clinical evaluation of fluvoxamine as a neuroprotective therapy for TBI.

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TI Parthenolide ameliorates neurological deficits and neuroinflammation in

mice with traumatic brain injury by suppressing STAT3/NF-κB and

inflammasome activation

SO INTERNATIONAL IMMUNOPHARMACOLOGY

LA English

DT Article

DE Parthenolide; Traumatic brain injury; Inflammation; Microglial

activation; STAT3

ID MICROGLIA; HYPOXIA; PATHWAY; APOPTOSIS; GROWTH; NLRC4; NLRP3; STAT3

AB Background: Traumatic brain injury (TBI) triggers a set of complex inflammation that results in secondary injury. Parthenolide (PTN) is a sesquiterpene lactone extracted from the herb Tanacetum parthenium (Feverfew) and has potent anti-inflammatory, anti-apoptosis and anti-oxidative stress effects in the central nervous system (CNS)related diseases. This study focuses on investigating the potential neuroprotective effect of PTN on TBI and the related mechanism.

Methods: Bv2 microglia, primary microglia were stimulated by LPS, and HT22 neuron cells were stimulated by OGD/R, and they were treated with different doses of PTN. The expression profiles of pro-inflammatory cytokines, proteins, oxidative stress mediators, STAT3/NF-kappa B pathway, inflammasomes were detected. Forty male/female C57BL/6 mice were randomly divided into the sham, PTN, TBI, and TBI + PTN groups (10 mice per group). A mouse TBI model was set up with a controlled cortical impact (CCI) device. The modified nerve severity score (mNSS) was implemented to check short-term neurological impairment in mice, and the mice's memory and learning were assessed by the Morris water maze test. The water content in the mice's brains was measured by the dry-wet method. Hematoxylin-eosin (H&E) staining, Nissl staining and terminal deoxynucleotidyl transferase-mediated dUTP-biotin nick end labeling (TUNEL) assay were applied for neuronal apoptosis.

Results: PTN dramatically alleviated LPS-induced inflammation in microglia, and OGD-mediated neuronal apoptosis and oxidative stress. In addition, PTN repressed LPS- or OGD-modulated STAT3/NF-kappa B and NLR family pyrin domain containing 1 (NLRP1), NLRP3, NLR family CARD domain containing 4 (NLRC4) inflammasomes activation. Administering the STAT3 inhibitor Stattic or NF-kappa B inhibitor Bay 11-7082 attenuated PTN-mediated effects. In vivo, PTN treatment relieved neural function deficits, brain edema and neuron apoptosis and improved the memory and learning function of TBI mice. Additionally, PTN impeded microglial activation and reduced the production of pro-inflammatory cytokines in brain lesions of TBI mice. Furthermore, PTN hindered STAT3/NF-kappa B and inflammasome activation.

Conclusion: PTN can curb microglial activation and neuron apoptosis by dampening the STAT3/NF-kappa B pathway, thus exerting neuroprotective effects in TBI mice.

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Z9 9

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TI A Focal Impact Model of Traumatic Brain Injury in <i>Xenopus</i>

Tadpoles Reveals Behavioral Alterations, Neuroinflammation, and an

Astroglial Response

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE TBI; brain injury; inflammation; astrocyte; microglia; Xenopus; tadpole

ID LAEVIS; EDEMA; INFLAMMATION; NEUROGENESIS; AUTISM; RISK

AB Traumatic Brain Injury (TBI) is a global driver of disability, and we currently lack effective therapies to promote neural repair and recovery. TBI is characterized by an initial insult, followed by a secondary injury cascade, including inflammation, excitotoxicity, and glial cellular response. This cascade incorporates molecular mechanisms that represent potential targets of therapeutic intervention. In this study, we investigate the response to focal impact injury to the optic tectum of Xenopus laevis tadpoles. This injury disrupts the blood-brain barrier, causing edema, and produces deficits in visually-driven behaviors which are resolved within one week. Within 3 h, injured brains show a dramatic transcriptional activation of inflammatory cytokines, upregulation of genes associated with inflammation, and recruitment of microglia to the injury site and surrounding tissue. Shortly afterward, astrocytes undergo morphological alterations and accumulate near the injury site, and these changes persist for at least 48 h following injury. Genes associated with astrocyte reactivity and neuroprotective functions also show elevated levels of expression following injury. Since our results demonstrate that the response to focal impact injury in Xenopus resembles the cellular alterations observed in rodents and other mammalian models, the Xenopus tadpole offers a new, scalable vertebrate model for TBI.

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PI BASEL

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TI Amide Proton Transfer-Weighted Magnetic Resonance Imaging for Detecting

Severity and Predicting Outcome after Traumatic Brain Injury in Rats

SO NEUROTRAUMA REPORTS

LA English

DT Article

DE amide proton transfer-weighted imaging; Barnes maze; microglia; MRI;

neuroinflammation; traumatic brain injury

ID TRANSFER MRI; NEUROINFLAMMATION; HEMORRHAGE; PROTEINS

AB After traumatic brain injury (TBI), early assessment of secondary injury severity is critically important for estimating prognosis and treatment stratification. Currently, secondary injury severity is difficult to estimate. The objective of this study was to investigate the capacity of non-invasive amide proton transfer-weighted (APTw) magnetic resonance imaging (MRI) techniques to assess TBI injury in different brain regions and predict long-term neurobehavior outcomes. Fifty-five male and female rats were subjected to a controlled cortical impact with one of three different impactor depths to produce different degrees of TBI. Multi-parameter MRI data were acquired on a 4.7-Tesla scanner at 1h, 1 day, and 3 days. Immunofluorescence staining was used to detect activated microglia at 3 days, and neurobehavioral tests were performed to assess long-term outcomes after 28 days. The APTw signal in the injury core at 1 day correlated with deficits in sensorimotor function, the sucrose preference test (a test for anhedonia), and spatial memory function on the Barnes maze. The APTw signal in the perilesion ipsilateral cortex gradually increased after TBI, and the value at 3 days correlated with microglia density at 3 days and with spatial memory decline and anhedonia at 28 days. The correlation between APTw and activated microglia was also observed in the ipsilateral thalamus, and its correlation to memory deficit and depression was evident in other ipsilateral sites. These results suggest that APTw imaging can be used for detecting secondary injury and as a potential predictor of long-term outcomes from TBI.

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TI Vitamin D Protects against Traumatic Brain Injury via Modulating

TLR4/MyD88/NF-κB Pathway-Mediated Microglial Polarization and

Neuroinflammation

SO BIOMED RESEARCH INTERNATIONAL

LA English

DT Article

AB Vitamin D (VD) deficiency is associated with neuroinflammation and neurocognitive deficits in patients with traumatic brain injury (TBI). The present study was aimed at investigating the therapeutic effects of VD and the molecular mechanisms after TBI. After the intraperitoneal injection of VD (1 mu g/kg), sensorimotor and cognitive function was assessed via a series of behavioral tests in TBI rats. Traumatic outcomes were investigated by brain edema, blood-brain barrier (BBB) disruption, and morphologic staining. In vitro, cellular viability and cytotoxicity in primary hippocampal neurons were detected via the MTT method and LDH release. Hippocampal oxidative stress-related enzymes and proinflammatory mediators and the serum concentration of VD were analyzed by ELISA. The expression of VDR, TLR4, MyD88, and NF-kappa B p65 was measured by Western blot. Furthermore, the levels of M1/M2 microglial markers were quantified using real-time PCR and Western blot. VD treatment significantly increased the serum level of VD and the hippocampal expression of VDR. VD not only effectively alleviated neurocognitive deficits, brain edema, and BBB disruption but also promoted hippocampal neuronal survival in vivo and in vitro. Moreover, VD therapy prevented excessive neuroinflammation and oxidative stress caused by TBI. Mechanically, the hippocampal expression of TLR4, MyD88, and nuclear NF-kappa B p65 was elevated in the TBI group but robustly restrained by VD treatment. Taken together, VD provides an important neuroprotection through modulating hippocampal microglial M2 polarization and neuroinflammation via the TLR4/MyD88/NF-kappa B pathway.

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TI Repeated mild traumatic brain injuries in mice cause age- and

sex-specific alterations in dendritic spine density

SO EXPERIMENTAL NEUROLOGY

LA English

DT Article; Early Access

DE Microglia; Behaviour; Synaptic pruning; Golgi-cox

ID MEDIAL PREFRONTAL CORTEX; HIGH-SCHOOL; SYNAPTIC PROTEINS; OXIDATIVE

STRESS; CORTICAL IMPACT; HEAD-INJURY; ADOLESCENCE; CONCUSSION;

EPIDEMIOLOGY; MATURATION

AB Mild traumatic brain injuries (mTBI) plague the human population and their prevalence is increasing annually. More so, repeated mTBIs (RmTBI) are known to manifest and compound neurological deficits in vulnerable populations. Age at injury and sex are two important factors influencing RmTBI pathophysiology, but we continue to know little about the specific effects of RmTBI in youth and females. In this study, we directly quantified the effects of RmTBI on adolescent and adult, male and female mice, with a closed-head lateral impact model. We report age-and sex-specific neurobehavioural deficits in motor function and working memory, microglia responses to injury, and the subsequent changes in dendritic spine density in select brain regions. Specifically, RmTBI caused increased footslips in adult male mice as assessed in a beam walk assay and signif-icantly reduced the time spent with a novel object in adolescent male and female mice. RmTBIs caused a sig-nificant reduction in microglia density in male mice in the motor cortex, but not female mice. Finally, RmTBI significantly reduced dendritic spine density in the agranular insular cortex (a region of the prefrontal cortex in mice) and increased dendritic spine density in the adolescent male motor cortex. Together, the data provided in this study sheds new light on the heterogeneity in RmTBI-induced behavioural, glial, and neuronal architecture changes dependent on age and sex.

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TI Activation of NLRP3 Is Required for a Functional and Beneficial

Microglia Response after Brain Trauma

SO PHARMACEUTICS

LA English

DT Article

DE traumatic brain injury; inflammasome; NLRP3; neuroinflammation;

microglia; astrocytes; MCC950

ID INFLAMMASOME; INJURY; ASTROCYTE; MODEL; NEUROINFLAMMATION; PROTEOLYSIS;

EXPRESSION; IL-1-BETA; INHIBITOR; MICE

AB Despite the numerous research studies on traumatic brain injury (TBI), many physiopathologic mechanisms remain unknown. TBI is a complex process, in which neuroinflammation and glial cells play an important role in exerting a functional immune and damage-repair response. The activation of the NLRP3 inflammasome is one of the first steps to initiate neuroinflammation and so its regulation is essential. Using a closed-head injury model and a pharmacological (MCC950; 3 mg/kg, pre- and post-injury) and genetical approach (NLRP3 knockout (KO) mice), we defined the transcriptional and behavioral profiles 24 h after TBI. Wild-type (WT) mice showed a strong pro-inflammatory response, with increased expression of inflammasome components, microglia and astrocytes markers, and cytokines. There was no difference in the IL1 beta production between WT and KO, nor compensatory mechanisms of other inflammasomes. However, some microglia and astrocyte markers were overexpressed in KO mice, resulting in an exacerbated cytokine expression. Pretreatment with MCC950 replicated the behavioral and blood-brain barrier results observed in KO mice and its administration 1 h after the lesion improved the damage. These findings highlight the importance of NLRP3 time-dependent activation and its role in the fine regulation of glial response.

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U2 4

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PA ST ALBAN-ANLAGE 66, CH-4052 BASEL, SWITZERLAND

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J9 PHARMACEUTICS

JI Pharmaceutics

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TI A Novel In Vivo Model for Multiplexed Analysis of Callosal Connections

upon Cortical Damage

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE brain damage; stroke; traumatic injury; neuronal regeneration;

pre-clinical models; callosal neurons; cortico-cortical connections;

perineuronal network; microglia

ID ISCHEMIC-STROKE; NEURAL REPAIR; ANIMAL-MODELS; ACTIVATION; INJURY

AB Brain damage is the major cause of permanent disability and it is particularly relevant in the elderly. While most studies focused on the immediate phase of neuronal loss upon injury, much less is known about the process of axonal regeneration after damage. The development of new refined preclinical models to investigate neuronal regeneration and the recovery of brain tissue upon injury is a major unmet challenge. Here, we present a novel experimental paradigm in mice that entails the (i) tracing of cortico-callosal connections, (ii) a mechanical lesion of the motor cortex, (iii) the stereological and histological analysis of the damaged tissue, and (iv) the functional characterization of motor deficits. By combining conventional microscopy with semi-automated 3D reconstruction, this approach allows the analysis of fine subcellular structures, such as axonal terminals, with the tridimensional overview of the connectivity and tissue integrity around the lesioned area. Since this 3D reconstruction is performed in serial sections, multiple labeling can be performed by combining diverse histological markers. We provide an example of how this methodology can be used to study cellular interactions. Namely, we show the correlation between active microglial cells and the perineuronal nets that envelop parvalbumin interneurons. In conclusion, this novel experimental paradigm will contribute to a better understanding of the molecular and cellular interactions underpinning the process of cortical regeneration upon brain damage.

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TI Engineering antioxidant poly (citrate-gallic acid)-Exosome hybrid

hydrogel with microglia immunoregulation for Traumatic Brain Injury-post

neuro-restoration

SO COMPOSITES PART B-ENGINEERING

LA English

DT Article

DE Bioactive materials; Polycitrate; Nanocomposites; Multifunctional

scaffolds; Tissue engineering

ID STEM-CELLS; PATHOPHYSIOLOGY

AB Traumatic brain injury (TBI) leads to high rates of morbidity and mortality worldwide with few effective treatments. Excessive oxidative stress and local inflammation at the injury site are considered as the critical factors that determine the therapeutic outcome. In our previous study, stem cells from human exfoliated deciduous teeth-derived exosomes (SHED-Exo) promoted functional recovery of TBI by amelioration of neuroinflammation. However, the effect was restricted due to unsustainable exosome release and existed oxidative stress that aggravate secondary insult of TBI. To obtain a long-time anti-inflammatory effect of SHED-Exo and attenuate oxidative stress simultaneously, we designed a bioactive antioxidant poly (citrate-gallic acid)-based hybrid hydrogel (FPGEGa) that encapsulate SHED-Exo for treating TBI. The thermosensitive, injectable, selfhealing and antioxidant FPGEGa presented ultralong sustained release of SHED-Exo (above 21 days) and significantly decreased the intracellular ROS production of microglia in the central nervous system. FPGEGa carrying SHED-Exo (FPGEGa@SHED-Exo) exhibited better anti-inflammatory potential on microglia, by promoting M2 (anti-inflammatory) polarization and inhibiting M1 (pro-inflammatory) polarization. FPGEGa@SHED-Exo could improve the neuro-regeneration of TBI rats by rescuing damaged motor functions in rats as well as regenerating impaired cortical tissues. The present work suggested that SHED-Exo engineered bioactive antioxidative hydrogel may be promising in achieving satisfactory functional recovery in TBI and other related neurological disorders.

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Chen, TB

Wang, TH

Huang, J

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Chen, Jilin

Chen, Tingbao

Wang, Tinghua

Huang, Jin

TI The AMPK-SIRT1-FoxO1-NF-κB signaling pathway participates in

hesperetin-mediated neuroprotective effects against traumatic brain

injury via the NLRP3 inflammasome

SO IMMUNOPHARMACOLOGY AND IMMUNOTOXICOLOGY

LA English

DT Article; Early Access

DE Hesperetin; traumatic brain injury; NLRP3; inflammation; microglial

activation

ID OXIDATIVE STRESS; ACTIVATION; BLOOD; RATS; NEUROINFLAMMATION;

INHIBITION; APOPTOSIS; MICROGLIA; SIRT1

AB Background Traumatic brain injury (TBI) induces inflammations that lead to secondary damage. Hesperetin (Hes) exerts anti-inflammatory activities against central nervous system (CNS) diseases. This article probes the possible neuroprotective effect and mechanism of Hes on TBI-induced acute cerebral damage. Methods Male C57BL/6J mice were subjected to controlled cortical impingement (CCI) and Hes (50 mg/kg) treatment after the surgery. Short-term neurological deficits were assessed with the modified neurological severity score (mNSS) and the Rota-rod test. The brain edema was tested by the wet/dry method. Neuron apoptosis was evaluated by Nissl staining and terminal deoxynucleotidyl transferase-mediated dUTP-biotin nick end labeling (TUNEL) staining. The blood-brain barrier (BBB) integrity was measured by Evans' blue staining, and immunohistochemistry (IHC) was conducted to study BV2 microglial activation. BV2 microglia and HT22 neuronal cells were stimulated by oxygen-glucose deprivation followed by recovery (OGD/R) and processed with Hes. Quantitative real-time-polymerase chain reaction (qRT-PCR) and enzyme-linked immunosorbent assay (ELISA) were implemented to gauge the expression of inflammatory cytokines, including tumor necrosis factor-alpha (TNF-alpha), interleukin-beta (IL-1-beta) and interleukin-6 (IL-6). Western blot (WB) was performed to check AMPK-SIRT1-FoxO1 both in vitro and in vivo. Results Hes eased neurological deficits, cerebral edema, and neuronal apoptosis in mice following TBI. Hes hampered microglial activation and pro-inflammatory cytokines production. Hes promoted AMPK and SIRT1 expression, whereas repressed the phosphorylation of FoxO1-NF-kappa B, and inhibited NLRP3 expression. The AMPK inhibitor Compound C markedly reversed Hes-mediated anti-inflammatory and neuron-protective effects. Conclusion Hes curbs microglial activation-mediated inflammation via the AMPK-SIRT1-FoxO1-NF-kappa B axis, thereby improving neurobehavioral function after TBI.

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Z9 7

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TI Microglia-specific deletion of histone deacetylase 3 promotes

inflammation resolution, white matter integrity, and functional recovery

in a mouse model of traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Conditional gene knockout; Controlled cortical impact; HDAC3;

Neuroinflammation

ID MICROGLIA/MACROPHAGE POLARIZATION; MILD; DEFICITS; HDAC3; MACROPHAGES;

PROGRESSION; INHIBITOR; BEHAVIOR; DAMAGE; ROLES

AB Background Histone deacetylases (HDACs) are believed to exacerbate traumatic brain injury (TBI) based on studies using pan-HDAC inhibitors. However, the HDAC isoform responsible for the detrimental effects and the cell types involved remain unknown, which may hinder the development of specific targeting strategies that boost therapeutic efficacy while minimizing side effects. Microglia are important mediators of post-TBI neuroinflammation and critically impact TBI outcome. HDAC3 was reported to be essential to the inflammatory program of in vitro cultured macrophages, but its role in microglia and in the post-TBI brain has not been investigated in vivo. Methods We generated HDAC3(LoxP) mice and crossed them with CX3CR1(CreER) mice, enabling in vivo conditional deletion of HDAC3. Microglia-specific HDAC3 knockout (HDAC3 miKO) was induced in CX3CR1(CreER):HDAC3(LoxP) mice with 5 days of tamoxifen treatment followed by a 30-day development interval. The effects of HDAC3 miKO on microglial phenotype and neuroinflammation were examined 3-5 days after TBI induced by controlled cortical impact. Neurological deficits and the integrity of white matter were assessed for 6 weeks after TBI by neurobehavioral tests, immunohistochemistry, electron microscopy, and electrophysiology. Results HDAC3 miKO mice harbored specific deletion of HDAC3 in microglia but not in peripheral monocytes. HDAC3 miKO reduced the number of microglia by 26%, but did not alter the inflammation level in the homeostatic brain. After TBI, proinflammatory microglial responses and brain inflammation were markedly alleviated by HDAC3 miKO, whereas the infiltration of blood immune cells was unchanged, suggesting a primary effect of HDAC3 miKO on modulating microglial phenotype. Importantly, HDAC3 miKO was sufficient to facilitate functional recovery for 6 weeks after TBI. TBI-induced injury to axons and myelin was ameliorated, and signal conduction by white matter fiber tracts was significantly enhanced in HDAC3 miKO mice. Conclusion Using a novel microglia-specific conditional knockout mouse model, we delineated for the first time the role of microglial HDAC3 after TBI in vivo. HDAC3 miKO not only reduced proinflammatory microglial responses, but also elicited long-lasting improvement of white matter integrity and functional recovery after TBI. Microglial HDAC3 is therefore a promising therapeutic target to improve long-term outcomes after TBI.

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TI Early posttraumatic CSF1R inhibition via PLX3397 leads to time- and

sex-dependent effects on inflammation and neuronal maintenance after

traumatic brain injury in mice

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article; Early Access

DE Traumatic brain injury; Colony stimulating factor 1 receptor; Microglia;

Inflammation; Phagocytosis; Hematoma; Therapy; RNAseq; Synapse; Sex

ID OXIDATIVE STRESS; MICROGLIAL ACTIVATION; NEUROTOXICITY; ALPHA;

INTERLEUKIN-1; DEGENERATION; CONTRIBUTES; DYSFUNCTION; HOMEOSTASIS;

APOPTOSIS

AB Background: There is a need for early therapeutic interventions after traumatic brain injury (TBI) to prevent neurodegeneration. Microglia/macrophage (M/M) depletion and repopulation after treatment with colony stimulating factor 1 receptor (CSF1R) inhibitors reduces neurodegeneration. The present study investigates short-and long-term consequences after CSF1R inhibition during the early phase after TBI.Methods: Sex-matched mice were subjected to TBI and CSF1R inhibition by PLX3397 for 5 days and sacrificed at 5 or 30 days post injury (dpi). Neurological deficits were monitored and brain tissues were examined for histo-and molecular pathological markers. RNAseq was performed with 30 dpi TBI samples.Results: At 5 dpi, CSF1R inhibition attenuated the TBI-induced perilesional M/M increase and associated gene expressions by up to 50%. M/M attenuation did not affect structural brain damage at this time-point, impaired hematoma clearance, and had no effect on IL-1 beta expression. At 30 dpi, following drug discontinuation at 5 dpi and M/M repopulation, CSF1R inhibition attenuated brain tissue loss regardless of sex, as well as hippocampal atrophy and thalamic neuronal loss in male mice. Selected gene markers of brain inflammation and apoptosis were reduced in males but increased in females after early CSF1R inhibition as compared to corresponding TBI vehicle groups. Neurological outcome in behaving mice was almost not affected. RNAseq and gene set enrich-ment analysis (GSEA) of injured brains at 30 dpi revealed more genes associated with dendritic spines and synapse function after early CSF1R inhibition as compared to vehicle, suggesting improved neuronal mainte-nance and recovery. In TBI vehicle mice, GSEA showed high oxidative phosphorylation, oxidoreductase activity and ribosomal biogenesis suggesting oxidative stress and increased abundance of metabolically highly active cells. More genes associated with immune processes and phagocytosis in PLX3397 treated females vs males, suggesting sex-specific differences in response to early CSF1R inhibition after TBI.Conclusions: M/M attenuation after CSF1R inhibition via PLX3397 during the early phase of TBI reduces long-term brain tissue loss, improves neuronal maintenance and fosters synapse recovery. Overall effects were not sex-specific but there is evidence that male mice benefit more than female mice.

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TI Spatial-temporal changes of iron deposition and iron metabolism after

traumatic brain injury in mice

SO FRONTIERS IN MOLECULAR NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury (TBI); iron deposition; iron metabolism; neuron;

oligodendrocyte; astrocyte; microglia

ID INTRACEREBRAL HEMORRHAGE; H-FERRITIN; NEURONAL DEATH; WHITE-MATTER;

POLARIZATION; MICROGLIA; RECEPTOR; OLIGODENDROCYTES; FERROPTOSIS; DAMAGE

AB Excessive iron released by hemoglobin and necrotic tissues is the predominant factor that aggravates the outcome of traumatic brain injury (TBI). Regulating the levels of iron and its metabolism is a feasible way to alleviate damage due to TBI. However, the spatial-temporal iron metabolism and iron deposition in neurons and glial cells after TBI remains unclear. In our study, male C57BL/6 mice (8-12 weeks old, weighing 20-26 g) were conducted using controlled cortical impact (CCI) models, combined with treatment of iron chelator deferoxamine (DFO), followed by systematical evaluation on iron deposition, cell-specific expression of iron metabolic proteins and ferroptosis in ipsilateral cortex. Herein, ferroptosis manifest by iron overload and lipid peroxidation was noticed in ipsilateral cortex. Furthermore, iron deposition and cell-specific expression of iron metabolic proteins were observed in the ipsilateral cortical neurons at 1-3 days post-injury. However, iron overload was absent in astrocytes, even though they had intense TBI-induced oxidative stress. In addition, iron accumulation in oligodendrocytes was only observed at 7-14 days post-injury, which was in accordance with the corresponding interval of cellular repair. Microglia play significant roles in iron engulfment and metabolism after TBI, and excessive affects the transformation of M1 and M2 subtypes and activation of microglial cells. Our study revealed that TBI led to ferroptosis in ipsilateral cortex, iron deposition and metabolism exhibited cell-type-specific spatial-temporal changes in neurons and glial cells after TBI. The different effects and dynamic changes in iron deposition and iron metabolism in neurons and glial cells are conducive to providing new insights into the iron-metabolic mechanism and strategies for improving the treatment of TBI.

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Wang, Jinhui

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TI Human umbilical cord mesenchymal stem cell-derived exosomes promote

neurological function recovery in rat after traumatic brain injury by

inhibiting the activation of microglia and astrocyte

SO REGENERATIVE THERAPY

LA English

DT Article; Early Access

DE Umbilical cord mesenchymal stem cells; Exosomes; Traumatic brain injury;

Microglia; Astrocytes

ID STROMAL CELLS; PATHOPHYSIOLOGY; GLIA

AB Traumatic brain injury (TBI) is a serious neurological disorder with increasing worldwide incidence. Emerging evidence has shown a significant therapeutic role of mesenchymal stem cells (MSCs) derived exosomes on traumatic brain injury with broad application prospects as a cell-free therapy. However, a comprehensive understanding of its underlying mechanism remained elusive. In this study, umbilical cord mesenchymal stem cells (UCMSCs)-derived exosomes (UC-MSCs-Exo) were isolated by ultracen-trifugation and injected intraventricularly in a rat model of TBI. Our results showed that UC-MSCs-Exo promoted functional recovery and reduced neuronal apoptosis in TBI rats. Moreover, UC-MSCs-Exo inhibited the activation of microglia and astrocytes during brain injury, thereby promoting functional recovery. However, the effect of UC-MSCs-Exo on the content of plasma inflammatory factors in rats was not significant. Collectively our study suggested that UC-MSCs-Exo promotes the recovery of neurological function in TBI rats by inhibiting the activation of microglia and astrocytes, providing a theoretical basis for new therapeutic strategies for central nervous system diseases.(c) 2022, The Japanese Society for Regenerative Medicine. Production and hosting by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/ 4.0/).

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NR 32

TC 10

Z9 11

U1 0

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JI Regen. Ther.

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TI Effects of acupuncture on microglial polarization and the

TLR4/TRIF/MyD88 pathway in a rat model of traumatic brain injury

SO ACUPUNCTURE IN MEDICINE

LA English

DT Article; Early Access

DE acupuncture; intracellular toll-interleukin-1 receptor domain-containing

adaptor inducing interferon-beta; microglial polarization; myeloid

differentiation factor 88; toll-like receptor 4; traumatic brain injury

ID RECEPTOR 4; TLR4; NEUROINFLAMMATION; PHENOTYPE; HEALTH

AB Objective: Neuroinflammation caused by traumatic brain injury (TBI) can lead to neurological deficits. Acupuncture can inhibit neuroinflammation and promote nerve repair; however, the specific mechanism is still unclear. The purpose of this study was to explore whether acupuncture could modulate the M1 and M2 phenotypic polarization of microglia in a rat model of TBI via the toll-like receptor 4 (TLR4)/intracellular toll-interleukin-1 receptor (TIR) domain-containing adaptor inducing interferon-beta (TRIF)/myeloid differentiation factor 88 (MyD88) pathway.

Methods: A total of 90 adult male Sprague-Dawley (SD) rats, SPF grade, were randomly divided into a normal group, model group and acupuncture group. Each group was further divided into three subgroups (first, third, and fifth day groups) according to the treatment time (n= 10 rats/subgroup). We used the modified neurological severity score (mNSS) method to quantify neurological deficits before and after modeling. We used Nissl staining to observe the pathological changes in brain tissue, flow cytometry to detect the proportion of M1 and M2 polarized microglia in the injured area on the first, third and fifth day, and co-immunoprecipitation (Co-IP) to examine TLR4/TRIF/MyD88 expression in microglia on the first, third and fifth day, as well as expression of the amount of binding of TLR4 with TRIF and MyD88.

Results: Compared to the model group, mNSS in the acupuncture group gradually decreased and pathological morphology improved. The proportion of CD11b/CD86 positive cells was decreased, while that of CD11b/CD206 was increased in the acupuncture group. Expression of IP TLR4, IP TRIF and IP MyD88 also decreased in the acupuncture group.

Conclusion: The results of this study demonstrate that one of the mechanisms through which acupuncture mitigates neuroinflammation and promotes nerve repair in TBI rats may be inhibition of M1 phenotypic polarization and promotion of M2 phenotypic polarization through inhibition of the TLR4/TRIF/MyD88 signaling pathway.

C1 [Cao, Lu-Xi; Lin, Shu-Jun; Zhao, Si-Si; Wang, Shi-Qi; Zeng, Hai; Chen, Wen-An; Lin, Zhuo-Wen; Chen, Jia-Xu; Zhu, Ming-Min; Zhang, Yi-Min] Jinan Univ, Sch Tradit Chinese Med, Guangzhou 510632, Peoples R China.

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TI Correction of Behavioral Disorders and State of Microglia with

Recombinant IL-1 Receptor Antagonist in Experimental Traumatic Brain

Injury

SO JOURNAL OF EVOLUTIONARY BIOCHEMISTRY AND PHYSIOLOGY

LA English

DT Article

DE TBI; corticosterone; rIL-1RA; behavior; microglia

ID CEREBRAL-ISCHEMIA; HPA AXIS; INTERLEUKIN-1; STRESS; NEUROINFLAMMATION;

EXPRESSION; CYTOKINE

AB Traumatic brain injury (TBI) is a multifactorial disease that can lead to the development of neurological diseases. To correct violations of physiological functions, anti-inflammatory cytokines are used, in particular, the IL-1 receptor antagonist (IL-1RA). The aim of this work is to evaluate the effectiveness of the rIL-1RA preparation for the correction of post-traumatic neuroinflammation. TBI in rats was simulated by dropping a 115 g weight from a height of 120 cm into the center of the parietal region, and the drug was injected subcutaneously at a dose of 50 mg/kg 60 min after injury. We studied blood corticosterone levels and behavioral responses in the "Open field" test. To characterize the activation pattern of microglia in different parts of the brain, the expression of the Iba1 marker and morphological changes of cells were evaluated. Counting the total number and changing the shape and size of Iba1-positive microglial cells on 7th day after TBI showed that in animals treated with rIL-1RA the number of activated microglial cells was significantly higher than in intact animals, but the degree of their activation was significantly lower. Studies of CNS function disorders after TBI showed that motor and orientation-exploratory activities were significantly inhibited, which, together with the disturbance of the emotional status of the animals, indicates the development of a neurological deficit in intact rats. In animals treated with rIL-1RA, changes in behavioral characteristics were less pronounced. The decrease in neurological deficit in treated animals was directly related to the normalization of the state of microglia. The data obtained in the work indicate that the use of rIL-1RA 1 h after TBI allows correction of motor, orientation-exploratory activity and reduce microglia activation in different parts of the CNS.

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TI Gut Microbiota Dysbiosis after Traumatic Brain Injury Contributes to

Persistent Microglial Activation Associated with Upregulated Lyz2 and

Shifted Tryptophan Metabolic Phenotype

SO NUTRIENTS

LA English

DT Article

DE gut microbiota; tryptophan metabolism; microglia; Lyz2; chronic

neuroinflammation; traumatic brain injury

ID INFLAMMATION; HEALTH; SERUM

AB Traumatic brain injury (TBI) is a common cause of disability and mortality, affecting millions of people every year. The neuroinflammation and immune response post-TBI initially have neuroprotective and reparative effects, but prolonged neuroinflammation leads to secondary injury and increases the risk of chronic neurodegenerative diseases. Persistent microglial activation plays a critical role in chronic neuroinflammation post-TBI. Given the bidirectional communication along the brain-gut axis, it is plausible to suppose that gut microbiota dysbiosis post-TBI influences microglial activation. In the present study, hippocampal microglial activation was observed at 7 days and 28 days post-TBI. However, in TBI mice with a depletion of gut microbiota, microglia were activated at 7 days post-TBI, but not at 28 days post-TBI, indicating that gut microbiota contributes to the long-term activation of microglia post-TBI. In addition, in conventional mice colonized by the gut microbiota of TBI mice using fecal microbiota transplant (FMT), microglial activation was observed at 28 days post-TBI, but not at 7 days post-TBI, supporting the role of gut microbiota dysbiosis in persistent microglial activation post-TBI. The RNA sequencing of the hippocampus identified a microglial activation gene, Lyz2, which kept upregulation post-TBI. This persistent upregulation was inhibited by oral antibiotics and partly induced by FMT. 16s rRNA gene sequencing showed that the composition and function of gut microbiota shifted over time post-TBI with progressive dysbiosis, and untargeted metabolomics profiling revealed that the tryptophan metabolic phenotype was differently reshaped at 7 days and 28 days post-TBI, which may play a role in the persistent upregulation of Lyz2 and the activation of microglia. This study implicates that gut microbiota and Lyz2 are potential targets for the development of novel strategies to address persistent microglial activation and chronic neuroinflammation post-TBI, and further investigations are warranted to elucidate the specific mechanism.

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TI LDC7559 Exerts Neuroprotective Effects by Inhibiting GSDMD-Dependent

Pyroptosis of Microglia in Mice with Traumatic Brain Injury

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE gasdermin D; inflammation; pyroptosis; traumatic brain injury

ID GASDERMIN-D; CELL-DEATH; NEUROINFLAMMATION; RESPONSES; MEMBRANE;

CASPASES; MEMORY

AB Pyroptosis is considered one of a critical factor in the recovery of neurological function following traumatic brain injury. Brain injury activates a molecular signaling cascade associated with pyroptosis and inflammation, including NLRP3, inflammatory cytokines, caspase-1, gasdermin D (GSDMD), and other pyroptosis-related proteins. In this study, we explored the neuroprotective effects of LDC7559, a GSDMD inhibitor. Briefly, LDC7559, siRNA-GSDMD (si-GSDMD), or equal solvent was administrated to mice with a lipopolysaccharide + nigericin (LPS + Nig) model in vitro or with controlled cortical impact brain injury. The findings revealed that inflammation and pyroptosis levels were decreased by LDC7559 or si-GSDMD treatment both in vitro and in vivo. Immunofluorescence staining, brain water content, hematoxylin and eosin staining, and behavioral investigations suggested that LDC7559 or si-GSDMD inhibited microglial proliferation, ameliorated cerebral edema, reduced brain tissue loss, and promoted brain function recovery. Taken together, LDC7559 may inhibit pyroptosis and reduce inflammation by inhibiting GSDMD, thereby promoting the recovery of neurological function.

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NR 62

TC 5

Z9 5

U1 4

U2 17

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J9 J NEUROTRAUM

JI J. Neurotrauma

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TI Extracellular Mitochondria Activate Microglia and Contribute to

Neuroinflammation in Traumatic Brain Injury

SO NEUROTOXICITY RESEARCH

LA English

DT Article; Early Access

DE Extracellular mitochondria; Traumatic brain injury; Microglia;

Neuroinflammation; Brain edema; Reactive oxygen species

ID NADPH OXIDASES; MICROPARTICLES; COAGULOPATHY; PATHWAY; MODEL

AB Traumatic brain injury (TBI)-induced neuroinflammation is closely associated with poor outcomes and high mortality in affected patients, with unmet needs for effective clinical interventions. A series of causal and disseminating factors have been identified to cause TBI-induced neuroinflammation. Among these are cellular microvesicles released from injured cerebral cells, endothelial cells, and platelets. In previous studies, we have put forward that cellular microvesicles can be released from injured brains that induce consumptive coagulopathy. Extracellular mitochondria accounted for 55.2% of these microvesicles and induced a redox-dependent platelet procoagulant activity that contributes to traumatic brain injury-induced coagulopathy and inflammation. These lead to the hypothesis that metabolically active extracellular mitochondria contribute to the neuroinflammation in traumatic brain injury, independent of their procoagulant activity. Here, we found that these extracellular mitochondria induced polarization of microglial M1-type pro-inflammatory phenotype, aggravating neuroinflammation, and mediated cerebral edema in a ROS-dependent manner. In addition, the effect of ROS can be alleviated by ROS inhibitor N-ethylmaleimide (NEM) in vitro experiments. These results revealed a novel pro-inflammatory activity of extracellular mitochondria that may contribute to traumatic brain injury-associated neuroinflammation.

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NR 38

TC 2

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TI STAT1 Contributes to Microglial/Macrophage Inflammation and Neurological

Dysfunction in a Mouse Model of Traumatic Brain Injury

SO JOURNAL OF NEUROSCIENCE

LA English

DT Article

DE behavioral test; conditional gene KO; controlled cortical impact;

fludarabine; neuroinflammation; signal transducer and activator of

transcription 1

ID WHITE-MATTER INJURY; MICROGLIA/MACROPHAGE POLARIZATION; MACROPHAGE

POLARIZATION; NEUROINFLAMMATION; FLUDARABINE; INHIBITION; ACTIVATION;

ALPHA

AB Traumatic brain injury (TBI) triggers a plethora of inflammatory events in the brain that aggravate secondary injury and impede tis-sue repair. Resident microglia (Mi) and blood-borne infiltrating macrophages (M phi) are major players of inflammatory responses in the post-TBI brain and possess high functional heterogeneity. However, the plasticity of these cells has yet to be exploited to develop therapies that can mitigate brain inflammation and improve the outcome after TBI. This study investigated the transcription factor STAT1 as a key determinant of proinflammatory Mi/M phi responses and aimed to develop STAT1 as a novel therapeutic target for TBI using a controlled cortical impact model of TBI on adult male mice. TBI induced robust upregulation of STAT1 in the brain at the subacute injury stage, which occurred primarily in Mi/M phi. Intraperitoneal administration of fludarabine, a selective STAT1 inhib-itor, markedly alleviated proinflammatory Mi/M phi responses and brain inflammation burden after TBI. Such phenotype-modulating effects of fludarabine on post-TBI Mi/M phi were reproduced by tamoxifen-induced, selective KO of STAT1 in Mi/M phi (STAT1 mKO). By propelling Mi/M phi away from a detrimental proinflammatory phenotype, STAT1 mKO was sufficient to reduce long-term neuro-logic deficits and brain lesion size after TBI. Importantly, short-term fludarabine treatment after TBI elicited long-lasting improvement of TBI outcomes, but this effect was lost on STAT1 mKO mice. Together, our study provided the first line of evidence that STAT1 causatively determines the proinflammatory phenotype of brain Mi/M phi after TBI. We also showed promising preclinical data sup-porting the use of fludarabine as a novel immunomodulating therapy to TBI.

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TI Post-Injury Buprenorphine Administration Is Associated with Long-Term

Region-Specific Glial Alterations in Rats

SO PHARMACEUTICS

LA English

DT Article

DE traumatic brain injury; buprenorphine; Bup-SR-Lab; microglia; astrocyte;

myelin; membrane disruption; somatosensory sensitivity

ID TRAUMATIC BRAIN-INJURY; SUSTAINED-RELEASE BUPRENORPHINE;

SPINAL-CORD-INJURY; INTRACRANIAL-PRESSURE; BEHAVIORAL MORBIDITY; AXONAL

INJURY; UNITED-STATES; LATE-ONSET; DAMAGE; PATHOLOGY

AB Traumatic brain injury (TBI) is a major leading cause of death and disability. While previous studies regarding focal pathologies following TBI have been done, there is a lack of information concerning the role of analgesics and their influences on injury pathology. Buprenorphine (Bup), an opioid analgesic, is a commonly used analgesic in experimental TBI models. Our previous studies investigated the acute effects of Buprenorphine-sustained release-Lab (Bup-SR-Lab) on diffuse neuronal/glial pathology, neuroinflammation, cell damage, and systemic physiology. The current study investigated the longer-term chronic outcomes of Bup-SR-Lab treatment at 4 weeks following TBI utilizing a central fluid percussion injury (cFPI) model in adult male rats. Histological assessments of physiological changes, neuronal damage, cortical and thalamic cytokine expression, microglial and astrocyte morphological changes, and myelin alterations were done, as we had done in our acute study. In the current study the Whisker Nuisance Task (WNT) was also performed pre- and 4w post-injury to assess changes in somatosensory sensitivity following saline or Bup-SR-Lab treatment. Bup-SR-Lab treatment had no impact on overall physiology or neuronal damage at 4w post-injury regardless of region or injury, nor did it have any significant effects on somatosensory sensitivity. However, greater IL-4 cytokine expression with Bup-SR-Lab treatment was observed compared to saline treated animals. Microglia and astrocytes also demonstrated region-specific morphological alterations associated with Bup-SR-Lab treatment, in which cortical microglia and thalamic astrocytes were particularly vulnerable to Bup-mediated changes. There were discernable injury-specific and region-specific differences regarding myelin integrity and changes in specific myelin basic protein (MBP) isoform expression following Bup-SR-Lab treatment. This study indicates that use of Bup-SR-Lab could impact TBI-induced glial alterations in a region-specific manner 4w following diffuse brain injury.

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Z9 1

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PU MDPI

PI BASEL

PA ST ALBAN-ANLAGE 66, CH-4052 BASEL, SWITZERLAND

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TI POSTINJURY FECAL MICROBIOME TRANSPLANT DECREASES LESION SIZE AND

NEUROINFLAMMATION IN TRAUMATIC BRAIN INJURY

SO SHOCK

LA English

DT Article

DE Anxiety; behavior; controlled cortical impact; dysbiosis; fecal

microbiome transplantation; microbiome; microglia; transcriptome;

trauma; traumatic brain injury

ID GUT DYSBIOSIS; INFLAMMATION; ACTIVATION; MICROGLIA; AXIS

AB Background: Traumatic brain injury (TBI) is an underrecognized public health threat. The constitutive activation of microglia after TBI has been linked to long-term neurocognitive deficits and the progression of neurodegenerative disease. Evolving evidence indicates a critical role for the gut-brain axis in this process. Specifically, TBI has been shown to induce the depletion of commensal gut bacteria. The resulting gut dysbiosis is associated with neuroinflammation and disease. Hypothesis: We hypothesized that fecal microbiota transplantation would attenuate microglial activation and improve neuropathology after TBI. Methods: C57Bl/6 mice were subjected to severe TBI (n = 10) or sham injury (n = 10) via an open-head controlled cortical impact. The mice underwent fecal microbiota transplantation (FMT) or vehicle alone via oral gavage once weekly for 4 weeks after injury. At 59 days after TBI, mice underwent three-dimensional, contrast-enhanced magnetic resonance imaging. Following imaging, mice were killed, brains harvested at 60 DPI, and CD45(+) cells isolated via florescence-activated cell sorting. cDNA libraries were prepared using the 10x Genomics Chromium Single Cell 3 ' Reagent kit followed by sequencing on a HiSeq4000 instrument, and computational analysis was performed. Results: Fecal microbiota transplantation resulted in a >marked reduction of ventriculomegaly (P < 0.002) and preservation of white matter connectivity at 59 days after TBI (P < 0.0001). In addition, microglia from FMT-treated mice significantly reduced inflammatory gene expression and enriched pathways involving the heat-shock response compared with mice treated with vehicle alone. Conclusions: We hypothesized that restoring gut microbial community structure via FMT would attenuate microglial activation and reduce neuropathology after TBI. Our data demonstrated significant preservation of cortical volume and white matter connectivity after an injury compared with mice treated with vehicle alone. This preservation of neuroanatomy after TBI was associated with a marked reduction in inflammatory gene expression within the microglia of FMT-treated mice. Microglia from FMT-treated mice enriched pathways in the heat-shock response, which is known to play a neuroprotective role in TBI and other neurodegenerative disease processes.

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NR 43

TC 3

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J9 SHOCK

JI Shock

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TI Microglial-oligodendrocyte interactions in myelination and neurological

function recovery after traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE White matter damage; Inflammation; Oligodendrocytes; Microglia; Na+; H+

exchanger; Traumatic brain injury

ID ACTIVATION; MEMORY; DIFFERENTIATION; MYELINOGENESIS; CELLS; MAZE

AB Differential microglial inflammatory responses play a role in regulation of differentiation and maturation of oligodendrocytes (OLs) in brain white matter. How microglia-OL crosstalk is altered by traumatic brain injury (TBI) and its impact on axonal myelination and neurological function impairment remain poorly understood. In this study, we investigated roles of a Na+/H+ exchanger (NHE1), an essential microglial pH regulatory protein, in microglial proinflammatory activation and OL survival and differentiation in a murine TBI model induced by controlled cortical impact. Similar TBI-induced contusion volumes were detected in the Cx3cr1-Cre(ERT2) control (Ctrl) mice and selective microglial Nhe1 knockout (Cx3cr1-Cre(ERT2);Nhe1(flox/flox), Nhe1 cKO) mice. Compared to the Ctrl mice, the Nhe1 cKO mice displayed increased resistance to initial TBI-induced white matter damage and accelerated chronic phase of OL regeneration at 30 days post-TBI. The cKO brains presented increased anti-inflammatory phenotypes of microglia and infiltrated myeloid cells, with reduced proinflammatory transcriptome profiles. Moreover, the cKO mice exhibited accelerated post-TBI sensorimotor and cognitive functional recovery than the Ctrl mice. These phenotypic outcomes in cKO mice were recapitulated in C57BL6J wild-type TBI mice receiving treatment of a potent NHE1 inhibitor HOE642 for 1-7 days post-TBI. Taken together, these findings collectively demonstrated that blocking NHE1 protein stimulates restorative microglial activation in oligodendrogenesis and neuroprotection, which contributes to accelerated brain repair and neurological function recovery after TBI.

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PU BMC

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TI Partial Depletion of Microglia Attenuates Long-Term Potentiation

Deficits following Repeated Blast Traumatic Brain Injury in Organotypic

Hippocampal Slice Cultures

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE electrophysiology; hippocampus; long-term potentiation; microglial

depletion; repeated blast traumatic brain injury

ID MICROGLIA/MACROPHAGE POLARIZATION DYNAMICS; TNF-ALPHA; CYTOKINE

RESPONSES; WHITE-MATTER; INFLAMMATION; EXPOSURE; CELL; BLOOD; IL-6;

NEUROINFLAMMATION

AB Blast-induced traumatic brain injury (bTBI) has been a health concern in both military and civilian populations due to recent military and geopolitical conflicts. Military service members are frequently exposed to repeated bTBI throughout their training and deployment. Our group has previously reported compounding functional deficits as a result of increased number of blast exposures. In this study, we further characterized the decrease in long-term potentiation (LTP) by varying the blast injury severity and the inter-blast interval between two blast exposures. LTP deficits were attenuated with increasing inter-blast intervals. We also investigated changes in microglial activation; expression of CD68 was increased and expression of CD206 was decreased after multiple blast exposures. Expression of macrophage inflammatory protein (MIP)-1 alpha, interleukin (IL)-1 beta, monocyte chemoattractant protein (MCP)-1, interferon gamma-inducible protein (IP)-10, and regulated on activation, normal T cell expressed and secreted (RANTES) increased, while expression of IL-10 decreased in the acute period after both single and repeated bTBI. By partially depleting microglia prior to injury, LTP deficits after injury were significantly reduced. Treatment with the novel drug, MW-189, prevented LTP deficits when administered immediately following a repeated bTBI and even when administered only for an acute period (24 h) between two blast injuries. These findings could inform the development of therapeutic strategies to treat the neurological deficits of repeated bTBI suggesting that microglia play a major role in functional neuronal deficits and may be a viable therapeutic target to lessen the neurophysiological deficits after bTBI.

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PT J

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Chen, SW

AF Zhang, Chunhao

Chen, Shiwen

TI Role of TREM2 in the Development of Neurodegenerative Diseases After

Traumatic Brain Injury

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article; Early Access

DE The Triggering Receptor Expressed on Myeloid Cells 2; TBI;

Neurodegenerative Diseases; Microglia

ID MYELOID CELLS 2; AMYLOID PROTEIN DEPOSITION; ALZHEIMERS-DISEASE;

ALPHA-SYNUCLEIN; RISK-FACTOR; PERIPHERAL MACROPHAGES; INFLAMMATORY

RESPONSES; MICROGLIAL ACTIVATION; RECEPTOR RESPONSES; PRECURSOR PROTEIN

AB Traumatic brain injury (TBI) has been found as the primary cause of morbidity and disability worldwide, which has posed a significant social and economic burden. The first stage of TBI produces brain edema, axonal damage, and hypoxia, thus having an effect on the blood-brain barrier function, promoting inflammatory responses, and increasing oxidative stress. Patients with TBI are more likely to develop post-traumatic epilepsy, behavioral issues, as well as mental illnesses. The long-term effects arising from TBI have aroused rising attention over the past few years. Microglia in the brain can express the triggering receptor expressed on myeloid cells 2 (TREM2), which is a single transmembrane receptor pertaining to the immunoglobulin superfamily. The receptor has been correlated with a number of neurodegenerative disorders, including Alzheimer's disease, Parkinson's disease, and other relevant diseases. In this review, it is demonstrated that TREM2 is promising to serve as a neuroprotective factor for neurodegenerative disorders following TBI by modulating the function of microglial cells. Accordingly, it has potential avenues for TREM2-related therapies to improve long-term recovery after TBI.

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TI Abrocitinib Attenuates Microglia-Mediated Neuroinflammation after

Traumatic Brain Injury via Inhibiting the JAK1/STAT1/NF-κB Pathway

SO CELLS

LA English

DT Article

DE TBI; abrocitinib; neuroinflammation; JAK1/STAT1/NF-kappa B

ID PATHOPHYSIOLOGY; INFLAMMATION; CELLS; MODULATION; ACTIVATION;

MECHANISMS; EXPRESSION; APOPTOSIS; OUTCOMES; DAMAGE

AB Background and Purpose: Neuroinflammation has been shown to play a critical role in secondary craniocerebral injury, leading to poor outcomes for TBI patients. Abrocitinib, a Janus kinase1 (JAK1) selective inhibitor approved to treat atopic dermatitis (AD) by the Food and Drug Administration (FDA), possesses a novel anti-inflammatory effect. In this study, we investigated whether abrocitinib could ameliorate neuroinflammation and exert a neuroprotective effect in traumatic brain injury (TBI) models. Methods: First, next-generation sequencing (NGS) was used to select genes closely related to neuroinflammation after TBI. Then, magnetic resonance imaging (MRI) was used to dynamically observe the changes in traumatic focus on the 1st, 3rd, and 7th days after the induction of fluid percussion injury (FPI). Moreover, abrocitinib's effects on neurobehaviors were evaluated. A routine peripheral blood test was carried out and Evans blue dye extravasation, cerebral cortical blood flow, the levels of inflammatory cytokines, and changes in the numbers of inflammatory cells were evaluated to investigate the function of abrocitinib on the 1st day post-injury. Furthermore, the JAK1/signal transducer and activator of transcription1 (STAT1)/nuclear factor kappa (NF-kappa B) pathway was assessed. Results: In vivo, abrocitinib treatment was found to shrink the trauma lesions. Compared to the TBI group, the abrocitinib treatment group showed better neurological function, less blood-brain barrier (BBB) leakage, improved intracranial blood flow, relieved inflammatory cell infiltration, and reduced levels of inflammatory cytokines. In vitro, abrocitinib treatment was shown to reduce the pro-inflammatory M1 microglia phenotype and shift microglial polarization toward the anti-inflammatory M2 phenotype. The WB and IHC results showed that abrocitinib played a neuroprotective role by restraining JAK1/STAT1/NF-kappa B levels after TBI. Conclusions: Collectively, abrocitinib treatment after TBI is accompanied by improvements in neurological function consistent with radiological, histopathological, and biochemical changes. Therefore, abrocitinib can indeed reduce excessive neuroinflammation by restraining the JAK1/STAT1/NF-kappa B pathway.

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TC 3

Z9 4

U1 3

U2 10

PU MDPI

PI BASEL

PA ST ALBAN-ANLAGE 66, CH-4052 BASEL, SWITZERLAND

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J9 CELLS-BASEL

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AU Katsumoto, A

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AF Katsumoto, Atsuko

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Lamb, Bruce T.

TI Triggering receptor expressed on myeloid cells 2 deficiency exacerbates

injury-induced inflammation in a mouse model of tauopathy

SO FRONTIERS IN IMMUNOLOGY

LA English

DT Article

DE TREM2; microglia; traumatic brain injury; tauopathy; neurodegeneration;

bloodbrain barrier; blood-brain barrier

ID TRAUMATIC BRAIN-INJURY; TREM2 DEFICIENCY; ALZHEIMERS-DISEASE; TAU

PATHOLOGY; AXONAL INJURY; MICROGLIA; NEURODEGENERATION; ACCUMULATION;

ACTIVATION; NEUROINFLAMMATION

AB Traumatic brain injury (TBI) promotes several Alzheimer's disease-like pathological features, including microtubule-associated protein tau (MAPT) accumulation within neurons. Macrophage activation in the injured hTau mouse model of tauopathy raises the question whether there is a relationship between MAPT pathology and alterations in macrophage activation following TBI. Triggering receptor expressed on myeloid cells 2 (TREM2) is a critical regulator of microglia and macrophage phenotype, but its mechanisms on TBI remain unclear. To address the association with TREM2 in TBI and MAPT pathology, we studied TREM2 deficiency in hTau mice (hTau;Trem2(-/-)) 3 (acute phase) and 120 (chronic phase) days after experimental TBI. At three days following injury, hTau;Trem2(-/-) mice exhibited reduced macrophage activation both in the cortex and hippocampus. However, to our surprise, hTau;Trem2(-/-) mice exposed to TBI augments macrophage accumulation in the corpus callosum and white matter near the site of tissue damage in a chronic phase, which results in exacerbated axonal injury, tau aggregation, and impaired neurogenesis. We further demonstrate that TREM2 deficiency in hTau injured mice promotes neuronal dystrophy in the white matter due to impaired phagocytosis of apoptotic cells. Remarkably, hTau;Trem2(-/-) exposed to TBI failed to restore blood-brain barrier integrity. These findings imply that TREM2 deficiency accelerates inflammation and neurodegeneration, accompanied by attenuated microglial phagocytosis and continuous blood-brain barrier (BBB) leakage, thus exacerbating tauopathy in hTau TBI mice.

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FX This work was supported by the Department of Defense grant

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NR 57

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Z9 1

U1 0

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PU FRONTIERS MEDIA SA

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WC Immunology

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PT J

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TI Traumatic Brain Injury-Mediated Neuroinflammation and Neurological

Deficits are Improved by 8-Methoxypsoralen Through Modulating

PPARγ/NF-κB Pathway

SO NEUROCHEMICAL RESEARCH

LA English

DT Article; Early Access

DE 8-Methoxypsoralen; Traumatic brain injury; microglia; inflammation; PPAR

gamma; NF-kappa B pathway

ID ACTIVATED RECEPTOR-GAMMA; TRANSPORT; BARRIER; CELLS; STAT3; MODEL

AB 8-Methoxypsoralen (8-MOP) has anti-inflammatory, antioxidant and tissue-repairing abilities. Here, we probed the function and mechanism of 8-MOP in traumatic brain injury (TBI). The in-vivo TBI model was constructed in Sprague-Dawley (SD) rats using controlled cortical impact (CCI) surgery. In parallel, BV2 microglia and HT22 neurons were activated by lipopolysaccharide (LPS) to establish an in-vitro model. The modified neurological score (mNSS) and the Morris water maze experiment were employed to evaluate the rats' neurological functions. The rats' brain edema was assessed by the dry and wet method, and neuronal apoptosis in damaged brain tissues was monitored by terminal deoxynucleotidyl transferase-mediated dUTP-biotin nick end labeling (TUNEL) and Nissl's staining. Immunohistochemistry (IHC) was applied to verify Iba1-microglial activation in brain lesions of rats. The expression of inflammatory cytokines in BV2 microglia and HT22 neurons in the injured lesion of TBI rats was examined by the enzyme-linked immunosorbent assay (ELISA). The levels of iNOS, COX2, TLR4, PPAR gamma, STAT3, and NF-kappa B in brain lesions, BV2 microglia and HT22 neurons were compared by Western blot. As a result, 8-MOP administration reduced inflammation and LPS-induced neuronal damage in BV2 microglia. In vivo, 8-MOP treatment relieved neurological deficits in TBI rats, improved cognitive, learning and motor functions and mitigated brain edema and neuroinflammation induced by TBI. Furthermore, LPS or TBI activated the NF-kappa B and STAT3 pathways and repressed the PPAR gamma expression. However, 8-MOP treatment attenuated NF-kappa B and STAT3 phosphorylation and elevated PPAR gamma levels. Hence, 8-MOP exerts neuroprotective and anti-inflammatory effects in TBI rats by modulating the PPAR gamma/NF-kappa B pathway.

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PT J

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Shaughness, M

Collier, S

Hopkins, D

Byrnes, KR

AF Smith, Austin N.

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Hopkins, Deanna

Byrnes, Kimberly R.

TI Therapeutic targeting of microglia mediated oxidative stress after

neurotrauma

SO FRONTIERS IN MEDICINE

LA English

DT Review

DE iron; microglia; mitochondria; NADPH oxidase; oxidative stress; spinal

cord injury; traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; SPINAL-CORD-INJURY; LIPOPOLYSACCHARIDE-INDUCED

NEUROTOXICITY; NADPH OXIDASE 2; SUPEROXIDE-PRODUCTION; RAT MODEL;

TERT-BUTYLHYDROQUINONE; INFLAMMATORY RESPONSE; ELECTRON-TRANSPORT;

NEURONAL DEATH

AB Inflammation is a primary component of the central nervous system injury response. Traumatic brain and spinal cord injury are characterized by a pronounced microglial response to damage, including alterations in microglial morphology and increased production of reactive oxygen species (ROS). The acute activity of microglia may be beneficial to recovery, but continued inflammation and ROS production is deleterious to the health and function of other cells. Microglial nicotinamide adenine dinucleotide phosphate (NADPH) oxidase (NOX), mitochondria, and changes in iron levels are three of the most common sources of ROS. All three play a significant role in post-traumatic brain and spinal cord injury ROS production and the resultant oxidative stress. This review will evaluate the current state of therapeutics used to target these avenues of microglia-mediated oxidative stress after injury and suggest avenues for future research.

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TI Fetuin-A alleviates neuroinflammation against traumatic brain

injury-induced microglial necroptosis by regulating Nrf-2/HO-1 pathway

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DE Traumatic brain injury (TBI); Neuroinflammation; Fetuin-A; Microglia;

Necroptosis; Oxidative stress; Nrf-2; HO-1 pathway

ID CELL-DEATH; PROTEIN; PHENOTYPE; APOPTOSIS; THERAPY

AB Background The microglia-mediated inflammatory response is a vital mechanism of secondary damage following traumatic brain injury (TBI), but the underlying mechanism of microglial activation is unclear. Methods Controlled cortical impact (CCI) was induced in adult male C57BL/6J mice, and glutamate was used to construct a classical in vitro injury model in the primary microglia. Microglial activation was determined by western blot and immunostaining. The inflammatory factors were measured by enzyme-linked immunosorbent assay. The oxidative stress marker and mitochondrial reactive oxygen species (ROS) were measured by immunoblotting and MitoSox Red staining. Transmission electron microscopy was used to observe the typical morphology of necroptotic cells. Results Our quantitative proteomics identified 2499 proteins; 157 were significantly differentially expressed in brain tissue between the 6 h after CCI (CCI6h) group and sham group, and 109 were significantly differentially expressed between the CCI24h and sham groups. Moreover, compared with the sham group, the terms "acute-phase response", "inflammation", and "protein binding" were significantly enriched in CCI groups. Fetuin-A, a liver-secreted acute-phase glycoprotein, was involved in these biological processes. Using an experimental TBI model, we found that the Fetuin-A level peaked at 6 h and then decreased gradually. Importantly, we showed that administration of Fetuin-A reduced the cortical lesion volume and edema area and inhibited the inflammatory response, which was associated with suppressing microglial necroptosis, thus decreasing microglial activation. Furthermore, administration of Fetuin-A attenuated mitochondrial oxidative stress in glutamate-treated microglial cells, which is a critical mechanism of necroptosis suppression. In addition, we demonstrated that Fetuin-A treatment promoted translocation of nuclear factor erythroid 2-related factor 2 (Nrf-2) from the cytoplasm to the nucleus in vivo; however, the Nrf-2 inhibitor ML385 and si-heme oxygenase-1 (si-HO-1) disrupted the regulation of oxidative stress by Fetuin-A and induced increased ROS levels and necroptosis in glutamate-treated microglial cells. Fetuin-A also protected neurons from adverse factors in vivo and in vitro. Conclusions Our results demonstrated that Fetuin-A activated Nrf-2/HO-1, suppressed oxidative stress and necroptosis levels, and thereby attenuates the abnormal inflammatory response following TBI. The findings suggest a potential therapeutic strategy for TBI treatment.

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TI Neuronal nuclear calcium signaling suppression of microglial reactivity

is mediated by osteoprotegerin after traumatic brain injury

SO JOURNAL OF NEUROINFLAMMATION

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DT Article

DE Traumatic brain injury; Microglia; Nuclear calcium; Osteoprotegerin;

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AB Background Traumatic brain injury (TBI) is characterized by massive changes in neuronal excitation, from acute excitotoxicity to chronic hyper- or hypoexcitability. Nuclear calcium signaling pathways are involved in translating changes in synaptic inputs and neuronal activity into discrete transcriptional programs which not only affect neuronal survival and synaptic integrity, but also the crosstalk between neurons and glial cells. Here, we report the effects of blunting neuronal nuclear calcium signals in the context of TBI. Methods We used AAV vectors to express the genetically encoded and nuclear-targeted calcium buffer parvalbumin (PV.NLS.mCherry) or the calcium/calmodulin buffer CaMBP4.mCherry in neurons only. Upon TBI, the extent of neuroinflammation, neuronal death and synaptic loss were assessed by immunohistochemistry and targeted transcriptome analysis. Modulation of the overall level of neuronal activity was achieved by PSAM/PSEM chemogenetics targeted to parvalbumin interneurons. The functional impact of neuronal nuclear calcium buffering in TBI was assessed by quantification of spontaneous whisking. Results Buffering neuronal nuclear calcium unexpectedly resulted in a massive and long-lasting increase in the recruitment of reactive microglia to the injury site, which was characterized by a disease-associated and phagocytic phenotype. This effect was accompanied by a substantial surge in synaptic loss and significantly reduced whisking activity. Transcriptome analysis revealed a complex effect of TBI in the context of neuronal nuclear calcium buffering, with upregulation of complement factors, chemokines and interferon-response genes, as well as the downregulation of synaptic genes and epigenetic regulators compared to control conditions. Notably, nuclear calcium buffering led to a substantial loss in neuronal osteoprotegerin (OPG), whereas stimulation of neuronal firing induced OPG expression. Viral re-expression of OPG resulted in decreased microglial recruitment and synaptic loss. OPG upregulation was also observed in the CSF of human TBI patients, underscoring its translational value. Conclusion Neuronal nuclear calcium signals regulate the degree of microglial recruitment and reactivity upon TBI via, among others, osteoprotegerin signals. Our findings support a model whereby neuronal activity altered after TBI exerts a powerful impact on the neuroinflammatory cascade, which in turn contributes to the overall loss of synapses and functional impairment.

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TI Dexmedetomidine Alters the Inflammatory Profile of Rat Microglia In

Vitro

SO NEUROCRITICAL CARE

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DE Dexmedetomidine; Microglia; Neuroinflammation; Traumatic brain injury

ID ACTIVATED MICROGLIA; MICROGLIA/MACROPHAGES; NEUROINFLAMMATION; PATIENT;

IL-10

AB Background Microglia are a primary mediator of the neuroinflammatory response to neurologic injury, such as that in traumatic brain injury. Their response includes changes to their cytokine expression, metabolic profile, and immunophenotype. Dexmedetomidine (DEX) is an alpha(2) adrenergic agonist used as a sedative in critically ill patients, such as those with traumatic brain injury. Given its pharmacologic properties, DEX may alter the phenotype of inflammatory microglia. Methods Primary microglia were isolated from Sprague-Dawley rats and cultured. Microglia were activated using multiple mediators: lipopolysaccharide (LPS), polyinosinic-polycytidylic acid (Poly I:C), and traumatic brain injury damage-associated molecular patterns (DAMP) from a rat that sustained a prior controlled cortical impact injury. After activation, cultures were treated with DEX. At the 24-h interval, the cell supernatant and cells were collected for the following studies: cytokine expression (tumor necrosis factor-alpha [TNF alpha], interleukin-10 [IL-10]) via enzyme-linked immunosorbent assay, 6-phosphofructokinase enzyme activity assay, and immunophenotype profiling with flow cytometry. Cytokine expression and metabolic enzyme activity data were analyzed using two-way analysis of variance. Cell surface marker expression was analyzed using FlowJo software. Results In LPS-treated cultures, DEX treatment decreased the expression of TNF alpha from microglia (mean difference = 121.5 +/- 15.96 pg/mL; p < 0.0001). Overall, DEX-treated cultures had a lower expression of IL-10 than nontreated cultures (mean difference = 39.33 +/- 14.50 pg/mL, p < 0.0001). DEX decreased IL-10 expression in LPS-stimulated microglia (mean difference = 74.93 +/- 12.50 pg/mL, p = 0.0039) and Poly I:C-stimulated microglia (mean difference = 23.27 +/- 6.405 pg/mL, p = 0.0221). In DAMP-stimulated microglia, DEX decreased the activity of 6-phosphofructokinase (mean difference = 18.79 +/- 6.508 units/mL; p = 0.0421). The microglial immunophenotype was altered to varying degrees with different inflammatory stimuli and DEX treatment. Conclusions DEX may alter the neuroinflammatory response of microglia. By altering the microglial profile, DEX may affect the progression of neurologic injury.

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TI TDP-43 condensates and lipid droplets regulate the reactivity of

microglia and regeneration after traumatic brain injury

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AB Decreasing the activation of pathology-activated microglia is crucial to prevent chronic inflammation and tissue scarring. In this study, we used a stab wound injury model in zebrafish and identified an injury-induced microglial state characterized by the accumulation of lipid droplets and TAR DNA-binding protein of 43 kDa (TDP-43)(+) condensates. Granulin-mediated clearance of both lipid droplets and TDP-43(+) condensates was necessary and sufficient to promote the return of microglia back to the basal state and achieve scarless regeneration. Moreover, in postmortem cortical brain tissues from patients with traumatic brain injury, the extent of microglial activation correlated with the accumulation of lipid droplets and TDP-43(+) condensates. Together, our results reveal a mechanism required for restoring microglia to a nonactivated state after injury, which has potential for new therapeutic applications in humans.

Zambusi, Novoselc et al. show that granulin-mediated clearance of cytoplasmic TDP-43(+) condensates and lipid droplets in injury-activated microglia is required for their return to the homeostatic state and successful brain regeneration.

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TI Amplified Gliosis and Interferon-Associated Inflammation in the Aging

Brain following Diffuse Traumatic Brain Injury

SO JOURNAL OF NEUROSCIENCE

LA English

DT Article

DE aging; interferon; microglia; neuroinflammation; STING; traumatic brain

injury

ID COGNITIVE DEFICITS; ALZHEIMERS-DISEASE; ACTIVATION; MICROGLIA; GAMMA;

ROD; NEUROINFLAMMATION; AGE

AB Traumatic brain injury (TBI) is associated with chronic psychiatric complications and increased risk for development of neurodegenera-tive pathology. Aged individuals account for most TBI-related hospitalizations and deaths. Nonetheless, neurobiological mechanisms that underlie worsened functional outcomes after TBI in the elderly remain unclear. Therefore, this study aimed to identify pathways that govern differential responses to TBI with age. Here, adult (2 months of age) and aged (16-18 months of age) male C57BL/6 mice were subjected to diffuse brain injury (midline fluid percussion), and cognition, gliosis, and neuroinflammation were determined 7 or 30 d postinjury (dpi). Cognitive impairment was evident 7 dpi, independent of age. There was enhanced morphologic restructuring of micro-glia and astrocytes 7 dpi in the cortex and hippocampus of aged mice compared with adults. Transcriptional analysis revealed robust age-dependent amplification of cytokine/chemokine, complement, innate immune, and interferon-associated inflammatory gene expression in the cortex 7 dpi. Ingenuity pathway analysis of the transcriptional data showed that type I interferon (IFN) signaling was significantly enhanced in the aged brain after TBI compared with adults. Age prolonged inflammatory signaling and microgliosis 30 dpi with an increased presence of rod microglia. Based on these results, a STING (stimulator of interferon genes) agonist, DMXAA, was used to determine whether augmenting IFN signaling worsened cortical inflammation and gliosis after TBI. DMXAA-treated Adult-TBI mice showed comparable expression of myriad genes that were overexpressed in the cortex of Aged-TBI mice, including Irf7, Clec7a, Cxcl10, and Ccl5. Overall, diffuse TBI promoted amplified IFN signaling in aged mice, resulting in extended inflammation and gliosis.

C1 [Wangler, Lynde M.; Bray, Chelsea E.; Packer, Jonathan M.; Tapp, Zoe M.; Davis, Amara C.; O'Neil, Shane M.; Baetz, Kara; Ouvina, Michelle; Witzel, Mollie; Godbout, Jonathan P.] Ohio State Univ, Wexner Med Ctr, Dept Neurosci, Columbus, OH 43210 USA.

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TI Exosomes derived from bone marrow mesenchymal stem cells inhibit

neuroinflammation after traumatic brain injury

SO NEURAL REGENERATION RESEARCH

LA English

DT Article

DE apoptosis; bone marrow mesenchymal stem cells; BV2 microglia; exosome;

interleukin 10; lentiviral transfection; microRNA-181b;

neuroinflammation; phenotype; signal transducer and activator of

transcription 3; traumatic brain injury

ID EXTRACELLULAR VESICLES; STROMAL CELLS; NEUROVASCULAR PLASTICITY;

FUNCTIONAL RECOVERY; MACROPHAGES; MICROGLIA; MICROENVIRONMENT; DELIVERY;

SYSTEM

AB Exosomes derived from bone marrow mesenchymal stem cells can inhibit neuroinflammation through regulating microglial phenotypes and promoting nerve injury repair. However, the underlying molecular mechanism remains unclear. In this study, we investigated the mechanism by which exosomes derived from bone marrow mesenchymal stem cells inhibit neuroinflammation. Our in vitro co-culture experiments showed that bone marrow mesenchymal stem cells and their exosomes promoted the polarization of activated BV2 microglia to their anti-inflammatory phenotype, inhibited the expression of proinflammatory cytokines, and increased the expression of anti-inflammatory cytokines. Our in vivo experiments showed that tail vein injection of exosomes reduced cell apoptosis in cortical tissue of mouse models of traumatic brain injury, inhibited neuroinflammation, and promoted the transformation of microglia to the anti-inflammatory phenotype. We screened some microRNAs related to neuroinflammation using microRNA sequencing and found that microRNA-181b seemed to be actively involved in the process. Finally, we regulated the expression of miR181b in the brain tissue of mouse models of traumatic brain injury using lentiviral transfection. We found that miR181b overexpression effectively reduced apoptosis and neuroinflamatory response after traumatic brain injury and promoted the transformation of microglia to the anti-inflammatory phenotype. The interleukin 10/STAT3 pathway was activated during this process. These findings suggest that the inhibitory effects of exosomes derived from bone marrow mesenchymal stem cells on neuroinflamation after traumatic brain injury may be realized by the action of miR181b on the interleukin 10/STAT3 pathway.

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TI Dental stem cell-derived extracellular vesicles transfer miR-330-5p to

treat traumatic brain injury by regulating microglia polarization

SO INTERNATIONAL JOURNAL OF ORAL SCIENCE

LA English

DT Article

ID INHIBITION; PROTEIN; ORIGIN

AB Traumatic brain injury (TBI) contributes to the key causative elements of neurological deficits. However, no effective therapeutics have been developed yet. In our previous work, extracellular vesicles (EVs) secreted by stem cells from human exfoliated deciduous teeth (SHED) offered new insights as potential strategies for functional recovery of TBI. The current study aims to elucidate the mechanism of action, providing novel therapeutic targets for future clinical interventions. With the miRNA array performed and Real-time PCR validated, we revealed the crucial function of miR-330-5p transferred by SHED-derived EVs (SHED-EVs) in regulating microglia, the critical immune modulator in central nervous system. MiR-330-5p targeted Ehmt2 and mediated the transcription of CXCL14 to promote M2 microglia polarization and inhibit M1 polarization. Identified in our in vivo data, SHED-EVs and their effector miR-330-5p alleviated the secretion of inflammatory cytokines and resumed the motor functional recovery of TBI rats. In summary, by transferring miR-330-5p, SHED-EVs favored anti-inflammatory microglia polarization through Ehmt2 mediated CXCL14 transcription in treating traumatic brain injury.

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TI Intranasally administered human MSC-derived extracellular vesicles

inhibit NLRP3-p38/MAPK signaling after TBI and prevent chronic brain

dysfunction

SO BRAIN BEHAVIOR AND IMMUNITY

LA English

DT Article; Early Access

DE Human mesenchymal stem cell -derived extra; cellular vesicles;

Interleukin-18; Traumatic brain injury; Mitogen-activated protein kinase

signaling; NLRP3 inflammasomes; Proinflammatory microglia

ID SPINAL-CORD-INJURY; NLRP3 INFLAMMASOME; NEUROLOGICAL OUTCOMES; DENTATE

GYRUS; MURINE MODEL; OLD-AGE; NEUROINFLAMMATION; NEUROGENESIS;

ACTIVATION; MEMORY

AB Traumatic brain injury (TBI) leads to lasting brain dysfunction with chronic neuroinflammation typified by nucleotide-binding domain leucine-rich repeat and pyrin domain-containing receptor 3 (NLRP3) inflammasome activation in microglia. This study probed whether a single intranasal (IN) administration of human mesen-chymal stem cell-derived extracellular vesicles (hMSC-EVs) naturally enriched with activated microglia-modulating miRNAs can avert chronic adverse outcomes of TBI. Small RNA sequencing confirmed the enrich-ment of miRNAs capable of modulating activated microglia in hMSC-EV cargo. IN administration of hMSC-EVs into adult mice ninety minutes after the induction of a unilateral controlled cortical impact injury resulted in their incorporation into neurons and microglia in both injured and contralateral hemispheres. A single higher dose hMSC-EV treatment also inhibited NLRP3 inflammasome activation after TBI, evidenced by reduced NLRP3, apoptosis-associated speck-like protein containing a CARD, activated caspase-1, interleukin-1 beta, and IL-18 levels in the injured brain. Such inhibition in the acute phase of TBI endured in the chronic phase, which could also be gleaned from diminished NLRP3 inflammasome activation in microglia of TBI mice receiving hMSC-EVs. Proteomic analysis and validation revealed that higher dose hMSC-EV treatment thwarted the chronic activation of the p38 mitogen-activated protein kinase (MAPK) signaling pathway by IL-18, which decreased the release of proinflammatory cytokines. Inhibition of the chronic activation of NLRP3-p38/MAPK signaling after TBI also prevented long-term cognitive and mood impairments. Notably, the animals receiving higher doses of hMSC-EVs after TBI displayed better cognitive and mood function in all behavioral tests than animals receiving the vehicle after TBI. A lower dose of hMSC-EV treatment also partially improved cognitive and mood function. Thus, an optimal IN dose of hMSC-EVs naturally enriched with activated microglia-modulating miRNAs can inhibit the chronic activation of NLRP3-p38/MAPK signaling after TBI and prevent lasting brain dysfunction.

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TI Temporal changes in the microglial proteome of male and female mice

after a diffuse brain injury using label-free quantitative proteomics

SO GLIA

LA English

DT Article; Early Access

DE biological sex; inflammation; microglia; proteomics; TBI; traumatic

brain injury

ID INFLAMMATORY RESPONSE; ACTIVATION; RECEPTOR; CELLS; AGE

AB Traumatic brain injury (TBI) triggers neuroinflammatory cascades mediated by microglia, which promotes tissue repair in the short-term. These cascades may exacerbate TBI-induced tissue damage and symptoms in the months to years post-injury. However, the progression of the microglial function across time post-injury and whether this differs between biological sexes is not well understood. In this study, we examined the microglial proteome at 3-, 7-, or 28-days after a midline fluid percussion injury (mFPI) in male and female mice using label-free quantitative proteomics. Data are available via ProteomeXchange with identifier PXD033628. We identified a reduction in microglial proteins involved with clearance of neuronal debris via phagocytosis at 3- and 7-days post-injury. At 28 days post-injury, pro-inflammatory proteins were decreased and anti-inflammatory proteins were increased in microglia. These results indicate a reduction in microglial clearance of neuronal debris in the days post-injury with a shift to anti-inflammatory function by 28 days following TBI. The changes in the microglial proteome that occurred across time post-injury did not differ between biological sexes. However, we did identify an increase in microglial proteins related to pro-inflammation and phagocytosis as well as insulin and estrogen signaling in males compared with female mice that occurred with or without a brain injury. Although the microglial response was similar between males and females up to 28 days following TBI, biological sex differences in the microglial proteome, regardless of TBI, has implications for the efficacy of treatment strategies targeting the microglial response post-injury.

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TI Lipocalin-2 Is a Key Regulator of Neuroinflammation in Secondary

Traumatic and Ischemic Brain Injury

SO NEUROTHERAPEUTICS

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DE Astrocyte; Microglia; Neuroinflammation; Lipocalin-2; Traumatic brain

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ID GELATINASE-ASSOCIATED LIPOCALIN; NEUROTROPHIC FACTOR; UP-REGULATION;

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AB Reactive glial cells are hallmarks of brain injury. However, whether these cells contribute to secondary inflammatory pathology and neurological deficits remains poorly understood. Lipocalin-2 (LCN2) has inflammatory and neurotoxic effects in various disease models; however, its pathogenic role in traumatic brain injury remains unknown. The aim of the present study was to investigate the expression of LCN2 and its role in neuroinflammation following brain injury. LCN2 expression was high in the mouse brain after controlled cortical impact (CCI) and photothrombotic stroke (PTS) injury. Brain levels of LCN2 mRNA and protein were also significantly higher in patients with chronic traumatic encephalopathy (CTE) than in normal subjects. RT-PCR and immunofluorescence analyses revealed that astrocytes were the major cellular source of LCN2 in the injured brain. Lcn2 deficiency or intracisternal injection of an LCN2 neutralizing antibody reduced CCI- and PTS-induced brain lesions, behavioral deficits, and neuroinflammation. Mechanistically, in cultured glial cells, recombinant LCN2 protein enhanced scratch injury-induced proinflammatory cytokine gene expression and inhibited Gdnf gene expression, whereas Lcn2 deficiency exerted opposite effects. Together, our results from CTE patients, rodent brain injury models, and cultured glial cells suggest that LCN2 mediates secondary damage response to traumatic and ischemic brain injury by promoting neuroinflammation and suppressing the expression of neurotropic factors.

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TI MiR-124 Reduced Neuroinflammation after Traumatic Brain Injury by

Inhibiting TRAF6

SO NEUROIMMUNOMODULATION

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DT Article

DE Traumatic brain injury; MiR-124; Microglia; Neuroinflammation; TRAF6;

TLR4

ID TOLL-LIKE RECEPTORS; M2 MICROGLIA; POLARIZATION; ACTIVATION; SYSTEM;

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AB Introduction: Neuroinflammation contributes to secondary injury after traumatic brain injury (TBI), which has been mainly mediated by the microglia. MiR-124 was reported to play an important role in the polarization of microglia by targeting TLR4 signaling pathway. However, the role and mechanism of miR-124 in neuroinflammation mediated by microglia after TBI is unclear. To clarify this, we performed this research. Methods: The expression of miR-124 was first measured by RT-PCR in the injured brain at 1/3/7 days post-TBI. Then, miR-124 mimics or inhibitors administration was used to interfere the expression of miR-124 at 24 h post-TBI. Subsequently, the microglia polarization markers were detected by RT-PCR, the expression of inflammatory cytokines was detected by ELISA, the expression of TLR4/MyD88/IRAK1/TRAF6/NF-kappa B was measured by WB, and the neurological deficit was evaluated by NSS and MWM test. At last, in vitro experiments were performed to explore the exact target molecule of miR-124 on TLR4 signaling pathway. Results: Animal research indicated that the expression of miR-124 was downregulated after TBI. Upregulation of miR-124 promoted the M2 polarization of microglia and inhibited the activity of TLR4 pathway, as well as reduced neuroinflammation and neurological deficit after TBI. In vitro experiments indicated that miR-124 promoted the M2 polarization of microglia and reduced neuroinflammation by inhibiting TRAF6. Conclusion: This study demonstrated that upregulation of miR-124 promoted the M2 polarization of microglia and reduced neuroinflammation after TBI by inhibiting TRAF6.

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TI Astrocytic Neuroimmunological Roles Interacting with Microglial Cells in

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DT Review

DE astrocyte; blood-brain barrier; 2ccPA; cPA; extracellular matrix;

microglia; neurodegenerative disease; neuroinflammation; reactive

astrocytes; reactive microglia; TN-C; traumatic brain injury

ID CYCLIC PHOSPHATIDIC-ACID; BLOOD-BRAIN-BARRIER; CENTRAL-NERVOUS-SYSTEM;

REACTIVE ASTROCYTES; TENASCIN-C; EXTRACELLULAR-MATRIX;

LYSOPHOSPHOLIPASE-D; NEURONAL DEATH; PPAR-GAMMA; EXPRESSION

AB Both astrocytic and microglial functions have been extensively investigated in healthy subjects and neurodegenerative diseases. For astrocytes, not only various sub-types were identified but phagocytic activity was also clarified recently and is making dramatic progress. In this review paper, we mostly focus on the functional role of astrocytes in the extracellular matrix and on interactions between reactive astrocytes and reactive microglia in normal states and in neurodegenerative diseases, because the authors feel it is necessary to elucidate the mechanisms among activated glial cells in the pathology of neurological diseases in order to pave the way for drug discovery. Finally, we will review cyclic phosphatidic acid (cPA), a naturally occurring phospholipid mediator that induces a variety of biological activities in the brain both in vivo and in vitro. We propose that cPA may serve as a novel therapeutic molecule for the treatment of brain injury and neuroinflammation.

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Z9 12

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Wang, Y

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Zhou, Y

Zhang, S

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Zhang, Jian-Ning

TI Maraviroc promotes recovery from traumatic brain injury in mice by

suppression of neuroinflammation and activation of neurotoxic reactive

astrocytes

SO NEURAL REGENERATION RESEARCH

LA English

DT Article

DE C-C chemokine receptor type 5 (CCR5) antagonist; high mobility group

protein B1 (HMGB1); maraviroc; M1 microglia; nuclear factor-kappa B

pathway; NACHT, LRR, and PYD domains-containing protein 3 (NLRP3)

inflammasome; neuroinflammation; neurological function; neurotoxic

reactive astrocytes; traumatic brain injury

ID NLRP3 INFLAMMASOME; CCR5; MICROGLIA; DEFICITS; MODEL

AB Neuroinflammation and the NACHT, LRR, and PYD domains-containing protein 3 inflammasome play crucial roles in secondary tissue damage following an initial insult in patients with traumatic brain injury (TBI). Maraviroc, a C-C chemokine receptor type 5 antagonist, has been viewed as a new therapeutic strategy for many neuroinflammatory diseases. We studied the effect of maraviroc on TBI-induced neuroinflammation. A moderate-TBI mouse model was subjected to a controlled cortical impact device. Maraviroc or vehicle was injected intraperitoneally 1 hour after TBI and then once per day for 3 consecutive days. Western blot, immunohistochemistry, and TUNEL (terminal deoxynucleotidyl transferase-mediated dUTP nick-end labeling) analyses were performed to evaluate the molecular mechanisms of maraviroc at 3 days post-TBI. Our results suggest that maraviroc administration reduced NACHT, LRR, and PYD domains-containing protein 3 inflammasome activation, modulated microglial polarization from M1 to M2, decreased neutrophil and macrophage infiltration, and inhibited the release of inflammatory factors after TBI. Moreover, maraviroc treatment decreased the activation of neurotoxic reactive astrocytes, which, in turn, exacerbated neuronal cell death. Additionally, we confirmed the neuroprotective effect of maraviroc using the modified neurological severity score, rotarod test, Morris water maze test, and lesion volume measurements. In summary, our findings indicate that maraviroc might be a desirable pharmacotherapeutic strategy for TBI, and C-C chemokine receptor type 5 might be a promising pharmacotherapeutic target to improve recovery after TBI.

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Innovative Drugs and Medical Devices, No. 19ZXYXSY00070 (to YW) and the

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Friess, SH

AF Celorrio, Marta

Shumilov, Kirill

Rodgers, Rachel

Schriefer, Lawrence

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Baldridge, Megan T.

Friess, Stuart H.

TI Innate and Peripheral Immune Alterations after Traumatic Brain Injury

Are Regulated in a Gut Microbiota-Dependent Manner in Mice

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE germ-free mice; gut microbiome; microglia; neurogenesis; T cells;

traumatic brain injury

ID NEUROGENESIS; MICROGLIA

AB Traumatic brain injury (TBI) patients are at high risk for disruption of the gut microbiome. Previously, we have demonstrated that broad-spectrum antibiotic exposure after TBI drastically alters the gut microbiota and modulates neuroinflammation, neurogenesis, and long-term fear memory. However, these data did not determine if the impact of antibiotic exposure on the brain's response to injury was mediated directly by antibiotics or indirectly via modulation of the gut microbiota. We designed two different approaches to address this knowledge gap. One was utilizing fecal microbiota transplantation (FMT) from control and antibiotic-treated mice (treated with vancomycin, neomycin, ampicillin, and metronidazole [VNAM]) into germ-free (GF) mice prior to injury, and the other was exposing specific pathogen-free (SPF) mice to a 2-week period of antibiotics prior to injury but discontinuing antibiotics 72 h prior to injury. GF mice receiving FMT from VNAM-treated mice (GF-VNAM) demonstrated reduced gut bacterial alpha diversity and richness compared with GF mice receiving control FMT. At 7 days post-injury, GF-VNAM had increased microglial activation, reduced infiltration of T cells, and decreased neurogenesis. Similarly, SPF mice exposed to antibiotics prior to but not after injury demonstrated similar alterations in neuroinflammation and neurogenesis compared with control mice. These data support our hypothesis implicating the gut microbiota as an important modulator of the neuroinflammatory process and neurogenesis after TBI and provide an exciting new approach for neuroprotective therapeutics for TBI.

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OI Baldridge, Megan/0000-0002-7030-6131;

FU National Institutes of Health [R01NS097721]; Washington University

Schoolof Medicine; Children's Discovery Institute of Wash-ington

University; St. Louis Children's Hospital [CDI-CORE-2015-505,

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NR 36

TC 4

Z9 4

U1 1

U2 7

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TI Multiparity Differentially Affects Specific Aspects of the Acute

Neuroinflammatory Response to Traumatic Brain Injury in Female Mice

SO NEUROSCIENCE

LA English

DT Article

DE pregnancy; TBI; blood brain barrier; microglia; astrocytes; leukocytes

ID CEREBRAL-ISCHEMIA; GLIAL-CELLS; PREGNANCY; NEURODEGENERATION;

NEUROPROTECTION; ACTIVATION; PROLACTIN; BEHAVIOR; BARRIER; WATER

AB is associated with profound acute and long-term physiological changes, but the effects of such changes on brain injury outcomes are unclear. Here, we examined the effects of previous pregnancy and maternal experience (parity) on acute neuroinflammatory responses to lateral fluid percussion injury (FPI), a well-defined experimental traumatic brain injury (TBI) paradigm. Multiparous (2-3 pregnancies and motherhood experiences) and age-matched nulliparous (no previous pregnancy or motherhood experience) female mice received either FPI or sham injury and were euthanized 3 days post-injury (DPI). Increased cortical Iba1, GFAP, and CD68 immunolabeling was observed following TBI independent of parity and microglia morphology did not differ between TBI groups. However, multiparous females had fewer CD45+ cells near the site of injury com-pared to nulliparous females, which was associated with preserved aquaporin-4 polarization, suggesting that par -ity may influence leukocyte recruitment to the site of injury and maintenance of blood brain barrier permeability following TBI. Additionally, relative cortical Il6 gene expression following TBI was dependent on parity such that TBI increased Il6 expression in nulliparous, but not multiparous, mice. Together, this work suggests that repro-ductive history may influence acute neuroinflammatory outcomes following TBI in females.(c) 2022 IBRO. Published by Elsevier Ltd. All rights reserved.

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TI Inhibition of autophagy in microglia and macrophages exacerbates innate

immune responses and worsens brain injury outcomes

SO AUTOPHAGY

LA English

DT Article; Early Access

DE Autophagy; innate immunity; macrophage; microglia; neuroinflammation;

traumatic brain injury

ID INFLAMMATION; ACTIVATION; NEUROPROTECTION; NEUROINFLAMMATION;

NEURODEGENERATION; PHAGOCYTOSIS; CHALLENGES; CLEARANCE; INFECTION;

DAMAGE

AB Excessive and prolonged neuroinflammation following traumatic brain injury (TBI) contributes to long-term tissue damage and poor functional outcomes. However, the mechanisms contributing to exacerbated inflammatory responses after brain injury remain poorly understood. Our previous work showed that macroautophagy/autophagy flux is inhibited in neurons following TBI in mice and contributes to neuronal cell death. In the present study, we demonstrate that autophagy is also inhibited in activated microglia and infiltrating macrophages, and that this potentiates injury-induced neuroinflammatory responses. Macrophage/microglia-specific knockout of the essential autophagy gene Becn1 led to overall increase in neuroinflammation after TBI. In particular, we observed excessive activation of the innate immune responses, including both the type-I interferon and inflammasome pathways. Defects in microglial and macrophage autophagy following injury were associated with decreased phagocytic clearance of danger/damage-associated molecular patterns (DAMP) responsible for activation of the cellular innate immune responses. Our data also demonstrated a role for precision autophagy in targeting and degradation of innate immune pathways components, such as the NLRP3 inflammasome. Finally, inhibition of microglial/macrophage autophagy led to increased neurodegeneration and worse long-term cognitive outcomes after TBI. Conversely, increasing autophagy by treatment with rapamycin decreased inflammation and improved outcomes in wild-type mice after TBI. Overall, our work demonstrates that inhibition of autophagy in microglia and infiltrating macrophages contributes to excessive neuroinflammation following brain injury and in the long term may prevent resolution of inflammation and tissue regeneration.

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TI A combination of umbilical cord mesenchymal stem cells and

monosialotetrahexosy 1 ganglioside alleviates neuroinflammation in

traumatic brain injury

SO EXPERIMENTAL BRAIN RESEARCH

LA English

DT Article; Early Access

DE Traumatic brain injury; Umbilical cord mesenchymal stem cells;

Monosialotetrahexosy 1 ganglioside; Microglial polarization;

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ID POLARIZATION; MICROGLIA; SECRETOME; IMMUNE; PROLIFERATION; NEUROGENESIS;

INFLAMMATION; METABOLISM; MECHANISMS; EXPRESSION

AB Neuro-inflammation and activated microglia play important roles in neuron damage in the traumatic brain injury (TBI). In this study, we determined the effect of neural network reconstruction after human umbilical cord mesenchymal stem cells (UMSCs) combined with monosialotetrahexosy 1 ganglioside (GM1) transplantation and the effect on the neuro-inflammation and polarization of microglia in a rat model of TBI, which was established in male rats using a fluid percussion brain injury device. Rats survived until day 7 after TBI were randomly treated with normal control (NC), saline (NS), GM1, UMSCs, and GM1 plus UMSCs. Modified neurological severity score (mNSS) was assessed on days 7 and 14, and the brain tissue of the injured region was collected. Immunofluorescence, RT-PCR, and western blot analysis found that inhibitory neuro-inflammatory cytokines TGF-beta and CD163 protein expression levels in injured brain tissues were significantly increased in rats treated with GM1 + UMSCs, GM1, or UMSCs and were up-regulated compared to saline-treated rats. Neuro-inflammatory cytokines IL-6, COX-2 and iNOS protein expressions were down-regulated compared to rats treated with saline. The protein expression levels of NE, NF-200, MAP-2 and beta-tubulin III were increased in the injured brain tissues from rats treated with GM1 + UMSCs, or GM1 and UMSCs alone compared to those in the rats treated with NS. The protein expression levels in rats treated with GM1 plus UMSCs were most significant on day 7 following UMSC transplantation. The rats treated with GM1 plus UMSCs had the lowest mNSS compared with that in the other groups. These data suggest that UMSCs and GM1 promote neural network reconstruction and reduce the neuro-inflammation and neurodegeneration through coordinating injury local immune inflammatory microenvironment to promote the recovery of neurological functions in the TBI.

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U1 2

U2 6

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TI MICROGLIA AND INFILTRATING T-CELLS ADOPT LONG-TERM, AGE-SPECIFIC,

TRANSCRIPTIONAL CHANGES AFTER TRAUMATIC BRAIN INJURY IN MICE

SO SHOCK

LA English

DT Article

DE Microglia; T cells; single-cell RNA sequencing; aging; traumatic brain

injury

ID NEUROINFLAMMATION; NEURODEGENERATION; HETEROGENEITY; IMPAIRMENT; MEMORY;

STATES

AB Aged traumatic brain injury (TBI) patients suffer increased mortality and long-term neurocognitive and neuropsychiatric morbidity compared with younger patients. Microglia, the resident innate immune cells of the brain, are complicit in both. We hypothesized that aged microglia would fail to return to a homeostatic state after TBI and adopt a long-term injury-associated state within aged brains compared with young brains after TBI. Young and aged male C57BL/6 mice underwent TBI via controlled cortical impact versus sham injury and were sacrificed 4 months post-TBI. We used single-cell RNA sequencing to examine age-associated cellular responses after TBI. Brains were harvested, and CD45+ cells were isolated via fluorescence-activated cell sorting. cDNA libraries were prepared using the 10x Genomics Chromium Single Cell 3 ' Reagent Kit, followed by sequencing on a HiSeq 4,000 instrument and computational analyses. Post-injury, aged mice demonstrated a disparate microglial gene signature and an increase in infiltrating T cells compared with young adult mice. Notably, aged mice post-injury had a subpopulation of age-specific, immune-inflammatory microglia resembling the gene profile of neurodegenerative disease-associated microglia with enriched pathways involved in leukocyte recruitment and brain-derived neurotrophic factor signaling. Meanwhile, post-injury, aged mice demonstrated heterogeneous T-cell infiltration with gene profiles corresponding to CD8 effector memory, CD8 naive-like, CD8 early active T cells, and Th1 cells with enriched pathways, such as macromolecule synthesis. Taken together, our data showed that the aged brain had an age-specific gene signature change in both T-cell infiltrates and microglia, which may contribute to its increased vulnerability to TBI and the long-term sequelae of TBI.

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U2 0

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JI Shock

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TI Brain and spinal cord trauma: what we know about the therapeutic

potential of insulin growth factor 1 gene therapy

SO NEURAL REGENERATION RESEARCH

LA English

DT Review

DE cognitive impairments; gene therapy; hippocampus; insulin growth factor

1; microglial cells; neurodegeneration; neurogenesis; neuroinflammation;

spinal cord injury; traumatic brain injury

ID ADULT HIPPOCAMPAL NEUROGENESIS; ENDOPLASMIC-RETICULUM STRESS; NEURAL

STEM-CELLS; COGNITIVE IMPAIRMENT; INJURY; IGF-1; NEUROINFLAMMATION;

ACTIVATION; MICROGLIA; APOPTOSIS

AB Although little attention has been paid to cognitive and emotional dysfunctions observed in patients after spinal cord injury, several reports have described impairments in cognitive abilities. Our group also has contributed significantly to the study of cognitive impairments in a rat model of spinal cord injury. These findings are very significant because they demonstrate that cognitive and mood deficits are not induced by lifestyle changes, drugs of abuse, and combined medication. They are related to changes in brain structures involved in cognition and emotion, such as the hippocampus. Chronic spinal cord injury decreases neurogenesis, enhances glial reactivity leading to hippocampal neuroinflammation, and triggers cognitive deficits. These brain distal abnormalities are recently called tertiary damage. Given that there is no treatment for Tertiary Damage, insulin growth factor 1 gene therapy emerges as a good candidate. Insulin growth factor 1 gene therapy recovers neurogenesis and induces the polarization from pro-inflammatory towards anti-inflammatory microglial phenotypes, which represents a potential strategy to treat the neuroinflammation that supports tertiary damage. Insulin growth factor 1 gene therapy can be extended to other central nervous system pathologies such as traumatic brain injury where the neuroinflammatory component is crucial. Insulin growth factor 1 gene therapy could emerge as a new therapeutic strategy for treating traumatic brain injury and spinal cord injury.

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PT J

AU Basit, RH

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TI Simulating traumatic brain injury <i>in vitro</i>: developing high

throughput models to test biomaterial based therapies

SO NEURAL REGENERATION RESEARCH

LA English

DT Review

DE astroglial scar; biomaterial; cortical culture; in vitro model;

microglial infiltration; multicellular model; penetrating injury;

scaffold; traumatic brain injury

ID CELL-CULTURE; PATHWAY; ASTROCYTES; MIGRATION; NEURONS; RAT

AB Traumatic brain injuries are serious clinical incidents associated with some of the poorest outcomes in neurological practice. Coupled with the limited regenerative capacity of the brain, this has significant implications for patients, carers, and healthcare systems, and the requirement for life-long care in some cases. Clinical treatment currently focuses on limiting the initial neural damage with long-term care/support from multidisciplinary teams. Therapies targeting neuroprotection and neural regeneration are not currently available but are the focus of intensive research. Biomaterial-based interventions are gaining popularity for a range of applications including biomolecule and drug delivery, and to function as cellular scaffolds. Experimental investigations into the development of such novel therapeutics for traumatic brain injury will be critically underpinned by the availability of appropriate high throughput, facile, ethically viable, and pathomimetic biological model systems. This represents a significant challenge for researchers given the pathological complexity of traumatic brain injury. Specifically, there is a concerted post-injury response mounted by multiple neural cell types which includes microglial activation and astroglial scarring with the expression of a range of growth inhibitory molecules and cytokines in the lesion environment. Here, we review common models used for the study of traumatic brain injury (ranging from live animal models to in vitro systems), focusing on penetrating traumatic brain injury models. We discuss their relative advantages and drawbacks for the developmental testing of biomaterial-based therapies.

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TI Novel image analysis tool for rapid screening of cell morphology in

preclinical animal models of disease

SO HELIYON

LA English

DT Article; Early Access

DE Automated image analysis; Microglia; Morphology; Organophosphate;

Traumatic brain injury; Unbiased image analysis

ID TRAUMATIC BRAIN-INJURY; INFLAMMATORY RESPONSE; GENE-EXPRESSION;

MICROGLIA; RAT; STEREOLOGY; PROTEIN

AB The field of cell biology has seen major advances in both cellular imaging modalities and the development of automated image analysis platforms that increase rigor, reproducibility, and throughput for large imaging data sets. However, there remains a need for tools that provide accurate morphometric analysis of single cells with complex, dynamic cytoarchitecture in a high -throughput and unbiased manner. We developed a fully automated image-analysis algorithm to rapidly detect and quantify changes in cellular morphology using microglia cells, an innate im-mune cell within the central nervous system, as representative of cells that exhibit dynamic and complex cytoarchitectural changes. We used two preclinical animal models that exhibit robust changes in microglia morphology: (1) a rat model of acute organophosphate intoxication, which was used to generate fluorescently labeled images for algorithm development; and (2) a rat model of traumatic brain injury, which was used to validate the algorithm using cells labeled using chromogenic detection methods. All ex vivo brain sections were immunolabeled for IBA-1 using fluorescence or diaminobenzidine (DAB) labeling, images were acquired using a high content imaging system and analyzed using a custom-built algorithm. The exploratory data set revealed eight statistically significant and quantitative morphometric parameters that distinguished be-tween phenotypically distinct groups of microglia. Manual validation of single-cell morphology was strongly correlated with the automated analysis and was further supported by a comparison with traditional stereology methods. Existing image analysis pipelines rely on high-resolution images of individual cells, which limits sample size and is subject to selection bias. However, our fully automated method integrates quantification of morphology and fluorescent/chromo-genic signals in images from multiple brain regions acquired using high-content imaging. In summary, our free, customizable image analysis tool provides a high-throughput, unbiased method for accurately detecting and quantifying morphological changes in cells with complex morphologies.

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TI Increased level of exosomal miR-20b-5p derived from hypothermia-treated

microglia promotes neurite outgrowth and synapse recovery after

traumatic brain injury

SO NEUROBIOLOGY OF DISEASE

LA English

DT Article; Early Access

DE Exosomes; Hypothermia; Microglia; Synapse; Traumatic brain injury

ID CENTRAL-NERVOUS-SYSTEM; AXON REGENERATION; INFLAMMATION; EXPRESSION;

MICRORNAS; MILD

AB Mild hypothermia has been proven to inhibit microglia activation after TBI. Exosomal microRNA derived from microglia played a critical role in promoting neurite outgrowth and synapse recovery. Here, we aimed to investigate the role of microRNAs in microglial exosomes after hypothermia treatment on neuronal regeneration after TBI. For in vitro study, stretch-injured neurons were co-cultured with microglial exosomes. For in vivo study, C57BL/6 mice were under controlled cortical impact and injected with microglial exosomes. The results showed that MG-LPS-EXOHT increased the number of dendrite branches and total length of dendrites both in vitro and in vivo, elevated the expression levels of PSD-95 and GluR1 in stretch-injured neurons, and increased spine density in the pericontusion region. Moreover, MG-LPS-EXOHT improved motor function and motor coordination. A high-throughput sequencing showed that miR-20b-5p was upregulated in MG-LPS-EXOHT. Elevating miR-20b-5p promoted neurite outgrowth and synapse recovery of injured neurons both in vitro and in vivo. Following mechanistic study demonstrated that miR-20b-5p might promote neurite outgrowth and synapse recovery by directly targeting PTEN and activating PI3K-AKT pathway. In conclusion, mild hypothermia could modify the microRNA prolife of exosomes derived from LPS activated BV2 cells. Furthermore, high level of microglial exosomal miR-20b-5p induced by mild hypothermia could transfer into injured neurons and promote neurite outgrowth and synapse recovery after TBI via activating the PI3K-AKT pathway by suppressing PTEN expression.

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TI Extracellular Vesicles Derived From Neural Stem Cells, Astrocytes, and

Microglia as Therapeutics for Easing TBI-Induced Brain Dysfunction

SO STEM CELLS TRANSLATIONAL MEDICINE

LA English

DT Review; Early Access

DE astrocytes; astrocyte-derived extracellular vesicles; exosomes;

microglia-derived extracellular vesicles; neural stem cell-derived

extracellular vesicles; neuroinflammation; traumatic brain injury

ID TRAUMATIC BRAIN; INJURY; EXOSOMES; NEUROGENESIS; PATHOGENESIS;

INFLAMMATION; BIOGENESIS; EPILEPSY; SURVIVAL; THERAPY

AB Extracellular vesicles (EVs) derived from neural stem cells (NSC-EVs), astrocytes (ADEVs), and microglia (MDEVs) have neuroregenerative properties. This review discusses the therapeutic efficacy of NSC-EVs, ADEVs, and MDEVs in traumatic brain injury (TBI) models. The translational value and future directions for such EV therapy are also deliberated. Studies have demonstrated that NSC-EV or ADEV therapy can mediate neuroprotective effects and improve motor and cognitive function after TBI. Furthermore, NSC-EVs or ADEVs generated after priming parental cells with growth factors or brain-injury extracts can mediate improved therapeutic benefits. However, the therapeutic effects of naive MDEVs are yet to be tested rigorously in TBI models. Studies using activated MDEVs have reported both adverse and beneficial effects. NSC-EV, ADEV, or MDEV therapy for TBI is not ready for clinical translation. Rigorous testing of their efficacy for preventing chronic neuroinflammatory cascades and enduring motor and cognitive impairments after treatment in the acute phase of TBI, an exhaustive evaluation of their miRNA or protein cargo, and the effects of delayed EV administration post-TBI for reversing chronic neuroinflammation and enduring brain impairments, are needed. Moreover, the most beneficial route of administration for targeting EVs into different neural cells in the brain after TBI and the efficacy of well-characterized EVs from NSCs, astrocytes, or microglia derived from human pluripotent stem cells need to be evaluated. EV isolation methods for generating clinical-grade EVs must also be developed. Overall, NSC-EVs and ADEVs promise to mitigate TBI-induced brain dysfunction, but additional preclinical studies are needed before their clinical translation.

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TI Immune Regulatory Functions of Macrophages and Microglia in Central

Nervous System Diseases

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

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DT Review

DE multiple sclerosis; Alzheimer's disease; Parkinson's disease; spinal

cord injury; traumatic brain injury; immune response; microglia;

macrophage

ID SPINAL-CORD-INJURY; MYELIN BASIC-PROTEIN; MAJOR HISTOCOMPATIBILITY

COMPLEX; TRAUMATIC BRAIN-INJURY; AMYLOID-BETA-PROTEIN;

MULTIPLE-SCLEROSIS LESIONS; FIBRILLARY ACIDIC PROTEIN;

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AB Macrophages can be characterized as a very multifunctional cell type with a spectrum of phenotypes and functions being observed spatially and temporally in various disease states. Ample studies have now demonstrated a possible causal link between macrophage activation and the development of autoimmune disorders. How these cells may be contributing to the adaptive immune response and potentially perpetuating the progression of neurodegenerative diseases and neural injuries is not fully understood. Within this review, we hope to illustrate the role that macrophages and microglia play as initiators of adaptive immune response in various CNS diseases by offering evidence of: (1) the types of immune responses and the processes of antigen presentation in each disease, (2) receptors involved in macrophage/microglial phagocytosis of disease-related cell debris or molecules, and, finally, (3) the implications of macrophages/microglia on the pathogenesis of the diseases.

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TI The Putative Role of Neuroinflammation in the Interaction between

Traumatic Brain Injuries, Sleep, Pain and Other Neuropsychiatric

Outcomes: A State-of-the-Art Review

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DT Review

DE traumatic brain injury; headache; concussion; neuroinflammation;

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ID COGNITIVE-BEHAVIORAL THERAPY; NEURODEGENERATIVE DISEASES; MICROGLIAL

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AB Sleep disturbances are widely prevalent following a traumatic brain injury (TBI) and have the potential to contribute to numerous post-traumatic physiological, psychological, and cognitive difficulties developing chronically, including chronic pain. An important pathophysiological mechanism involved in the recovery of TBI is neuroinflammation, which leads to many downstream consequences. While neuroinflammation is a process that can be both beneficial and detrimental to individuals' recovery after sustaining a TBI, recent evidence suggests that neuroinflammation may worsen outcomes in traumatically injured patients, as well as exacerbate the deleterious consequences of sleep disturbances. Additionally, a bidirectional relationship between neuroinflammation and sleep has been described, where neuroinflammation plays a role in sleep regulation and, in turn, poor sleep promotes neuroinflammation. Given the complexity of this interplay, this review aims to clarify the role of neuroinflammation in the relationship between sleep and TBI, with an emphasis on long-term outcomes such as pain, mood disorders, cognitive dysfunctions, and elevated risk of Alzheimer's disease and dementia. In addition, some management strategies and novel treatment targeting sleep and neuroinflammation will be discussed in order to establish an effective approach to mitigate long-term outcomes after TBI.

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TI Acute intranasal treatment with nerve growth factor limits the onset of

traumatic brain injury in young rats

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AB Background and PurposeTraumatic brain injury (TBI) comprises a primary injury directly induced by impact, which progresses into a secondary injury leading to neuroinflammation, reactive astrogliosis, and cognitive and motor damage. To date, treatment of TBI consists solely of palliative therapies that do not prevent and/or limit the outcomes of secondary damage and only stabilize the deficits. The neurotrophin, nerve growth factor (NGF), delivered to the brain parenchyma following intranasal application, could be a useful means of limiting or improving the outcomes of the secondary injury, as suggested by pre-clinical and clinical data. Experimental ApproachWe evaluated the effect of acute intranasal treatment of young (20-postnatal day) rats, with NGF in a TBI model (weight drop/close head), aggravated by hypoxic complications. Immediately after the trauma, rats were intranasally treated with human recombinant NGF (50 mu g center dot kg(-1)), and motor behavioural test, morphometric and biochemical assays were carried out 24 h later. Key ResultsAcute intranasal NGF prevented the onset of TBI-induced motor disabilities, and decreased reactive astrogliosis, microglial activation and IL-1 beta content, which after TBI develops to the same extent in the impact zone and the hypothalamus. Conclusion and ImplicationsIntranasal application of NGF was effective in decreasing the motor dysfunction and neuroinflammation in the brain of young rats in our model of TBI. This work forms an initial pre-clinical evaluation of the potential of early intranasal NGF treatment in preventing and limiting the disabling outcomes of TBI, a clinical condition that remains one of the unsolved problems of paediatric neurology.

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TI Traumatic Brain Injury Induces Microglial and Caspase3 Activation in the

Retina

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DE TBI; brain; injury; microglia; caspase; apoptosis; retina; degeneration

ID MECHANISMS; MORPHOLOGY; MODEL; PATHOPHYSIOLOGY; HETEROGENEITY; DEATH

AB Traumatic brain injury (TBI) is among the main causes of sudden death after head trauma. These injuries can result in severe degeneration and neuronal cell death in the CNS, including the retina, which is a crucial part of the brain responsible for perceiving and transmitting visual information. The long-term effects of mild-repetitive TBI (rmTBI) are far less studied thus far, even though damage induced by repetitive injuries occurring in the brain is more common, especially amongst athletes. rmTBI can also have a detrimental effect on the retina and the pathophysiology of these injuries is likely to differ from severe TBI (sTBI) retinal injury. Here, we show how rmTBI and sTBI can differentially affect the retina. Our results indicate an increase in the number of activated microglial cells and Caspase3-positive cells in the retina in both traumatic models, suggesting a rise in the level of inflammation and cell death after TBI. The pattern of microglial activation appears distributed and widespread but differs amongst the various retinal layers. sTBI induced microglial activation in both the superficial and deep retinal layers. In contrast to sTBI, no significant change occurred following the repetitive mild injury in the superficial layer, only the deep layer (spanning from the inner nuclear layer to the outer plexiform layer) shows microglial activation. This difference suggests that alternate response mechanisms play a role in the case of the different TBI incidents. The Caspase3 activation pattern showed a uniform increase in both the superficial and deep layers of the retina. This suggests a different action in the course of the disease in sTBI and rmTBI models and points to the need for new diagnostic procedures. Our present results suggest that the retina might serve as such a model of head injuries since the retinal tissue reacts to both forms of TBI and is the most accessible part of the human brain.

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TI Delayed TBI-Induced Neuronal Death in the Ipsilateral Hippocampus and

Behavioral Deficits in Rats: Influence of Corticosterone-Dependent

Survivorship Bias?

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Article

DE traumatic brain injury; hippocampus; corticosterone; memory;

neurodegeneration; neuroinflammation; microglia; survivorship bias

ID PERCUSSION BRAIN-INJURY; NEUROINFLAMMATION; RELEVANCE; NUMBER

AB Acute and chronic corticosterone (CS) elevations after traumatic brain injury (TBI) may be involved in distant hippocampal damage and the development of late posttraumatic behavioral pathology. CS-dependent behavioral and morphological changes were studied 3 months after TBI induced by lateral fluid percussion in 51 male Sprague-Dawley rats. CS was measured in the background 3 and 7 days and 1, 2 and 3 months after TBI. Tests including open field, elevated plus maze, object location, new object recognition tests (NORT) and Barnes maze with reversal learning were used to assess behavioral changes in acute and late TBI periods. The elevation of CS on day 3 after TBI was accompanied by early CS-dependent objective memory impairments detected in NORT. Blood CS levels > 860 nmol/L predicted delayed mortality with an accuracy of 0.947. Ipsilateral neuronal loss in the hippocampal dentate gyrus, microgliosis in the contralateral dentate gyrus and bilateral thinning of hippocampal cell layers as well as delayed spatial memory deficits in the Barnes maze were revealed 3 months after TBI. Because only animals with moderate but not severe posttraumatic CS elevation survived, we suggest that moderate late posttraumatic morphological and behavioral deficits may be at least partially masked by CS-dependent survivorship bias.

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TI KDM4A, involved in the inflammatory and oxidative stress caused by

traumatic brain injury-hemorrhagic shock, partly through the regulation

of the microglia M1 polarization

SO BMC NEUROSCIENCE

LA English

DT Article

DE KDM4A; TBI; HS; Microglia; M1 polarization

ID NEUROINFLAMMATION; APOPTOSIS; BARRIER; MODELS; JMJD2A; TBI

AB BackgroundMicroglial polarization and the subsequent neuroinflammatory response and oxidative stress are contributing factors for traumatic brain injury (TBI) plus hemorrhagic shock (HS) induced brain injury. In the present work, we have explored whether Lysine (K)-specific demethylase 4 A (KDM4A) modulates microglia M1 polarization in the TBI and HS mice.ResultsMale C57BL/6J mice were used to investigate the microglia polarization in the TBI + HS model in vivo. Lipopolysaccharide (LPS)-induced BV2 cells were used to examine the mechanism of KDM4A in regulating microglia polarization in vitro. We found that TBI + HS resulted in neuronal loss and microglia M1 polarization in vivo, reflected by the increased level of Iba1, tumor necrosis factor (TNF)-alpha, interleukin (IL)-1 beta, malondialdehyde (MDA) and the decreased level of reduced glutathione (GSH). Additionally, KDM4A was upregulated in response to TBI + HS and microglia were among the cell types showing the increased level of KDM4A. Similar to the results in vivo, KDM4A also highly expressed in LPS-induced BV2 cells. LPS-induced BV2 cells exhibited enhanced microglia M1 polarization, and enhanced level of pro-inflammatory cytokines, oxidative stress and reactive oxygen species (ROS), while this enhancement was abolished by the suppression of KDM4A.ConclusionAccordingly, our findings indicated that KDM4A was upregulated in response to TBI + HS and microglia were among the cell types showing the increased level of KDM4A. The important role of KDM4A in TBI + HS-induced inflammatory response and oxidative stress was at least partially realized through regulating microglia M1 polarization.

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TI Understanding microglial responses in large animal models of traumatic

brain injury: an underutilized resource for preclinical and

translational research

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Review

DE Mild TBI; Neuroinflammation; Microglia; Large animal models; Preclinical

models

ID NECROSIS-FACTOR-ALPHA; DIFFUSE AXONAL INJURY; SEX-DIFFERENCES;

WHITE-MATTER; NEUROINFLAMMATORY RESPONSE; GLUTAMATE-RECEPTOR;

ACTIVATION; DEATH; INFLAMMATION; COMPLEMENT

AB Traumatic brain injury (TBI) often results in prolonged or permanent brain dysfunction with over 2.8 million affected annually in the U.S., including over 56,000 deaths, with over 5 million total survivors exhibiting chronic deficits. Mild TBI (also known as concussion) accounts for over 75% of all TBIs every year. Mild TBI is a heterogeneous disorder, and long-term outcomes are dependent on the type and severity of the initial physical event and compounded by secondary pathophysiological consequences, such as reactive astrocytosis, edema, hypoxia, excitotoxicity, and neuroinflammation. Neuroinflammation has gained increasing attention for its role in secondary injury as inflammatory pathways can have both detrimental and beneficial roles. For example, microglia-resident immune cells of the central nervous system (CNS)-influence cell death pathways and may contribute to progressive neurodegeneration but also aid in debris clearance and neuroplasticity. In this review, we will discuss the acute and chronic role of microglia after mild TBI, including critical protective responses, deleterious effects, and how these processes vary over time. These descriptions are contextualized based on interspecies variation, sex differences, and prospects for therapy. We also highlight recent work from our lab that was the first to describe microglial responses out to chronic timepoints after diffuse mild TBI in a clinically relevant large animal model. The scaled head rotational acceleration of our large animal model, paired with the gyrencephalic architecture and appropriate white:gray matter ratio, allows us to produce pathology with the same anatomical patterns and distribution of human TBI, and serves as an exemplary model to examine complex neuroimmune response post-TBI. An improved understanding of microglial influences in TBI could aid in the development of targeted therapeutics to accentuate positive effects while attenuating detrimental post-injury responses over time.

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TI Interleukin-4 mitigates anxiety-like behavior and loss of neurons and

fiber tracts in limbic structures in a microglial PPAR?-dependent manner

after traumatic brain injury

SO NEUROBIOLOGY OF DISEASE

LA English

DT Article; Early Access

DE Neural circuit; Hippocampus; Amygdala; PPAR?; Mood disorders

ID MOOD DISORDERS; STRESS; HIPPOCAMPUS; DOPAMINE; CIRCUITS; MODEL; SEX

AB Traumatic brain injury (TBI) is commonly followed by intractable psychiatric disorders and long-term changes in affect, such as anxiety. The present study sought to investigate the effect of repetitive intranasal delivery of interleukin-4 (IL-4) nanoparticles on affective symptoms after TBI in mice. Adult male C57BL/6 J mice (10-12 weeks of age) were subjected to controlled cortical impact (CCI) and assessed by a battery of neurobehavioral tests up to 35 days after CCI. Neuron numbers were counted in multiple limbic structures, and the integrity of limbic white matter tracts was evaluated using ex vivo diffusion tensor imaging (DTI). As STAT6 is a critical mediator of IL-4-specific transcriptional activation, STAT6 knockout mice were used to explore the role of endogenous IL-4/STAT6 signaling axis in TBI-induced affective disorders. We also employed microglia/macro-phage (Mi/M phi)-specific PPAR gamma conditional knockout (mKO) mice to test if Mi/M phi PPAR gamma critically contributes to IL-4-afforded beneficial effects. We observed anxiety-like behaviors up to 35 days after CCI, and these measures were exacerbated in STAT6 KO mice but mitigated by repetitive IL-4 delivery. We discovered that IL-4 protected against neuronal loss in limbic structures, such as the hippocampus and the amygdala, and improved the structural integrity of fiber tracts connecting the hippocampus and amygdala. We also observed that IL-4 boosted a beneficial Mi/M phi phenotype (CD206+/Arginase 1+/PPAR gamma+ triple-positive) in the subacute injury phase, and that the numbers of Mi/M phi appositions with neurons were robustly correlated with long-term behavioral per-formances. Remarkably, PPAR gamma-mKO completely abolished IL-4-afforded protection. Thus, CCI induces long-term anxiety-like behaviors in mice, but these changes in affect can be attenuated by transnasal IL-4 delivery. IL-4 prevents the long-term loss of neuronal somata and fiber tracts in key limbic structures, perhaps due to a shift in Mi/M phi phenotype. Exogenous IL-4 therefore holds promise for future clinical management of mood disturbances following TBI.

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TI Hemolytic iron regulation in traumatic brain injury and alcohol use

SO ALCOHOL

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DE Alcohol; Hemorrhage; Iron; Microglia; TBI

ID NEUROINFLAMMATION; TRANSFUSION; DYSFUNCTION; ACTIVATION; MICROGLIA;

DEATH

AB Hemorrhage is a major component of traumatic brain injury (TBI). Red blood cells, accumulated at the hemorrhagic site, undergo hemolysis upon energy depletion and release free iron into the central ner-vous system. This iron must be managed to prevent iron neurotoxicity and ferroptosis. As prior alcohol consumption is often associated with TBI, we examined iron regulation in a rat model of chronic alcohol feeding subjected to fluid percussion-induced TBI. We found that alcohol consumption prior to TBI altered the expression profiles of the lipocalin 2/heme oxygenase 1/ferritin iron management system. Notably, unlike TBI alone, TBI following chronic alcohol consumption sustained the expression of all three regulatory proteins for 1, 3, and 7 days post-injury. In addition, alcohol significantly affected TBI-induced expression of ferritin light chain at 3 days post-injury. We also found that alcohol exacerbated TBI-induced activation of microglia at 7 days post-injury. Finally, we propose that microglia may also play a role in iron management through red blood cell clearance.(c) 2023 Elsevier Inc. All rights reserved.

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TI Microglial activation persists beyond clinical recovery following sport

concussion in collegiate athletes

SO FRONTIERS IN NEUROLOGY

LA English

DT Article

DE sport concussion; traumatic brain injury; positron emission tomography;

neuroinflammation; molecular imaging

ID TRAUMATIC BRAIN-INJURY; PROFESSIONAL FOOTBALL; HIGH-SCHOOL; RECURRENT

CONCUSSION; WHITE-MATTER; NEUROCOGNITIVE PERFORMANCE; COGNITIVE

IMPAIRMENT; AGE-DIFFERENCES; OPTIMIZATION; REGISTRATION

AB IntroductionIn concussion, clinical and physiological recovery are increasingly recognized as diverging definitions. This study investigated whether central microglial activation persisted in participants with concussion after receiving an unrestricted return-to-play (uRTP) designation using [F-18]DPA-714 PET, an in vivo marker of microglia activation. MethodsEight (5 M, 3 F) current athletes with concussion (Group 1) and 10 (5 M, 5 F) healthy collegiate students (Group 2) were enrolled. Group 1 completed a pre-injury (Visit1) screen, follow-up Visit2 within 24 h of a concussion diagnosis, and Visit3 at the time of uRTP. Healthy participants only completed assessments at Visit2 and Visit3. At Visit2, all participants completed a multidimensional battery of tests followed by a blood draw to determine genotype and study inclusion. At Visit3, participants completed a clinical battery of tests, brain MRI, and brain PET; no imaging tests were performed outside of Visit3. ResultsFor Group 1, significant differences were observed between Visits 1 and 2 (p < 0.05) in ImPACT, SCAT5 and SOT performance, but not between Visit1 and Visit3 for standard clinical measures (all p > 0.05), reflecting clinical recovery. Despite achieving clinical recovery, PET imaging at Visit3 revealed consistently higher [F-18]DPA-714 tracer distribution volume (VT) of Group 1 compared to Group 2 in 10 brain regions (p < 0.001) analyzed from 164 regions of the whole brain, most notably within the limbic system, dorsal striatum, and medial temporal lobe. No notable differences were observed between clinical measures and VT between Group 1 and Group 2 at Visit3. DiscussionOur study is the first to demonstrate persisting microglial activation in active collegiate athletes who were diagnosed with a sport concussion and cleared for uRTP based on a clinical recovery.

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TI Fish oil fat emulsion alleviates traumatic brain injury in mice by

regulation of microglia polarization

SO NEUROSCIENCE LETTERS

LA English

DT Article

DE Traumatic brain injury; LCT; MCT fat emulsion; Fish oil fat emulsion;

Microglia; Neuroinflammation; Microglia polarization

ID OMEGA-3-FATTY-ACIDS; MODELS; IMPACT

AB Microglia activation, a hallmark of brain neuroinflammation, contributes to the secondary damage following traumatic brain injury (TBI). To explore the potential roles of different fat emulsions-long chain triglyceride (LCT) / medium chain triglyceride (MCT) and fish oil (FO) fat emulsion in neuroprotection and neuro-inflammation in TBI, in this study, we first generated the controlled cortical impact (CCI) model of TBI mice. Then either LCT/MCT or FO fat emulsion treated mice were studied by Nissl staining to assess the lesion volume. Sham and TBI mice treated with 0.9% saline were used as controls. The fatty acid composition in different TBI mouse brains was further evaluated by gas chromatography. Immunofluorescent staining and quantitative RT-PCR both demonstrated the suppression of pro-inflammatory microglia and upregulated anti-inflammatory microglia in FO fat emulsion treated TBI brain or primary microglia induced by lipopolysaccharide (LPS) in vitro. Furthermore, motor and cognitive behavioral tests showed FO fat emulsion could partially improve the motor function in TBI mice. Together, our results indicate that FO fat emulsion significantly alleviates the TBI injury and neuroinflammation probably by regulating microglia polarization.

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TI Cofilin Inhibitor Protects against Traumatic Brain Injury-Induced

Oxidative Stress and Neuroinflammation

SO BIOLOGY-BASEL

LA English

DT Article

DE cofilin inhibitor; TBI; oxidative stress; microglial activation; ROS;

Nrf2

ID PEROXIDE-INDUCED APOPTOSIS; MICROGLIAL ACTIVATION; ROS PRODUCTION; NRF2;

MITOCHONDRIAL; SLINGSHOT; NEUROTOXICITY; ANTIOXIDANT; DYSFUNCTION;

DYNAMICS

AB Simple Summary Traumatic brain injury (TBI) is a significant healthcare problem and a leading cause of death in the United States. There is a critical need to develop potential therapeutics to treat TBI-related injuries. Oxidative stress is considered a major mechanism that worsens the damage. Microglia, the first line of defense in the brain, is overactivated following injury causing the death of neuronal cells. Cofilin is a cytoskeleton protein that is activated during such brain injuries. As reported in previous in vivo studies, targeting cofilin has already been shown as a promising therapeutic strategy in other brain diseases. This study investigated the potential benefits of a new cofilin inhibitor in reducing microglial cell activation and the death of neurons in in vitro immortalized cells. We also explored this cofilin inhibitor's effect in a mouse TBI model. Following brain injury, we measured the levels of different genes and proteins in mice brains. We found that the administration of cofilin inhibitor reduced various inflammatory and oxidative markers in in vitro and in vivo mice models. Microglial activation and failure of the antioxidant defense mechanisms are major hallmarks in different brain injuries, particularly traumatic brain injury (TBI). Cofilin is a cytoskeleton-associated protein involved in actin binding and severing. In our previous studies, we identified the putative role of cofilin in mediating microglial activation and apoptosis in ischemic and hemorrhagic conditions. Others have highlighted the involvement of cofilin in ROS production and the resultant neuronal death; however, more studies are needed to delineate the role of cofilin in oxidative stress conditions. The present study aims to investigate the cellular and molecular effects of cofilin in TBI using both in vitro and in vivo models as well as the first-in-class small-molecule cofilin inhibitor (CI). An in vitro H2O2-induced oxidative stress model was used in two different types of cells, human neuroblastoma (SH-SY5Y) and microglia (HMC3), along with an in vivo controlled cortical impact model of TBI. Our results show that treatment with H2O2 increases the expression of cofilin and slingshot-1 (SSH-1), an upstream regulator of cofilin, in microglial cells, which was significantly reduced in the CI-treated group. Cofilin inhibition significantly attenuated H2O2-induced microglial activation by reducing the release of proinflammatory mediators. Furthermore, we demonstrate that CI protects against H2O2-induced ROS accumulation and neuronal cytotoxicity, activates the AKT signaling pathway by increasing its phosphorylation, and modulates mitochondrial-related apoptogenic factors. The expression of NF-E2-related factor 2 (Nrf2) and its associated antioxidant enzymes were also increased in CI-treated SY-SY5Y. In the mice model of TBI, CI significantly activated the Nrf2 and reduced the expression of oxidative/nitrosative stress markers at the protein and gene levels. Together, our data suggest that cofilin inhibition provides a neuroprotective effect in in vitro and in vivo TBI mice models by inhibiting oxidative stress and inflammatory responses, the pivotal mechanisms involved in TBI-induced brain damage.

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TI Pathophysiology and Neuroimmune Interactions Underlying Parkinson's

Disease and Traumatic Brain Injury

SO INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES

LA English

DT Review

DE alpha-synuclein; microglia; neuroinflammation; neurodegeneration;

neuromelanin; oxidative stress; Parkinson's disease; S100B; TBI

ID ALPHA-SYNUCLEIN; HEAD-INJURY; NEURODEGENERATIVE DISEASES; RISK; IRON;

AQUAPORIN-4; LINK; MUTATION; NEUROPROTECTION; CONCUSSION

AB Parkinson's disease (PD) is a progressive neurodegenerative disorder clinically defined by motor instability, bradykinesia, and resting tremors. The clinical symptomatology is seen alongside pathologic changes, most notably the loss of dopaminergic neurons in the substantia nigra pars compacta (SNpc) and the accumulation of a-synuclein and neuromelanin aggregates throughout numerous neural circuits. Traumatic brain injury (TBI) has been implicated as a risk factor for developing various neurodegenerative diseases, with the most compelling argument for the development of PD. Dopaminergic abnormalities, the accumulation of a-synuclein, and disruptions in neural homeostatic mechanisms, including but not limited to the release of pro-inflammatory mediators and the production of reactive oxygen species (ROS), are all present following TBI and are closely related to the pathologic changes seen in PD. Neuronal iron accumulation is discernable in degenerative and injured brain states, as is aquaporin-4 (APQ4). APQ4 is an essential mediator of synaptic plasticity in PD and regulates edematous states in the brain after TBI. Whether the cellular and parenchymal changes seen post-TBI directly cause neurodegenerative diseases such as PD is a point of considerable interest and debate; this review explores the vast array of neuroimmunological interactions and subsequent analogous changes that occur in TBI and PD. There is significant interest in exploring the validity of the relationship between TBI and PD, which is a focus of this review.

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TI Colony-Stimulating Factor-1 Receptor Inhibition Transiently Attenuated

the Peripheral Immune Response to Experimental Traumatic Brain Injury

SO NEUROTRAUMA REPORTS

LA English

DT Article

DE concussion; inflammation; microglia; peripheral immune response; PLX

ID MICROGLIAL-DEPLETION; R PACKAGE; NEUROINFLAMMATION; INFLAMMATION;

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AB To investigate microglial mechanisms in central and peripheral inflammation after experimental traumatic brain injury (TBI), we inhibited the colony-stimulating factor-1 receptor (CSF-1R) with PLX5622 (PLX). We hypothesized that microglia depletion would attenuate central inflammation acutely with no effect on peripheral inflammation. After randomization, male mice (n = 105) were fed PLX or control diets (21 days) and then received midline fluid percussion injury or sham injury. Brain and blood were collected at 1, 3, or 7 days post-injury (DPI). Immune cell populations were quantified in the brain and blood by flow cytometry. Cytokines (interleukin [IL]-6, IL-1 beta, tumor necrosis factor-alpha, interferon-gamma, IL-17A, and IL-10) were quantified in the blood using a multi-plex enzyme-linked immunosorbent assay. Data were analyzed using Bayesian multi-variate, multi-level models. PLX depleted microglia at all time points and reduced neutrophils in the brain at 7 DPI. PLX also depleted CD115(+) monocytes, reduced myeloid cells, neutrophils, and Ly6C(low) monocytes in blood, and elevated IL-6. TBI induced a central and peripheral immune response. TBI elevated leukocytes, microglia, and macrophages in the brain and elevated peripheral myeloid cells, neutrophils, Ly6C(int) monocytes, and IL-1 beta in the blood. TBI lowered peripheral CD115(+) and Ly6C(low) monocytes in the blood. TBI PLX mice had fewer leukocytes and microglia in the brain at 1 DPI, with elevated neutrophils at 7 DPI compared to TBI mice on a control diet. TBI PLX mice also had fewer peripheral myeloid cells, CD115(+), and Ly6C(low) monocytes in the blood at 3 DPI, but elevated Ly6C(high), Ly6C(int), and CD115(+) monocyte populations at 7 DPI, compared to TBI mice on a control diet. TBI PLX mice had elevated proinflammatory cytokines and lower anti-inflammatory cytokines in the blood at 7 DPI compared to TBI mice on a control diet. CSF-1R inhibition reduced the immune response to TBI at 1 and 3 DPI, but elevated peripheral inflammation at 7 DPI.

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TI Efficacy of acupuncture on repair of glial scars in rats with traumatic

brain injury

SO JOURNAL OF TRADITIONAL CHINESE MEDICINE

LA English

DT Article

DE acupuncture; astrocytes; microglia; gliosis; brain injuries; traumatic

ID CENTRAL-NERVOUS-SYSTEM; MICROGLIA; NEUROINFLAMMATION

AB OBJECTIVE: To explore the underlying mechanism of acupuncture on nerve repair by investigating its effect on the differentiation of glial cells and the repair of glial scars. METHODS: Sprague-Dawley rats were randomly allocated to three groups: normal group, model group, and acupuncture group. Acupuncture was applied at Renzhong (GV26), Baihui (GV20), Fengfu (GV16), Yamen (GV15) and Hegu (LI4) within 12 h after TBI modeling with a frequency of one session per day for 4 weeks. Neurobehavioral assessment, hematoxylin and eosin staining, immunofluorescence detection, and magnetic resonance imaging scanning were performed on days 3, 7, 14, and 28 after modeling of traumatic brain injury (TBI). RESULTS: Acupuncture promoted the proliferation of glial cells and glial scars at an early stage but inhibited the proliferation of glial cells and glial scars at a late stage. Morphological observations and immunofluorescence histochemistry showed that the morphology of the perilesional cortex in the acupuncture group was improved and the number of neurons was increased when compared with the model group. The lesion size of ipsilateral brain parenchyma in the acupuncture group was smaller than in the model group on days 7, 14, and 28 (P < 0.05) after TBI modeling. CONCLUSIONS: Acupuncture might have a bidirectional regulatory effect on glial scar repair after TBI by promoting the proliferation of glial cells and glial scars to limit the injured area and relieve nerve injury during the early stages, and by inhibiting glial scar hyperplasia to benefit the regeneration and repair of neurons and axons and promote neurological function recovery during the later stages.

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FU National Natural Science Foundation of China: Explore the Effect and

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[81574066]; National Natural Science Foundation of China: Explore the

Effect of Acupuncture on Brain Function Remodeling and the Benign

Bidirectional Regulation to Autophagy After TBI [2021A1515110146];

National Natural Science Foundation of China: Basing on the Theory of

Bidirectional Benign Regulation to Investigate the Mechanism of

Acupuncture Regulating Microglia-mediated Immune Imbalance to Promote

Nerve Repair After TBI [82205249]; National Natural Science Foundation

of China: Based on Iron Metabolic Pathway to Explore the Mechanism of

Electroacupuncture Inhibits Ferroptosis to Reduce TBI Nerve Injury

[2022M710912]; China Postdoctoral Science Foundation: the Effect and

Mechanism of Electroacupuncture in Promoting Neural Function Repair by

Regulating Iron Metabolism in Neurons of TBI Rats [81873362]; Natural

Science Foundation of Guangdong Province: the Effect and Mechanism of

Acupuncture on the Linkage of Neuron Autophagy and Apoptosis after TBI

Based on PI3K/AKT/mTOR Signaling Pathway; Natural Science Foundation of

Guangdong Province: the Effect and Mechanism of Acupuncture on

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PT J

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TI Human umbilical cord-derived mesenchymal stem cells-harvested

mitochondrial transplantation improved motor function in TBI models

through rescuing neuronal cells from apoptosis and alleviating

astrogliosis and microglia activation

SO INTERNATIONAL IMMUNOPHARMACOLOGY

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DT Article; Early Access

DE Traumatic brain injury; Mitochondrial transplantation; Mesenchymal stem

cells; Apoptosis; Sensorimotor behaviour

ID TRAUMATIC BRAIN-INJURY; COMBINATION THERAPY; OXIDATIVE STRESS;

DYSFUNCTION; REPERFUSION; PROGESTERONE; EXPRESSION; INJECTION; RECOVERY;

DAMAGE

AB Each year, traumatic brain injury (TBI) causes a high rate of mortality throughout the world and those who survive have lasting disabilities. Given that the brain is a particularly dynamic organ with a high energy consumption rate, the inefficiency of current TBI treatment options highlights the necessity of repairing damaged brain tissue at the cellular and molecular levels, which according to research is aggravated due to ATP deficiency and reactive oxygen species surplus. Taking into account that mitochondria contribute to generating energy and controlling cellular stress, mitochondrial transplantation as a new treatment approach has lately reduced complications in a number of diseases by supplying healthy and functional mitochondria to the damaged tissue. For this reason, in this study, we used this technique to transplant human umbilical cord-derived mesenchymal stem cells (hUC-MSCs)-derived mitochondria as a suitable source for mitochondrial isolation into rat models of TBI to examine its therapeutic benefit and the results showed that the successful mitochondrial internalisation in the neuronal cells significantly reduced the number of brain cells undergoing apoptosis, alleviated astrogliosis and microglia activation, retained normal brain morphology and cytoarchitecture, and improved sensorimotor functions in a rat model of TBI. These data indicate that human umbilical cord-derived mesenchymal stem cellsisolated mitochondrial transplantation improves motor function in a rat model of TBI via rescuing neuronal cells from apoptosis and alleviating astrogliosis and microglia activation, maybe as a result of restoring the lost mitochondrial content.

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TC 5

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U1 3

U2 6

PU ELSEVIER

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TI Neuroinflammation aggravated by traumatic brain injury at high altitude

is reversed by L-serine <i>via</i> NFAT1-mediated microglial

polarization

SO FRONTIERS IN CELLULAR NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury; high altitude; L-serine; neuroprotection;

microglia; nuclear factor of activated T-cell 1

ID ACUTE MOUNTAIN-SICKNESS; MICROGLIA/MACROPHAGE POLARIZATION; WHITE-MATTER

AB Traumatic brain injury (TBI) is one of the main causes of disability and death, especially in plateau areas, where the degree of injury is often more serious than in plain areas. It is likely that high altitude (HA) aggravates neuroinflammation; however, prior studies are limited. This study was designed to evaluate the effects of HA on the degree of TBI and the neuroprotective effects and underlying mechanisms of L-serine against TBI at HA (HA-TBI). In in vivo experiments, wild-type mice and mice with Nfat1 (Nfat1(-/-)) deficiency in the C57BL/6 background were kept in a hypobaric chamber for 3 days under simulated conditions of 4,000 m, 6,000 m and 8,000 m above sea level. After leaving the chamber, the standardized TBI model was established immediately. Mice were then intraperitoneally injected with L-serine (342 mg.kg(-1)) 2 h after TBI and then daily for 5 days. Behavioral tests and histological analysis were assessed at different time points post TBI induction. In vitro, we applied primary cultured microglia for hypoxia treatment (1% O-2 for 24 h). The major findings include the following: (1) with increasing altitude, the neurological function of TBI mice decreased, and the damage to cerebral gray matter and white matter became more significant, (2) L-serine significantly improved the sensorimotor function of mice, reversed the increase in brain lesion volume, and promoted the renovation of brain tissue after HA-TBI, (3) L-serine significantly decreased the activation of microglia and promoted microglia polarization toward the protective M2 phenotype both in vivo and in vitro, (4) L-serine significantly suppressed the expression of NFAT1 in mice after HA-TBI and inhibited NFAT1 expression in primary microglia after hypoxia, and (5) knockout of Nfat1 inhibited the inflammatory reaction caused by excessive activation of microglia, and L-serine lost its neuroprotective effect in Nfat1 knockout mice. The present study suggests that HA aggravates brain damage after TBI and that the damage also increases with increasing altitude. As an endogenous amino acid, L-serine may be a neuroprotective agent against HA-TBI, and suppression of NFAT1 in microglia is a potential therapy for neuroinflammation in the future.

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TI Bumetanide induces post-traumatic microglia-interneuron contact to

promote neurogenesis and recovery

SO BRAIN

LA English

DT Article; Early Access

DE traumatic brain injury; microglia; chloride homeostasis;

neuroinflammation; GABAergic transmission

ID TRAUMATIC BRAIN-INJURY; ADULT HIPPOCAMPAL NEUROGENESIS; NEURAL

STEM-CELL; THETA RHYTHM; WORKING-MEMORY; UP-REGULATION; INFLAMMATION;

OSCILLATIONS; MECHANISM; DYSFUNCTION

AB Although the Na-K-Cl cotransporter (NKCC1) inhibitor bumetanide has prominent positive effects on the pathophysiology of many neurological disorders, the mechanism of action is obscure. Attention paid to elucidating the role of Nkcc1 has mainly been focused on neurons, but recent single cell mRNA sequencing analysis has demonstrated that the major cellular populations expressing NKCC1 in the cortex are non-neuronal. We used a combination of conditional transgenic animals, in vivo electrophysiology, two-photon imaging, cognitive behavioural tests and flow cytometry to investigate the role of Nkcc1 inhibition by bumetanide in a mouse model of controlled cortical impact (CCI). Here, we found that bumetanide rescues parvalbumin-positive interneurons by increasing interneuron-microglia contacts shortly after injury. The longitudinal phenotypic changes in microglia were significantly modified by bumetanide, including an increase in the expression of microglial-derived BDNF. These effects were accompanied by the prevention of CCI-induced decrease in hippocampal neurogenesis. Treatment with bumetanide during the first week post-CCI resulted in significant recovery of working and episodic memory as well as changes in theta band oscillations 1 month later. These results disclose a novel mechanism for the neuroprotective action of bumetanide mediated by an acceleration of microglial activation dynamics that leads to an increase in parvalbumin interneuron survival following CCI, possibly resulting from increased microglial BDNF expression and contact with interneurons. Salvage of interneurons may normalize ambient GABA, resulting in the preservation of adult neurogenesis processes as well as contributing to bumetanide-mediated improvement of cognitive performance.

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TI Fixed Time-Point Analysis Reveals Repetitive Mild Traumatic Brain Injury

Effects on Resting State Functional Magnetic Resonance Imaging

Connectivity and Neuro-Spatial Protein Profiles

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE CHIMERA; diffusion tensor imaging; microglia; optic tract; repetitive

mild TBI; resting state fMRI; thalamus

ID DISEASE; ROBUST; RISK

AB Repetitive mild traumatic brain injuries (rmTBIs) are serious trauma events responsible for the development of numerous neurodegenerative disorders. A major challenge in developing diagnostics and treatments for the consequences of rmTBI is the fundamental knowledge gaps of the molecular mechanisms responsible for neurodegeneration. It is both critical and urgent to understand the neuropathological and functional consequences of rmTBI to develop effective therapeutic strategies. Using the Closed-Head Impact Model of Engineered Rotational Acceleration, or CHIMERA, we measured neural changes following injury, including brain volume, diffusion tensor imaging, and resting-state functional magnetic resonance imaging coupled with graph theory and functional connectivity analyses. We determined the effect of rmTBI on markers of gliosis and used NanoString-GeoMx to add a digital-spatial protein profiling analysis of neurodegenerative disease-associated proteins in gray and white matter regions. Our analyses revealed aberrant connectivity changes in the thalamus, independent of microstructural damage or neuroinflammation. We also identified distinct changes in the levels of proteins linked to various neurodegenerative processes including total and phospho-tau species and cell proliferation markers. Together, our data show that rmTBI significantly alters brain functional connectivity and causes distinct protein changes in morphologically intact brain areas.

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TI Gastrodin Prevents Neuronal Apoptosis and Improves Neurological Deficits

in Traumatic Brain Injury Rats through PKA/CREB/Bcl2 Axis

SO FRONTIERS IN BIOSCIENCE-LANDMARK

LA English

DT Article

DE gastrodin; neurological protection; microglial activation; traumatic

brain injury

ID PATHWAY

AB Background: Gastrodin (Gas) exhibits anti-inflammatory properties against diseases associated with the central nervous system (CNS). This study aimed to investigate the potential neuroprotective role of Gas in traumatic brain injury (TBI).Methods: A rat TBI model was established in male adult Sprague-Dawley (SD) rats by controlled cortical impingement (CCI), and lipopolysaccharide (LPS) was applied to induce the activation of BV2 microglia and HT22 hippocampal neurons. Neurological deficits, motor function and brain water content were evaluated in TBI rats. TUNEL and Nissl's staining were applied to measure neuronal degeneration and apoptosis. Microglial activation, the mRNA and protein profiles of pro-inflammatory cytokines were tested by immunohistochemistry (IHC), quantitative reverse transcriptase-polymerase chain reaction (qRT-PCR) and enzyme-linked immunosorbent assay (ELISA), respectively.Results: Gas significantly reduced neurological deficits, cerebral edema, and neuronal apoptosis and improved motor function in TBI mice. In addition, Gas inactivated microglia and blocked the production of pro-inflammatory cytokines on the damaged side of the TBI rat brain. In vitro, Gas attenuated BV2 microglia inflammation and reduced HT22 hippocampal neuronal apoptosis. On the other hand, Gas activated the PKA/CREB/BDNF pathway both in vivo and in vitro.Conclusions: Gas blocks microglial activation-mediated inflammation through the PKA/CREB/BDNF pathway, thereby improving neurobehavioral function after TBI, which provides a potential therapeutic benefit for treating TBI.

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TI Deletion of PTEN in microglia ameliorates chronic neuroinflammation

following repetitive mTBI

SO MOLECULAR AND CELLULAR NEUROSCIENCE

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DT Article

DE Traumatic brain injury; TBI; Microglia; Microglial activation;

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ID TRAUMATIC BRAIN-INJURY; ALZHEIMERS-DISEASE; MOUSE MODEL; TAU PATHOLOGY;

RISK-FACTOR; IN-VIVO; INFLAMMATION; ACTIVATION; MATTER; WHITE

AB Traumatic brain injury is a leading cause of morbidity and mortality in adults and children in developed nations. Following the primary injury, microglia, the resident innate immune cells of the CNS, initiate several inflammatory signaling cascades and pathophysiological responses that may persist chronically; chronic neuroinflammation following TBI has been closely linked to the development of neurodegeneration and neurological dysfunction. Phosphoinositide 3-kinases (PI3Ks) are a family of lipid kinases that have been shown to regulate several key mechanisms in the inflammatory response to TBI. Increasing evidence has shown that the modulation of the PI3K/AKT signaling pathway has the potential to influence the cellular response to inflammatory stimuli. However, directly targeting PI3K signaling poses several challenges due to its regulatory role in several cell survival pathways. We have previously identified that the phosphatase and tensin homolog deleted on chromosome 10 (PTEN), the major negative regulator of PI3K/AKT signaling, is dysregulated following exposure to repetitive mild traumatic brain injury (r-mTBI). Moreover, this dysregulated PI3K/AKT signaling was correlated with chronic microglial-mediated neuroinflammation. Therefore, we interrogated microglial-specific PTEN as a therapeutic target in TBI by generating a microglial-specific, Tamoxifen inducible conditional PTEN knockout model using a CX3CR1 Cre recombinase mouse line PTENfl/fl/CX3CR1+/CreERT2 (mcg-PTENcKO), and exposed them to our 20-hit r-mTBI paradigm. Animals were treated with tamoxifen at 76 days post-last injury, and the effects of microglia PTEN deletion on immune-inflammatory responses were assessed at 90-days post last injury. We observed that the deletion of microglial PTEN ameliorated the proinflammatory response to repetitive brain trauma, not only reducing chronic microglial activation and proinflammatory cytokine production but also rescuing TBI-induced reactive astrogliosis, demonstrating that these effects extended beyond microglia alone. Additionally, we observed that the pharmacological inhibition of PTEN with BpV(HOpic) ameliorated the LPSinduced activation of microglial NF kappa B signaling in vitro. Together, these data provide support for the role of PTEN as a regulator of chronic neuroinflammation following repetitive mild TBI.

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TI Myelin degradation, axonal changes and expression trajectories of glial

cells stimulated by rapid head insult in humans to estimate approximate

time elapsed since trauma

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LA English

DT Article

DE Forensic; Severe traumatic brain injury; Myelin degeneration; Axonal

changes; Astroglia; Microglia

ID AMYLOID PRECURSOR PROTEIN; WHITE-MATTER; BRAIN-INJURY; CORPUS-CALLOSUM;

NEUROFILAMENT COMPACTION; MILD; NEUROINFLAMMATION; OLIGODENDROCYTES;

INTERLEUKIN-6; DEMYELINATION

AB BackgroundPost severe traumatic brain injury (sTBI), axonal alterations lead to myelin loss and its degeneration. In the recovery phase, numerous intermingled biochemical pathways involving complex inflammatory reactions cloud the understanding of this yet undiscerned process that also varies with agonal period. In cases with dubious histories, approximating the survival time can be challenging, and expression levels of characteristic markers may aid forensic experts in the same.MethodsThis exploratory study recruited 100 samples-68 sTBI, 22 non-TBI and 10 age- and sex-matched control samples. Male:female ratio was 87:13. Histochemical staining using H&E was used to characterize myelination pattern, and IHC of GFAP and CD-68 were performed to assess astroglial and microglial reactions with respect to survival time in specific sites.ResultAmong sTBI, non-TBI and control recruits, sTBI patients depicted significant myelination abnormalities, astroglial proliferation and microglial reaction and varying with survival time. Non-TBI and control samples depicted nearly similar profiles.ConclusionIn order to untangle the complex mesh of biochemical responses, nuanced research on individual factors (both pre- and post mortem) with regard to specific site and survival time are warranted. Standardizing experimental data and converting it into empirical data shall aid forensic experts in suggesting approximate agonal period.

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AF Andreu, MaryLourdes

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TI Dose-dependent modulation of microglia activation in rats after

penetrating traumatic brain injury (pTBI) by transplanted human neural

stem cells

SO PLOS ONE

LA English

DT Article

ID COGNITIVE DEFICITS; MECHANISM

AB Traumatic brain injury (TBI) often results in long-lasting patterns of neurological deficits including motor, sensory, and cognitive abnormalities. Cranial gunshot survivors are among the most disabled TBI patients and face a lifetime of disability with no approved strategies to protect or repair the brain after injury. Recent studies using a model of penetrating TBI (pTBI) have reported that human neural stem cells (hNSCs) transplantation can lead to dose and location-dependent neuroprotection. Evidence for regional patterns of microglial activation has also been reported after pTBI with evidence for microglial cell death by pyroptosis. Because of the importance of injury-induced microglial activation in the pathogenesis of TBI, we tested the hypothesis that dose-dependent hNSC mediated neuroprotection after pTBI was associated with reduced microglial activation in pericontusional cortical areas. To test this hypothesis, quantitative microglial/macrophage Iba1 immunohistochemistry and Sholl analysis was conducted to investigate the arborization patterns using four experimental groups including, (i) Sham operated (no injury) + low dose (0.16 million cells/rat), (ii) pTBI + vehicle (no cells), (iii) pTBI + low dose hNSCs (0.16 million/rat), and (iv) pTBI + high dose hNSCs (1.6 million cells/rat). At 3 months post-transplantation (transplants at one week after pTBI), the total number of intersections was significantly reduced in vehicle treated pTBI animals versus sham operated controls indicating increased microglia/macrophage activation. In contrast, hNSC transplantation led to a dose-dependent increase in the number of intersections compared to pTBI vehicle indicating less microglia/macrophage activation. The peak of Sholl intersections at 1 mu m from the center of the microglia/macrophages ranged from similar to 6,500-14,000 intersections for sham operated, similar to 250-500 intersections for pTBI vehicle, similar to 550-1,000 intersections for pTBI low dose, and similar to 2,500-7,500 intersections for pTBI high dose. Plotting data along the rostrocaudal axis also showed that pericontusional cortical areas protected by hNSC transplantation had increased intersections compared to nontreated pTBI animals. These studies using a non-biased Sholl analysis demonstrated a dose-dependent reduction in inflammatory cell activation that may be associated with a neuroprotective effect driven by the cellular transplant in perilesional regions after pTBI.

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Lin, Long

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Lin, Yuanxiang

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Ding, Chenyu

Kang, Dezhi

TI ADAM17 Aggravates the Inflammatory Response by Modulating Microglia

Polarization Through the TGF-β1/Smad Pathway Following Experimental

Traumatic Brain Injury

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE ADAM17; microglia; neuroinflammation; TGF-beta 1/Smad pathway; traumatic

brain injury

ID TGF-BETA; NEUROINFLAMMATION; ACTIVATION; BARRIER

AB Microglia-mediated neuroinflammatory responses play important roles in secondary neurological injury after traumatic brain injury (TBI). The TGF-beta pathway participates in the regulation of M1/M2 phenotype transformation of microglia. TGF-beta can activate the Smad pathway by binding to TGF-beta Rs, which is regulated by the cleavage function of A disintegrin and metalloproteinase 17 (ADAM17). However, the role of ADAM17 and the associated signaling pathways in the pathological process after TBI remain unclear. Herein, we assessed the transformation of microglia M1/M2 phenotype polarization and the neuroinflammatory response after the inhibition of ADAM17. The formation of TGF-beta Rs and TGF-beta 1/TGF-beta RII complexes on microglia were detected to evaluate the effect of ADAM17 inhibition on the TGF-beta 1/Smad pathway. ADAM17 was highly expressed after TBI and mainly located in themicroglia. the inhibition of ADAM17 improved neurological function after TBI. The neuroprotective effect of ADAM17 inhibition was related to a shift from the M1 microglial phenotype to the M2 microglial phenotype, thus reducing TBI-induced neuroinflammation. ADAM17 inhibition increased expression of TGF-beta Rs on the microglia membrane, promoted formation of TGF-beta 1/TGF-beta RII complexes, and induced intranuclear translocation of Smads, which activated the TGF-beta/Smad pathway. In conclusion, our study suggested that ADAM17 inhibition regulated microglia M1/M2 phenotype polarization through the TGF-beta 1/Smad pathway and influenced the neuroinflammatory response after TBI.

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TI B cell treatment promotes a neuroprotective microenvironment after

traumatic brain injury through reciprocal immunomodulation with

infiltrating peripheral myeloid cells

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Traumatic brain injury; B cells; Myeloid cells; Monocytes; Macrophages;

Microglia; Immunomodulation

ID MACROPHAGES; CNS; REGENERATION; MICROGLIA; MONOCYTES; DEPLETION; STROKE

AB Traumatic brain injury (TBI) remains a major cause of death and severe disability worldwide. We found previously that treatment with exogenous naive B cells was associated with structural and functional neuroprotection after TBI. Here, we used a mouse model of unilateral controlled cortical contusion TBI to investigate cellular mechanisms of immunomodulation associated with intraparenchymal delivery of mature naive B lymphocytes at the time of injury. Exogenous B cells showed a complex time-dependent response in the injury microenvironment, including significantly increased expression of IL-10, IL-35, and TGF beta, but also IL-2, IL-6, and TNF alpha. After 10 days in situ, B cell subsets expressing IL-10 or TGF beta dominated. Immune infiltration into the injury predominantly comprised myeloid cells, and B cell treatment did not alter overall numbers of infiltrating cells. In the presence of B cells, significantly more infiltrating myeloid cells produced IL-10, TGF beta, and IL-35, and fewer produced TNF alpha, interferon-gamma and IL-6 as compared to controls, up to 2 months post-TBI. B cell treatment significantly increased the proportion of CD206(+) infiltrating monocytes/macrophages and reduced the relative proportion of activated microglia starting at 4 days and up to 2 months post-injury. Ablation of peripheral monocytes with clodronate liposomes showed that infiltrating peripheral monocytes/macrophages are required for inducing the regulatory phenotype in exogenous B cells. Reciprocally, B cells specifically reduced the expression of inflammatory cytokines in infiltrating Ly6C(+) monocytes/macrophages. These data support the hypothesis that peripheral myeloid cells, particularly infiltrating monocyte/macrophages, are key mediators of the neuroprotective immunomodulatory effects observed after B cell treatment.

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TC 2

Z9 2

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U2 1

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J9 J NEUROINFLAMM

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TI The Central Fluid Percussion Brain Injury in a Gyrencephalic Pig Brain:

Scalable Diffuse Injury and Tissue Viability for Glial Cell

Immunolabeling following Long-Term Refrigerated Storage

SO BIOMEDICINES

LA English

DT Article

DE traumatic brain injury; axonal injury; micro pig; diffuse pathology;

microglia; aged tissue

ID TRAUMATIC BRAIN; AXONAL INJURY; MICROGLIAL ACTIVATION; LUNG INJURY;

MODEL; RESPONSES; PROTEIN; DAMAGE; NEUROINFLAMMATION; NEURODEGENERATION

AB Traumatic brain injury (TBI) affects millions of people annually; however, our knowledge of the diffuse pathologies associated with TBI is limited. As diffuse pathologies, including axonal injury and neuroinflammatory changes, are difficult to visualize in the clinical population, animal models are used. In the current study, we used the central fluid percussion injury (CFPI) model in a micro pig to study the potential scalability of these diffuse pathologies in a gyrencephalic brain of a species with inflammatory systems very similar to humans. We found that both axonal injury and microglia activation within the thalamus and corpus callosum are positively correlated with the weight-normalized pressure pulse, while subtle changes in blood gas and mean arterial blood pressure are not. We also found that the majority of tissue generated up to 10 years previously is viable for immunofluorescent labeling after long-term refrigeration storage. This study indicates that a micro pig CFPI model could allow for specific investigations of various degrees of diffuse pathological burdens following TBI.

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TC 1

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U2 4

PU MDPI

PI BASEL

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TI Interleukin 4 Reduces Brain Hyperexcitability after Traumatic Injury by

Downregulating TNF-α, Upregulating IL-10/TGF-β, and Potential Directing

Macrophage/Microglia to the M2 Anti-inflammatory Phenotype

SO INFLAMMATION

LA English

DT Article; Early Access

DE arginase1; kindling; rat; trauma; traumatic epilepsy

ID RECOMBINANT HUMAN INTERLEUKIN-4; CONTROLLED CORTICAL IMPACT;

CENTRAL-NERVOUS-SYSTEM; NECROSIS-FACTOR-ALPHA; KINDLING EPILEPTOGENESIS;

CEREBRAL-ISCHEMIA; TISSUE-REPAIR; IL-4; ACTIVATION; EXPRESSION

AB -Macrophage/microglia are activated after Traumatic brain injury (TBI), transform to inflammatory phenotype (M1) and trigger neuroinflammation, which provokes epileptogenesis. Interleukin-4 (IL-4) is a well-known drive of macrophage/microglia to the anti-inflammatory phenotype (M2). We tested effect of IL-4 on speed of epileptogenesis, brain expression of inflammatory and anti-inflammatory cytokines, and lesion size in TBI-injured male rats. Rats underwent TBI by Controlled Cortical Impact. Then 100 ng IL-4 was injected into cerebral ventricles. One day after TBI, pentylenetetrazole (PTZ) kindling started and development of generalized seizures was recorded. The lesion size, cell survival rate, TNF-alpha, TGF-beta, IL-10, and Arginase1 (Arg1) was measured in the brain 6 h, 12 h, 24 h, 48 h, and 5 days after TBI. Astrocytes and macrophage/microglia activation/polarization was assessed by GFAP/Arg1 and Iba1/Arg1 immunostaining. TBI-injured rats were kindled by 50% less PTZ injections than control and sham-operated rats. IL-4 did not change kindling rate in sham-operated rats but inhibited acceleration of kindling rate in the TBI-injured rats. IL-4 decreased damage volume and number of destroyed neurons. IL-4 stopped TNF-alpha whereas upregulated TGF-beta, IL-10, and Arg1 expressions. Iba1/Arg1 positive macrophage/microglia was notably increased 48 h after IL-4 administration. IL-4 suppresses TBI-induced acceleration of epileptogenesis in rats by directing TBI neuroinflammation toward an anti-inflammatory tone and inhibition of cell death.

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TI Mass cytometric analysis of the immune cell landscape after traumatic

brain injury elucidates the role of complement and complement receptors

in neurologic outcomes

SO ACTA NEUROPATHOLOGICA COMMUNICATIONS

LA English

DT Article

DE Traumatic brain injury; Complement; Neuroinflammation; Complement

inhibition; Microglia; Mass cytometry

ID SPINAL-CORD; C5A RECEPTOR; MICROGLIA; PMX53; INFLAMMATION; ANTAGONISTS;

RECOVERY; MODEL

AB Following traumatic brain injury (TBI), a neuroinflammatory response can persist for years and contribute to the development of chronic neurological manifestations. Complement plays a central role in post-TBI neuroinflammation, and C3 opsonins and the anaphylatoxins (C3a and C5a) have been implicated in promoting secondary injury. We used single cell mass cytometry to characterize the immune cell landscape of the brain at different time points after TBI. To specifically investigate how complement shapes the post-TBI immune cell landscape, we analyzed TBI brains in the context of CR2-Crry treatment, an inhibitor of C3 activation. We analyzed 13 immune cell types, including peripheral and brain resident cells, and assessed expression of various receptors. TBI modulated the expression of phagocytic and complement receptors on both brain resident and infiltrating peripheral immune cells, and distinct functional clusters were identified within same cell populations that emerge at different phases after TBI. In particular, a CD11c+ (CR4) microglia subpopulation continued to expand over 28 days after injury, and was the only receptor to show continuous increase over time. Complement inhibition affected the abundance of brain resident immune cells in the injured hemisphere and impacted the expression of functional receptors on infiltrating cells. A role for C5a has also been indicated in models of brain injury, and we found significant upregulation of C5aR1 on many immune cell types after TBI. However, we demonstrated experimentally that while C5aR1 is involved in the infiltration of peripheral immune cells into the brain after injury, it does not alone affect histological or behavioral outcomes. However, CR2-Crry improved post-TBI outcomes and reduced resident immune cell populations, as well as complement and phagocytic receptor expression, indicating that its neuroprotective effects are mediated upstream of C5a generation, likely via modulating C3 opsonization and complement receptor expression.

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TI Omega-3 Polyunsaturated Fatty Acids Protect Neurological Function After

Traumatic Brain Injury by Suppressing Microglial Transformation to the

Proinflammatory Phenotype and Activating Exosomal NGF/TrkA Signaling

SO MOLECULAR NEUROBIOLOGY

LA English

DT Article; Early Access

DE Traumatic brain injury; Omega-3 polyunsaturated fatty acid; Microglia;

ADAM17; TNF-& alpha;; NF-& kappa;B pathway; Exosome; Nerve growth factor

ID OMEGA-3-FATTY-ACIDS; INFLAMMATION; ALPHA; IMPAIRMENT; EXPRESSION

AB The transformation of microglia to a pro-inflammatory phenotype at the site of traumatic brain injury (TBI) drives the progression of secondary neurodegeneration and irreversible neurological impairment. Omega-3 polyunsaturated fatty acids (PUFA) have been shown to suppress this phenotype transformation, thereby reducing neuroinflammation following TBI, but the molecular mechanisms are unknown. We found that Omega-3 PUFA suppressed the expression of disintegrin metalloproteinase (ADAM17), the enzyme required to convert tumor necrosis factor-a (TNF-a) to the soluble form, thereby inhibiting the TNF-a/NF-?B pathway both in vitro and in a mouse model of TBI. Omega-3 PUFA also prevented the reactive transformation of microglia and promoted the secretion of microglial exosomes containing nerve growth factor (NGF), activating the neuroprotective NGF/TrkA pathway both in culture and TBI model mice. Moreover, Omega-3 PUFA suppressed the pro-apoptotic NGF/P75NTR pathway at the TBI site and reduced apoptotic neuronal death, brain edema, and disruption of the blood-brain barrier. Finally, Omega-3 PUFA preserved sensory and motor function as assessed by two broad-spectrum test batteries. The beneficial effects of Omega-3 PUFA were blocked by an ADAM17 promotor and by a NGF inhibitor, confirming the pathogenic function of ADAM17 and the central neuroprotective role of NGF. Collectively, these findings provide a strong experimental basis for Omega-3 PUFA as a potential clinical treatment for TBI.

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TC 2

Z9 2

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TI Deletion of glutamate carboxypeptidase II (GCPII), but not GCPIII,

provided long-term benefits in mice with traumatic brain injury

SO CNS NEUROSCIENCE & THERAPEUTICS

LA English

DT Article; Early Access

DE bovine serum albumin; glutamate carboxypeptidase II (GCPII); glutamate

carboxypeptidase III (GCPIII); glutamate excitotoxicity; learning and

memory; microglia; N-acetylaspartylglutamate (NAAG); traumatic brain

injury

ID LINKED ACIDIC DIPEPTIDASE; ACETYL-ASPARTYL-GLUTAMATE;

REGIONAL-DISTRIBUTION; NAAG; ENZYME; INFLAMMATION; NEUROPROTECTION;

EXPRESSION; NAALADASE; REVEALS

AB Main ProblemN-acetylaspartylglutamate (NAAG) has neuroprotective effects in traumatic brain injury (TBI) by activating metabotropic glutamate receptor 3 (mGluR3) and reducing glutamate release. Glutamate carboxypeptidase II (GCPII) is the primary enzyme responsible for the hydrolysis of NAAG. It remains unclear whether glutamate carboxypeptidase III (GCPIII), a homolog of GCPII, can partially compensate for GCPII's function. MethodsGCPII(-/-), GCPIII(-/-), and GCPII/III-/- mice were generated using CRISPR/Cas9 technology. Mice brain injury model was established through moderate controlled cortical impact (CCI). The relationship between GCPII and GCPIII was explored by analyzing injury response signals in the hippocampus and cortex of mice with different genotypes at the acute (1 day) and subacute (7 day) phase after TBI. ResultsIn this study, we found that deletion of GCPII reduced glutamate production, excitotoxicity, and neuronal damage and improved cognitive function, but GCPIII deletion had no significant neuroprotective effect. Additionally, there was no significant difference in the neuroprotective effect between the combination of GCPII and GCPIII deletion and GCPII deletion alone. ConclusionThese results suggest that GCPII inhibition may be a therapeutic option for TBI, and that GCPIII may not act as a complementary enzyme to GCPII in this context.

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TI Persistence of Hyper-Ramified Microglia in Porcine Cortical Gray Matter

after Mild Traumatic Brain Injury

SO BIOMEDICINES

LA English

DT Article

DE mild TBI; neuroinflammation; microglia; large animal models; fibrinogen

ID AXONAL PATHOLOGY; DISRUPTION; ACCUMULATION; PERMEABILITY; CELLS

AB Traumatic brain injury (TBI) is a major contributor to morbidity and mortality in the United States as several million people visit the emergency department every year due to TBI exposures. Unfortunately, there is still no consensus on the pathology underlying mild TBI, the most common severity sub-type of TBI. Previous preclinical and post-mortem human studies have detailed the presence of diffuse axonal injury following TBI, suggesting that white matter pathology is the predominant pathology of diffuse brain injury. However, the inertial loading produced by TBI results in strain fields in both gray and white matter. In order to further characterize gray matter pathology in mild TBI, our lab used a pig model (n = 25) of closed-head rotational acceleration-induced TBI to evaluate blood-brain barrier disruptions, neurodegeneration, astrogliosis, and microglial reactivity in the cerebral cortex out to 1 year post-injury. Immunohistochemical staining revealed the presence of a hyper-ramified microglial phenotype-more branches, junctions, endpoints, and longer summed process length-at 30 days post injury (DPI) out to 1 year post injury in the cingulate gyrus (p < 0.05), and at acute and subacute timepoints in the inferior temporal gyrus (p < 0.05). Interestingly, we did not find neuronal loss or astroglial reactivity paired with these chronic microglia changes. However, we observed an increase in fibrinogen reactivity-a measure of blood-brain barrier disruption-predominately in the gray matter at 3 DPI (p = 0.0003) which resolved to sham levels by 7 DPI out to chronic timepoints. Future studies should employ gene expression assays, neuroimaging, and behavioral assays to elucidate the effects of these hyper-ramified microglia, particularly related to neuroplasticity and responses to potential subsequent insults. Further understanding of the brain's inflammatory activity after mild TBI will hopefully provide understanding of pathophysiology that translates to clinical treatment for TBI.

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TI Microglial Nogo delays recovery following traumatic brain injury in mice

SO GLIA

LA English

DT Article; Early Access

DE astrocytes; CCI traumatic brain injury; Cx3cr1; microglia; Nogo

knock-out

ID FUNCTIONAL RECOVERY; GENETIC DELETION; COGNITIVE FUNCTION; AXON

REGENERATION; NEURITE GROWTH; WATER-MAZE; RECEPTOR; NEUROINFLAMMATION;

EXPRESSION; PLASTICITY

AB Nogo-A, B, and C are well described members of the reticulon family of proteins, most well known for their negative regulatory effects on central nervous system (CNS) neurite outgrowth and repair following injury. Recent research indicates a relationship between Nogo-proteins and inflammation. Microglia, the brain's immune cells and inflammation-competent compartment, express Nogo protein, although specific roles of the Nogo in these cells is understudied. To examine inflammation-related effects of Nogo, we generated a microglial-specific inducible Nogo KO (MinoKO) mouse and challenged the mouse with a controlled cortical impact (CCI) traumatic brain injury (TBI). Histological analysis shows no difference in brain lesion sizes between MinoKO-CCI and Control-CCI mice, although MinoKO-CCI mice do not exhibit the levels of ipsilateral lateral ventricle enlargement as injury matched controls. Microglial Nogo-KO results in decreased lateral ventricle enlargement, microglial and astrocyte immunoreactivity, and increased microglial morphological complexity compared to injury matched controls, suggesting decreased tissue inflammation. Behaviorally, healthy MinoKO mice do not differ from control mice, but automated tracking of movement around the home cage and stereotypic behavior, such as grooming and eating (termed cage "activation"), following CCI is significantly elevated. Asymmetrical motor function, a deficit typical of unilaterally brain lesioned rodents, was not detected in CCI injured MinoKO mice, while the phenomenon was present in CCI injured controls 1-week post-injury. Overall, our studies show microglial Nogo as a negative regulator of recovery following brain injury. To date, this is the first evaluation of the roles microglial specific Nogo in a rodent injury model.

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NR 101

TC 1

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U2 4

PU WILEY

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JI Glia

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PT J

AU Wang, ZH

Lu, ZC

Chen, YX

Wang, CX

Gong, PP

Jiang, R

Liu, QQ

AF Wang, Ziheng

Lu, Zhichao

Chen, Yixun

Wang, Chenxing

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Liu, Qianqian

TI Targeting the AKT-P53/CREB pathway with epicatechin for improved

prognosis of traumatic brain injury

SO CNS NEUROSCIENCE & THERAPEUTICS

LA English

DT Article; Early Access

DE epicatechin; inflammation; microglia; neurological function; traumatic

brain injury

ID INFLAMMATION; MICROGLIA

AB AimsThe aim of this study was to evaluate the effect of epicatechin, on neurological recovery and neuroinflammation after traumatic brain injury (TBI) to investigate its potential value in clinical practice. MethodsTBI model was established in adult rats by CCI method. The effect of epicatechin was evaluated after intraperitoneal injection. Neurological recovery after TBI was assessed by Morris Water Maze, mNSS score, Rotarod test and Adhesive removal test. Protein and gene expression was assessed by Western blot, ELISA, PCR and immunofluorescence. Furthermore, the use of AKT pathway inhibitors blocked the therapeutic effects of epicatechin clarifying AKT-P53/CREB as a potential pathway for the effects of epicatechin. ResultsAdministering epicatechin after TBI prevented neuronal death, reduced neuroinflammation, and promoted neurological function restoration in TBI rats. Network pharmacology study suggested that epicatechin may exert its therapeutic benefits through the AKT-P53/CREB pathway ConclusionThese results indicate that epicatechin, a monomeric compound derived from tea polyphenols, possesses potent antioxidant and anti-inflammatory properties after TBI. The mechanism may be related to the regulation of the AKT-P53/CREB signal pathway.

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FX Nantong Natural Science Foundation (JC12022063); National Natural

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NR 27

TC 1

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U2 16

PU WILEY

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AU Zhang, M

Hao, ZL

Wu, JY

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Qiu, Wusi

Cheng, Jun

TI Curcumin ameliorates traumatic brain injury via C1ql3-mediated microglia

M2 polarization

SO TISSUE & CELL

LA English

DT Article

DE Traumatic brain injury; Microglia; Curcumin; Complement 1q-like-3

protein

ID IMPROVES FUNCTIONAL RECOVERY; NEUROINFLAMMATION; ACTIVATION; EXPRESSION;

PATHWAYS; FAMILY; MODEL

AB Purpose: Curcumin can regulate the polarization of microglia and alleviate traumatic brain injury (TBI). However, its detailed action mechanism on downregulating Complement 1q-like-3 protein (C1ql3) in TBI is less reported. The purpose of this study is to explore the role and mechanism of curcumin-regulated C1ql3 in TBI. Method: GSE23639 dataset was used to acquire gene data for microglia. C57BL/6 J wild-type (WT) mice were subjected to establish a controlled cortical impact model of TBI. The effects of curcumin (200 mg/kg) on the brain injury, inflammatory cytokine levels, microglia polarization, and C1ql3 protein expression in mice and BV2 cells were detected by H & E staining, qRT-PCR, immunofluorescence, and Western blot, respectively. The effects of curcumin (5, 10, 20 & mu;mol/L) and lipopolysaccharides (LPS, 1 & mu;g/mL) on the viability of BV-2 cells were determined by MTT assay. After the transfection of C1ql3 overexpression plasmid, C1ql3 expression, IL-1 & beta; and IL-6 levels, and the number of CD16+/32+ and CD206+ cells were determined by qRT-PCR, ELISA and flow cytometry, respectively. Result: C1ql3 expression was down-regulated in microglia after the curcumin treatment. Curcumin treatment could alleviate the TBI-induced brain injury in mice, reduce IL-1 & beta; and IL-6 levels, promote M2 polarization of microglia, and decrease C1ql3 protein expression. For BV-2 cells, curcumin treatment had no significant toxic effect on cell viability, but reversed the effect of LPS on cells, while C1ql3 overexpression counteracted the effect of curcumin. Conclusion: Curcumin induces M2 microglia polarization through down-regulating C1ql3 expression, which may become a new treatment method for TBI. Availability of data and materials: The analyzed data sets generated during the study are available from the corresponding author on reasonable request.

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NR 37

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PU CHURCHILL LIVINGSTONE

PI EDINBURGH

PA JOURNAL PRODUCTION DEPT, ROBERT STEVENSON HOUSE, 1-3 BAXTERS PLACE,

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J9 TISSUE CELL

JI Tissue Cell

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TI Modeling the Inflammatory Response of Traumatic Brain Injury Using Human

Induced Pluripotent Stem Cell Derived Microglia

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE cytokine; inflammation; microdialysis; microglia; neuroinflammation;

traumatic brain injury

ID NEUROINFLAMMATION

AB The neuroinflammatory response after traumatic brain injury (TBI) is implicated as a key mediator of secondary injury in both the acute and chronic periods after primary injury. Microglia are the key innate immune cell in the central nervous system, responding to injury with the release of cytokines and chemokines. In this context, we aimed to characterize the downstream cytokine response of human induced pluripotent stem cell (iPSC)-derived microglia when stimulated with five separate cytokines identified after human TBI. The iPSC-derived microglia were exposed to interleukin (IL)-1 & beta;, IL-4, IL-6, IL-10, and tumor necrosis factor (TNF) in the concentration ranges identified in clinical TBI studies. The downstream cytokine response was measured against a panel of 37 separate cytokines over a 72h time-course. The secretome revealed concentration-, time- and combined concentration and time-dependent downstream responses. TNF appeared to be the strongest inducer of downstream cytokine changes (51), followed by IL-1 & beta; (26) and IL-4 (19). IL-10 (11) and IL-6 (10) produced fewer responses. We also compare these responses with our previous studies of iPSC-derived neuronal and astrocyte cultures and the in vivo human TBI cytokine response. Notably, we found microglial culture to induce both a wider range of downstream cytokine responses and a greater fold change in concentration for those downstream responses, compared with astrocyte and neuronal cultures. In summary, we present a dataset for human microglial cytokine responses specific to the secretome found in the clinical context of TBI. This reductionist approach complements our previous datasets for astrocyte and neuronal responses and will provide a platform to enable future studies to unravel the complex neuroinflammatory network activated after TBI.

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U2 5

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TI Senolytic therapy is neuroprotective and improves functional outcome

long-term after traumatic brain injury in mice

SO FRONTIERS IN NEUROSCIENCE

LA English

DT Article

DE traumatic brain injury; senescent cell; senolytic drug; astrocyte;

microglia; inflammation

ID CENTRAL-NERVOUS-SYSTEM; CELLULAR SENESCENCE; CEREBRAL EDEMA; CELLS;

INFLAMMATION; TISSUE; NEUROINFLAMMATION; NEURODEGENERATION;

POLARIZATION; ACTIVATION

AB IntroductionChronic neuroinflammation can exist for months to years following traumatic brain injury (TBI), although the underlying mechanisms remain poorly understood. MethodsIn the current study, we used a controlled cortical impact mouse model of TBI to examine whether proinflammatory senescent cells are present in the brain long-term (months) after TBI and whether ablation of these cells via administration of senolytic drugs can improve long-term functional outcome after TBI. The results revealed that astrocytes and microglia in the cerebral cortex, hippocampus, corpus callosum and lateral posterior thalamus colocalized the senescent cell markers, p16(Ink4a) or p21(Cip1/Waf1) at 5 weeks post injury (5wpi) and 4 months post injury (4mpi) in a controlled cortical impact (CCI) model. Intermittent administration of the senolytic drugs, dasatinib and quercetin (D + Q) beginning 1-month after TBI for 13 weeks significantly ablated p16(Ink4a)-positive- and p21(Cip1/Waf1)-positive-cells in the brain of TBI animals, and significantly reduced expression of the major senescence-associated secretory phenotype (SASP) pro-inflammatory factors, interleukin-1 & beta; and interleukin-6. Senolytic treatment also significantly attenuated neurodegeneration and enhanced neuron number at 18 weeks after TBI in the ipsilateral cortex, hippocampus, and lateral posterior thalamus. Behavioral testing at 18 weeks after TBI further revealed that senolytic therapy significantly rescued defects in spatial reference memory and recognition memory, as well as depression-like behavior in TBI mice. DiscussionTaken as a whole, these findings indicate there is robust and widespread induction of senescent cells in the brain long-term after TBI, and that senolytic drug treatment begun 1-month after TBI can efficiently ablate the senescent cells, reduce expression of proinflammatory SASP factors, reduce neurodegeneration, and rescue defects in reference memory, recognition memory, and depressive behavior.

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TI Microglia and astrocytes mediate synapse engulfment in a MER tyrosine

kinase-dependent manner after traumatic brain injury

SO NEURAL REGENERATION RESEARCH

LA English

DT Article

DE animal model; astrocyte; dendritic spines; lysosome; macrophage; MER

proto-oncokinase; microglia; neurologic function; phagocytosis; synapse

engulfment; traumatic brain injury

ID DENDRITIC SPINES; PHAGOCYTOSIS; INHIBITION; RECOVERY; CELLS

AB Recent studies have shown that microglia/macrophages and astrocytes can mediate synaptic phagocytosis through the MER proto-oncokinase in developmental or stroke models, but it is unclear whether the same mechanism is also active in traumatic brain injury. In this study, we established a mouse model of traumatic brain injury and found that both microglia/macrophages and astrocytes phagocytosed synapses and expression of the MER proto-oncokinase increased 14 days after injury. Specific knockout of MER in microglia/macrophages or astrocytes markedly reduced injury volume and greatly improved neurobehavioral function. In addition, in both microglia/macrophages-specific and astrocytes-specific MER knock-out mice, the number of microglia/macrophage and astrocyte phagocytosing synapses was markedly decreased, and the total number of dendritic spines was increased. Our study suggested that MER proto-oncokinase expression in microglia/macrophages and astrocytes may play an important role in synaptic phagocytosis, and inhibiting this process could be a new strategy for treating traumatic brain injury.

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TI Inhibition of P2X4 and P2X7 receptors improves histological and

behavioral outcomes after experimental traumatic brain injury in rats

SO EXPERIMENTAL AND THERAPEUTIC MEDICINE

LA English

DT Article

DE adenosine triphosphate; gliotransmitter; microglia; purinergic

receptors; traumatic brain injury

ID REACTIVE ASTROCYTES; AXONAL INJURY; ACTIVATION; CYTOKINES; GLUTAMATE;

MICROGLIA; STRIATUM; PROTECTS; ABLATION; MICE

AB Release of large amounts of adenosine triphosphate (ATP), a gliotransmitter, into the extracellular space by traumatic brain injury (TBI) is considered to activate the microglia followed by release of inflammatory cytokines resulting in excessive inflammatory response that induces secondary brain injury. The present study investigated whether antagonists of ATP receptors (P2X4 and/or P2X7) on microglia are beneficial for reducing the post-injury inflammatory response that leads to secondary injury, a prognostic aggravation factor of TBI. Adult male Sprague-Dawley rats were subjected to cortical contusion injury (CCI) and randomly assigned to injury and drug treatment conditions, as follows: i) No surgical intervention (naive group); ii) dimethyl sulfoxide treatment after CCI (CCI-control group); iii) 5-BDBD (antagonist of P2X4 receptor) treatment after CCI (CCI-5-BDBD group); iv) CCI-AZ11645373 (antagonist of P2X7 receptor) treatment after CCI (CCI-AZ11645373 group); v) or 5-BDBD and AZ11645373 treatment after CCI (CCI-5-BDBD + AZ11645373 group). In the CCI-5-BDBD, CCI-AZ11645373, and CCI-5-BDBD + AZ11645373 groups, expression of activated microglia was suppressed in the ipsilateral cortex and hippocampus 3 days after the CCI. Western blotting with ionized calcium-binding adaptor molecule 1 antibody revealed that administration of CCI-5-BDBD and/or CCI-AZ11645373 suppressed expression of microglia and reduced expression of inflammatory cytokine mRNA 3 days after the CCI. Furthermore, the plus maze test, which reflects the spatial memory function and involves the hippocampal function, showed improvement 28 days after secondary injury to the hippocampus. These findings confirmed that blocking the P2X4 and P2X7 receptors, which are ATP receptors central in gliotransmission, suppresses microglial activation and subsequent cytokine expression after brain injury, and demonstrates the potential as an effective treatment for reducing secondary brain injury.

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TI Do astrocytes act as immune cells after pediatric TBI?

SO NEUROBIOLOGY OF DISEASE

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DT Article; Early Access

DE Astrocyte; Pediatric traumatic brain injury; Inflammation; Blood-brain

barrier; Microglia; Neuro-vascular unit; Cytokines; Preclinical models

ID TRAUMATIC BRAIN-INJURY; ENDOTHELIAL GROWTH-FACTOR; IMMATURE RAT;

CEREBROSPINAL-FLUID; SUPRAOPTIC NUCLEUS; CEREBRAL-CORTEX; AXONAL INJURY;

WHITE-MATTER; PIGLET BRAIN; MOUSE MODEL

AB Astrocytes are in contact with the vasculature, neurons, oligodendrocytes and microglia, forming a local network with various functions critical for brain homeostasis. One of the primary responders to brain injury are astrocytes as they detect neuronal and vascular damage, change their phenotype with morphological, proteomic and transcriptomic transformations for an adaptive response. The role of astrocytic responses in brain dysfunction is not fully elucidated in adult, and even less described in the developing brain. Children are vulnerable to trau-matic brain injury (TBI), which represents a leading cause of death and disability in the pediatric population. Pediatric brain trauma, even with mild severity, can lead to long-term health complications, such as cognitive impairments, emotional disorders and social dysfunction later in life. To date, the underlying pathophysiology is still not fully understood. In this review, we focus on the astrocytic response in pediatric TBI and propose a potential immune role of the astrocyte in response to trauma. We discuss the contribution of astrocytes in the local inflammatory cascades and secretion of various immunomodulatory factors involved in the recruitment of local microglial cells and peripheral immune cells through cerebral blood vessels. Taken together, we propose that early changes in the astrocytic phenotype can alter normal development of the brain, with long-term consequences on neurological outcomes, as described in preclinical models and patients.

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TI Inhibition of the interaction between microglial adenosine 2A receptor

and NLRP3 inflammasome attenuates neuroinflammation posttraumatic brain

injury

SO CNS NEUROSCIENCE & THERAPEUTICS

LA English

DT Article; Early Access

DE adenosine 2A receptor; microglia; neuroinflammation; NLRP3 inflammasome;

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ID WHITE-MATTER; ACTIVATION; EXPRESSION; PATHWAY

AB AimsAdenosine 2A receptor (A(2A)R) is widely expressed in the brain and plays important roles in neuroinflammation, and the nucleotide-binding oligomerization domain, leucine-rich repeat, and pyrin domain-containing protein 3 (NLRP3) inflammasome is a crucial component of the innate immune system while the regulation of A(2A)R on it in the central nervous system (CNS) has not been clarified. MethodsThe effects of microglial A(2A)R on NLRP3 inflammasome assembly and activation were investigated in wild-type, A(2A)R- or NLRP3-knockout primary microglia with pharmacological treatment. Microglial A(2A)R or NLRP3 conditional knockout mice were used to interrogate the effects of this regulation on neuroinflammation posttraumatic brain injury (TBI). ResultsWe found that A(2A)R directly interacted with NLRP3 and facilitated NLRP3 inflammasome assembly and activation in primary microglia while having no effects on mRNA levels of inflammasome components. Inhibition of the interaction via A(2A)R agonist or knockout attenuated inflammasome assembly and activation in vitro. In the TBI model, microglial A(2A)R and NLRP3 were co-expressed at high levels in microglia next to the peri-injured cortex, and abrogating of this interaction by microglial NLRP3 or A(2A)R conditional knockout attenuated the neurological deficits and neuropathology post-TBI via reducing the NLRP3 inflammasome activation. ConclusionOur results demonstrated that inhibition of the interaction between A(2A)R and NLRP3 in microglia could mitigate the NLRP3 inflammasome assembly and activation and ameliorate the neuroinflammation post-TBI. It provides new insights into the effects of A(2A)R on neuroinflammation regulation post-TBI and offers a potential target for the treatment of NLRP3 inflammasome-related CNS diseases.

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TI Electroacupuncture regulates microglial polarization via inhibiting

NF-κB/COX2 pathway following traumatic brain injury

SO BRAIN RESEARCH

LA English

DT Article; Early Access

DE Traumatic brain injury; Electroacupuncture; Frequency; NF-kappa B/COX2

pathway; Neuroinflammation; Oxidative stress; Microglial polarization

ID NF-KAPPA-B; SIGNALING PATHWAY; ACUPUNCTURE; TIME; TRANSCRIPTION;

DYNAMICS; PROTECTS; NEURONS

AB Background: Neuroinflammation and oxidative stress are important pathological mechanisms following traumatic brain injury (TBI). The NF-.B/COX2 pathway regulates neuroinflammation and oxidative damage, while microglia also play an important role in neuroinflammation. Since NF-.B is involved in microglial polarization, targeting this pathway and microglial polarization is a critical component of TBI treatment. Currently, electroacupuncture (EA) is widely used to treat various symptoms after TBI, but the mechanisms of EA remain poorly understood. Additionally, the optimal frequency of EA remains unclear, which affects its efficacy. This study focuses on exploring the optimal frequency parameters of EA on TBI and investigating the underlying mechanisms of EA through NF-.B/COX2 pathway and microglial polarization. Methods: The study was divided into two parts. In Experiment 1, 42 Sprague Dawley (SD) rats were induced and randomly divided into seven groups (n = 6). Except for the sham group, all rats underwent controlled cortical impact (CCI) to establish TBI model. Four EA groups (with different frequencies) and manual acupuncture (without current stimulation) received stimulation on the acupoints of Shuigou (GV26), Fengchi (GB20) and Neiguan (PC6) once a day for 7 days. The neurological function was assessed by modified Neurological Severity Scores (mNSS), and the rats' memory and learning were examined by the Morris water maze (MWM). SOD, MDA, and GSH-Px were detected to evaluate the levels of oxidative stress. The levels of IL-1 ss, IL-6, and TNF- alpha were evaluated by Enzyme Linked Immunosorbent Assay (ELISA). Detection of the above indicators indicated a treatment group that exerted the strongest neuroprotection against TBI, we then conducted Experiment 2 using this screened acupuncture treatment to investigate the mechanism of acupuncture. 48 rats were randomly divided into four groups (n = 12): sham, TBI model, acupuncture and PDTC (NF-kappa B inhibitor). Evaluations of mNSS, MWM test, SOD, MDA, GSH-Px, IL-1 ss, IL-6, TNF-alpha, and IL-10 were the same as in Experiment 1. Western blot was applied for detecting the expression levels of NF-kappa B, p-NF-kappa B, COX2, and Arg-1. TUNEL was used to examine neuronal apoptosis. Brain structure was observed by H&E. Iba-1, COX2, and Arg-1 were investigated by immunofluorescence staining. Results: EA with frequency of 2/100 Hz markedly improved neuronal and cognitive function as compared to the other treatment groups. Moreover, it downregulated the expression of MDA, IL-6, IL-1 ss, and TNF- a and upregulated the levels of SOD and GSH-Px. In addition, Both EA with 2/100 Hz and PDTC reduced the levels of p-NF kappa B, COX2 and M1 markers (COX2, IL-6, IL-1 ss, TNF-alpha) and increased the levels of M2 markers (Arg-1, IL-10). Moreover, they had similar effects on reducing inflammation, oxidative stress and apoptosis, and improving neuronal and cognitive function. Conclusions: The collective findings strongly suggest that EA with 2/100 Hz can improve neurologic function by suppressing neuroinflammation, oxidative stress and apoptosis. Additionally, we confirm that EA promotes microglial polarization towards the M2 phenotype through the suppression of NF-.B/COX2 pathway, thus exerting neuroprotective effects after TBI.

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TI Progression of reactive gliosis and astroglial phenotypic changes

following stab wound-induced traumatic brain injury in mice

SO JOURNAL OF NEUROCHEMISTRY

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DT Article; Early Access

DE glia; LPS; microglia; neuroinflammation

ID ASTROCYTES; MICROGLIA; DEGENERATION; REVEALS

AB Astrocytes are the main homeostatic cells in the central nervous system (CNS) and they have an essential role in preserving neuronal physiology. After brain injury, astrocytes become reactive, and that involves a profound change in the astroglial gene expression program as well as intense cytoskeleton remodeling that has been classically shown by the up-regulation of glial fibrillary acidic protein (GFAP), a pan-reactive gene over-expressed in reactive astrocytes, independently of the type of injury. Using the stab wound rodent model of penetrating traumatic injury in the cortex, we here studied the reactive astroglial morphology and reactive microgliosis in detail at 1, 3, 7, 14, and 28 days post-injury (dpi). By combining immunohistochemistry, morphometrical parameters, and Sholl analysis, we segmented the astroglial cell population into clusters of reactive astrocytes that were localized in the core, penumbra, and distal regions of the stab wound. Specifically, highly reactive clusters with more complex morphology, increased C3, decreased aquaporin-4 (AQP4), and glutamine synthetase (GS) expression, were enriched at 7 dpi when behavioral alterations, microgliosis, and neuronal alterations in injured mice were most significant. While pro-inflammatory gain of function with peripheral lipopolysaccharide (LPS) administration immediately after a stab wound expanded these highly reactive astroglial clusters, the treatment with the NF-?B inhibitor sulfasalazine reduced the abundance of this highly reactive cluster. Increased neuronal loss and exacerbated reactive microgliosis at 7 dpi were associated with the expansion of the highly reactive astroglial cluster. We conclude that highly reactive astrocytes found in stab wound injury, but expanded in pro-inflammatory conditions, are a population of astrocytes that become engaged in pathological remodeling with a pro-inflammatory gain of function and loss of homeostatic capacity. Controlling this astroglial population may be a tempting strategy to reduce neuronal loss and neuroinflammation in the injured brain.

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TI Selective neuroimmune modulation by type I interferon drives

neuropathology and neurologic dysfunction following traumatic brain

injury

SO ACTA NEUROPATHOLOGICA COMMUNICATIONS

LA English

DT Article

DE Traumatic brain injury; Type I interferon; Neuroinflammation;

Neurodegeneration; Microglia; Neuroimmune

ID MICROGLIAL ACTIVATION; NEUROINFLAMMATION; INFLAMMATION; BETA;

NEURODEGENERATION; OUTCOMES; GENDER; PLAYS

AB Accumulating evidence suggests that type I interferon (IFN-I) signaling is a key contributor to immune cell-mediated neuropathology in neurodegenerative diseases. Recently, we demonstrated a robust upregulation of type I interferon-stimulated genes in microglia and astrocytes following experimental traumatic brain injury (TBI). The specific molecular and cellular mechanisms by which IFN-I signaling impacts the neuroimmune response and neuropathology following TBI remains unknown. Using the lateral fluid percussion injury model (FPI) in adult male mice, we demonstrated that IFN & alpha;/& beta; receptor (IFNAR) deficiency resulted in selective and sustained blockade of type I interferon-stimulated genes following TBI as well as decreased microgliosis and monocyte infiltration. Molecular alteration of reactive microglia also occurred with diminished expression of genes needed for MHC class I antigen processing and presentation following TBI. This was associated with decreased accumulation of cytotoxic T cells in the brain. The IFNAR-dependent modulation of the neuroimmune response was accompanied by protection from secondary neuronal death, white matter disruption, and neurobehavioral dysfunction. These data support further efforts to leverage the IFN-I pathway for novel, targeted therapy of TBI.

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TI The Role of Microglial Exosomes and miR-124-3p in Neuroinflammation and

Neuronal Repair after Traumatic Brain Injury

SO LIFE-BASEL

LA English

DT Review

DE traumatic brain injury; mesenchymal stem cell-derived exosomes;

microglia; miR-124-3p

ID CELL-DERIVED EXOSOMES; IN-VITRO; INFLAMMATION; CONNEXIN-43; RECOVERY;

BIOLOGY; RAT

AB (1) Background: In this study, we aimed to explore the regulatory mechanism of miR-124-3p microglial exosomes, as they were previously reported to modulate neuroinflammation and promote neuronal repair following traumatic brain injury (TBI). (2) Methods: Studies investigating the impact of microglial exosomal miRNAs, specifically miR-124-3p, on injured neurons and brain microvascular endothelial cells (BMVECs) in the context of TBI were reviewed. (3) Results: Animal models of TBI, in vitro cell culture experiments, RNA sequencing analysis, and functional assays were employed to elucidate the mechanisms underlying the effects of miR-124-3p-loaded exosomes on neuroinflammation and neuronal repair. Anti-inflammatory M2 polarization of microglia, mTOR signaling suppression, and BMVECs-mediated autophagy were reported as the main processes contributing to neuroprotection, reduced blood-brain barrier leakage, and improved neurologic outcomes in animal models of TBI. (4) Conclusions: Microglial exosomes, particularly those carrying miR-124-3p, have emerged as promising candidates for therapeutic interventions in TBI. These exosomes exhibit neuroprotective effects, attenuate neuroinflammation, and promote neuronal repair and plasticity. However, further research is required to fully elucidate the underlying mechanisms and optimize their delivery strategies for effective treatment in human TBI cases.

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TI Infiltrating anti-inflammatory monocytes modulate microglial activation

through toll-like receptor 4/interferon-dependent pathways following

traumatic brain injury

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SP Pediat Trauma Soc

DE Microglia; TBI; TLR4; neuroinflammation

AB BACKGROUND: Traumatic brain injury (TBI) is the leading cause of morbidity and mortality in the pediatric population. Microglia and infiltrating monocyte-derivedmacrophages are crucial immune cells thatmodulate the neuroinflammatory response following TBI. Using C34, a novel pharmacologic toll-like receptor 4 inhibitor, we investigated the intricate interactions between these cells in a murine TBI model.

METHODS: A murine controlled cortical impact model was used, and the results were analyzed on postinjury days 1, 7, 28, and 35. The experimental groups are as follows: (1) shamC57BL/6 wild-type (WT), (2) TBIWT, (3) shamWT + C34, and (4) TBIWT + C34. Quantitative real-time polymerase chain reaction was used to quantify gene expression associated with microglial activation, apoptotic pathways, and type 1 interferon pathway. Flow cytometry was used to isolate microglia and infiltratingmonocytes. Brain lesion volumes were assessed using magnetic resonance imaging. Last, neurocognitive outcomes were evaluated using the Morris Water Maze test. Student's t test and one-way analysis of variance were used for statistical analysis with significance achieved when p < 0.05.

RESULTS: Toll-like receptor 4 inhibition leads to improved neurological sequela post-TBI, possibly because of an increase in infiltrating anti-inflammatory monocytes and a decrease in IFN regulatory factor 7 during acute inflammation, followed by a reduction in apoptosis and M2 microglial expression during chronic inflammation.

CONCLUSION: Toll-like receptor 4 inhibition with C34 skews infiltrating monocytes toward an anti-inflammatory phenotype, leading to enhanced neurocognitive outcomes. Moreover, although M2 microglia have been consistently shown as inducers of neuroprotection, our results clearly demonstrate their detrimental role during the chronic phases of healing post-TBI. (J Trauma Acute Care Surg. 2023;95: 368-375. Copyright (c) 2023 Wolters Kluwer Health, Inc. All rights reserved.)

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NR 36

TC 2

Z9 2

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JI J. Trauma Acute Care Surg.

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ER

PT J

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TI Neutrophil extracellular traps facilitate sympathetic hyperactivity by

polarizing microglia toward M1 phenotype after traumatic brain injury

SO FASEB JOURNAL

LA English

DT Article

DE microglial polarization; neutrophil extracellular traps;

sympathoexcitation; traumatic brain injury

ID HMGB1; NEUROINFLAMMATION; DYSAUTONOMIA; ASTROCYTES; MANAGEMENT; CELLS;

STORM

AB Traumatic brain injury (TBI), particularly diffuse axonal injury (DAI), often results in sympathetic hyperactivity, which can exacerbate the prognosis of TBI patients. A key component of this process is the role of neutrophils in causing neuroinflammation after TBI by forming neutrophil extracellular traps (NETs), but the connection between NETs and sympathetic excitation following TBI remains unclear. Utilizing a DAI rat model, the current investigation examined the role of NETs and the HMGB1/JNK/AP1 signaling pathway in this process. The findings revealed that sympathetic excitability intensifies and peaks 3 days post-injury, a pattern mirrored by the activation of microglia, and the escalated NETs and HMGB1 levels. Subsequent in vitro exploration validated that HMGB1 fosters microglial activation via the JNK/AP1 pathway. Moreover, in vivo experimentation revealed that the application of anti-HMGB1 and AP1 inhibitors can mitigate microglial M1 polarization post-DAI, effectively curtailing sympathetic hyperactivity. Therefore, this research elucidates that post-TBI, NETs within the PVN may precipitate sympathetic hyperactivity by stimulating M1 microglial polarization through the HMGB1/JNK/AP1 pathway.

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NR 53

TC 0

Z9 0

U1 6

U2 7

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J9 FASEB J

JI Faseb J.

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WC Biochemistry & Molecular Biology; Biology; Cell Biology

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ER

PT J

AU He, N

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TI New insights into the biological roles of immune cells in neural stem

cells in post-traumatic injury of the central nervous system

SO NEURAL REGENERATION RESEARCH

LA English

DT Review

DE B cells; central nervous system injury; macrophages; microglia; neural

stem cells; spinal cord injury; T cells; traumatic brain injury???????

ID SPINAL-CORD-INJURY; TRAUMATIC BRAIN-INJURY; ACTIVATED T-CELLS;

MACROPHAGE-DEPLETION; INFLAMMATORY RESPONSE; MICROGLIAL DEPLETION; ADULT

NEUROGENESIS; CNS REMYELINATION; OLIGODENDROGENESIS; DIFFERENTIATION

AB Traumatic injuries in the central nervous system, such as traumatic brain injury and spinal cord injury, are associated with tissue inflammation and the infiltration of immune cells, which simultaneously affect the self-renewal and differentiation of neural stem cells. However, the tissue repair process instigated by endogenous neural stem cells is incapable of restoring central nervous system injuries without external intervention. Recently, resident/peripheral immune cells have been demonstrated to exert significant effects on neural stem cells. Thus, the restoration of traumatic injuries in the central nervous system by the immune intervention in neural stem cells represents a potential therapeutic method. In this review, we discuss the roles and possible mechanisms of immune cells on the self-renewal and differentiation of neural stem cells along with the prognosis of central nervous system injuries based on immune intervention. Finally, we discuss remaining research challenges that need to be considered in the future. Further elucidation of these challenges will facilitate the successful application of neural stem cells in central nervous system injuries.

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TC 2

Z9 3

U1 7

U2 17

PU WOLTERS KLUWER MEDKNOW PUBLICATIONS

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AU Tang, LJ

Xu, Y

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AF Tang, Linjun

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Pan, Jingjing

TI Adipose-derived stem cell exosomes ameliorate traumatic brain injury

through the NLRP3 signaling pathway

SO NEUROREPORT

LA English

DT Article

DE adipose-derived stem cells; exosomes; microglia; neuroinflammation;

NLRP3; traumatic brain injury

ID MESENCHYMAL STROMAL CELLS; INFLAMMASOME ACTIVATION; RECOVERY; DEFICITS;

STROKE

AB The exosomes of mesenchymal stem cells have immunoregulatory properties and can effectively mitigate secondary neuroinflammation due to traumatic brain injury (TBI). In this study, we found that adipose-derived stem cell exosomes (ADSCs-Exo) could reduce the inflammatory response after traumatic brain injury by reducing NLRP3 inflammasome secretion by microglial. ADSCs-Exo were monitored by Western blot and electron microscopy. An in-vitro lipopolysaccharide (LPS)-caused primary microglia model and a TBI rat model were constructed. Functional recovery was examined using the modified neurological severity score and foot fault tests. Inflammasome inactivation in LPS-stimulated microglial, ADSCs-Exo can reduce the secretion of interleukin (IL)-1 & beta;, IL-6 and tumor necrosis factor & alpha;. Compared with PBS-processed controls, the sensorimotor functional recovery was significantly improved by exosome treatment after injury at 14-35 days. Additionally, NLRP3 inflammasome was stimulated within 24 h after TBI. ADSCs-Exo application led to remarkable down-expression of NLRP3 and caspase-1. ADSCs-Exo can ameliorate LPS-induced inflammatory activation by reducing microglial pro-inflammatory cytokines. Moreover, the neuroprotective effect of ADSCs-Exo may be partially attributed to the inhibition thereof on the formation of NLRP3-mediated inflammasome. Such findings imply a potential function of ADSCs-Exo in treating TBI.

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NR 52

TC 0

Z9 0

U1 3

U2 4

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TI Neuroprotective Effects of CXCR2 Antagonist SB332235 on Traumatic Brain

Injury Through Suppressing NLRP3 Inflammasome

SO NEUROCHEMICAL RESEARCH

LA English

DT Article; Early Access

DE Traumatic brain injury; CXCR2 antagonist; SB332235; NLRP3 inflammasome;

Anti-inflammation; Microglia

ID MICROGLIA ACTIVATION; NEUROINFLAMMATION; DEFICITS; OVEREXPRESSION;

CXCL1/CXCR2; INHIBITION; MODULATION; CORTEX; DEATH; MODEL

AB The inflammatory process mediated by nucleotide-binding oligomerization domain (NOD)-like receptor family pyrin domain comprising 3 (NLRP3) inflammasome plays a predominant role in the neurological dysfunction following traumatic brain injury (TBI). SB332235, a highly selective antagonist of chemokine receptor 2 (CXCR2), has been demonstrated to exhibit anti-inflammatory properties and improve neurological outcomes in the central nervous system. We aimed to determine the neuroprotective effects of SB332235 in the acute phase after TBI in mice and to elucidate its underlying mechanisms. Male C57BL/6J animals were exposed to a controlled cortical impact, then received 4 doses of SB332235, with the first dose administered at 30 min after TBI, followed by additional doses at 6, 24, and 30 h. Neurological defects were assessed by the modified neurological severity score, while the motor function was evaluated using the beam balance and open field tests. Cognitive performance was evaluated using the novel object recognition test. Brain tissues were collected for pathological, Western blot, and immunohistochemical analyses. The results showed that SB332235 significantly ameliorated TBI-induced deficits, including motor and cognitive impairments. SB332235 administration suppressed expression of both CXCL1 and CXCR2 in TBI. Moreover, SB332235 substantially mitigated the augmented expression levels and activation of the NLRP3 inflammasome within the peri-contusional cortex induced by TBI. This was accompanied by the blocking of subsequent production of pro-inflammatory cytokines. Additionally, SB332235 hindered microglial activity induced by TBI. These findings confirmed the neuroprotective effects of SB332235 against TBI, and the involved mechanisms were in part due to the suppression of NLRP3 inflammasome activity. This study suggests that SB332235 may act as an anti-inflammatory agent to improve functional outcomes in brain injury when applied clinically.

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J9 NEUROCHEM RES

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AU Chen, SY

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AF Chen, Songyu

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Zhang, Feng

Zeng, Tao

Li, Lei

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TI Exosomes from ADSCs ameliorate nerve damage in the hippocampus caused by

post traumatic brain injury via the delivery of circ-Scmh1 promoting

microglial M2 polarization

SO INJURY-INTERNATIONAL JOURNAL OF THE CARE OF THE INJURED

LA English

DT Article

DE Exosomes; Adipose-derived mesenchymal stem cells; Traumatic brain

injury; circ-Scmh1; Microglial M2 polarization

ID MESENCHYMAL STEM-CELL; SPINAL-CORD-INJURY; RECOVERY

AB Background: Traumatic brain injury (TBI) is an urgent global health issue. Neuroinflammation, due partially to microglia, can worsen or even cause neuropsychiatric disorders after a TBI. An increasing number of studies have found that adipose-derived stem cell (ADSC) derived exosomes can alleviate many diseases by delivering noncoding RNAs including circRNA and miRNAs, but the mechanism of action remains unclear.Methods: In the present investigation, we produced a TBI mouse model and isolated exosomes from their ADSCs before and after an hypoxic pretreatment. We then used next generation sequencing (NGS) to identify differentially expressed circRNAs and luciferase report assays to determine the relationship between the different noncoding RNAs (miRNA, circRNA and mRNA).Results: The results show that we successfully isolated ADSCs which possessed a multidirectional differentiation potential. We then isolated exosomes from untreated ADSCs (Exos) and from hypoxia pretreated ADSCs (HExos). The HExos significantly decreased hippocampal nerve injury after TBI by decreasing M1 microglia mediated inflammatory cytokine expression and caused recovery of cognitive function. NGS data revealed that abnormal circ-Scmh1 expression plays a role in HExo mediated brain tissue preservation after TBI. Furthermore, luciferase report analysis found that miR-154-5p and STAT6 were the targets for circ-Scmh1. Interestingly, miR-154-5p overexpression or STAT6 inhibition reversed the circ-Scmh1 induced M2 microglial polarization. Overexpression of circ-Scmh1 increased the therapeutic effect of Exo on hippocampal nerve injury after TBI by promotion of M2 microglial polarization and decreased inflammatory induced hippocampal nerve injury. Conclusion: Taken together, we found that exosomes from ADSCs ameliorate nerve damage in the hippocampus post TBI through the delivery of circ-Scmh1 and the promotion of microglial M2 polarization.

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TI PET imaging of microglia using PBR28suv determines therapeutic efficacy

of autologous bone marrow mononuclear cells therapy in traumatic brain

injury

SO SCIENTIFIC REPORTS

LA English

DT Article

ID ACTIVATED MICROGLIAL/MACROPHAGE RESPONSE; MITOCHONDRIAL

BENZODIAZEPINE-RECEPTOR; DEPENDENT ANION CHANNEL; INFLAMMATORY RESPONSE;

ASSOCIATION; ASTROCYTES; ROLES

AB Traumatic brain injury (TBI) results in activated microglia. Activated microglia can be measured in vivo by using positron emission topography (PET) ligand peripheral benzodiazepine receptor standardized uptake values (PBR28suv). Cell based therapies have utilized autologous bone marrow mononuclear cells (BMMNCs) to attenuate activated microglia after TBI. This study aims to utilize in vivo PBR28suv to assess the efficacy of BMMNCs therapy after TBI. Seventy-two hours after CCI injury, BMMNCs were harvested from the tibia and injected via tail-vein at 74 h after injury at a concentration of 2 million cells per kilogram of body weight. There were three groups of rats: Sham, CCI-alone and CCI-BMMNCs (AUTO). One hundred twenty days after injury, rodents were imaged with PBR28 and their cognitive behavior assessed utilizing the Morris Water Maze. Subsequent ex vivo analysis included brain volume and immunohistochemistry. BMMNCs therapy attenuated PBR28suv in comparison to CCI alone and it improved spatial learning as measured by the Morris Water Maze. Ex vivo analysis demonstrated preservation of brain volume, a decrease in amoeboid-shaped microglia in the dentate gyrus and an increase in the ratio of ramified to amoeboid microglia in the thalamus. PBR28suv is a viable option to measure efficacy of BMMNCs therapy after TBI.

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TI Fundamental Neurochemistry Review: Microglial immunometabolism in

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TRICARBOXYLIC-ACID CYCLE; NADPH OXIDASE; KETOGENIC DIET;

GLUCOSE-METABOLISM; APOLIPOPROTEIN-E; TYPE-4 ALLELE

AB Traumatic brain injury (TBI) is a devastating neurological disorder caused by a physical impact to the brain that promotes diffuse damage and chronic neurodegeneration. Key mechanisms believed to support secondary brain injury include mitochondrial dysfunction and chronic neuroinflammation. Microglia and brain-infiltrating macrophages are responsible for neuroinflammatory cytokine and reactive oxygen species (ROS) production after TBI. Their production is associated with loss of homeostatic microglial functions such as immunosurveillance, phagocytosis, and immune resolution. Beyond providing energy support, mitochondrial metabolic pathways reprogram the pro- and anti-inflammatory machinery in immune cells, providing a critical immunometabolic axis capable of regulating immunologic response to noxious stimuli. In the brain, the capacity to adapt to different environmental stimuli derives, in part, from microglia's ability to recognize and respond to changes in extracellular and intracellular metabolite levels. This capacity is met by an equally plastic metabolism, capable of altering immune function. Microglial pro-inflammatory activation is associated with decreased mitochondrial respiration, whereas anti-inflammatory microglial polarization is supported by increased oxidative metabolism. These metabolic adaptations contribute to neuroimmune responses, placing mitochondria as a central regulator of post-traumatic neuroinflammation. Although it is established that profound neurometabolic changes occur following TBI, key questions related to metabolic shifts in microglia remain unresolved. These include (a) the nature of microglial mitochondrial dysfunction after TBI, (b) the hierarchical positions of different metabolic pathways such as glycolysis, pentose phosphate pathway, glutaminolysis, and lipid oxidation during secondary injury and recovery, and (c) how immunometabolism alters microglial phenotypes, culminating in chronic non-resolving neuroinflammation. In this basic neurochemistry review article, we describe the contributions of immunometabolism to TBI, detail primary evidence of mitochondrial dysfunction and metabolic impairments in microglia and macrophages, discuss how major metabolic pathways contribute to post-traumatic neuroinflammation, and set out future directions toward advancing immunometabolic phenotyping in TBI.image

In this fundamental neurochemistry review, we examine the relationship between traumatic brain injury (TBI) and immune cell metabolism, primarily focusing on microglia. TBI leads to widespread damage and chronic neurodegeneration, with secondary brain injury involving mitochondrial dysfunction and ongoing neuroinflammation. Microglia contribute to inflammation via cytokine and reactive oxygen species production chronically post-TBI, and promote long-term neurodegeneration. Metabolic pathways in these cells regulate immune responses, with metabolic adaptations driving pro- or anti-inflammatory states. However, questions about microglial mitochondrial dysfunction, the roles of specific metabolic pathways, and how immunometabolism influences chronic neuroinflammation following TBI remain. The review explores these aspects to advance understanding of immunometabolic changes in TBI.image

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TI Regulation of microglial responses after pediatric traumatic brain

injury: exploring the role of SHIP-1

SO FRONTIERS IN NEUROSCIENCE

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DT Article

DE immune responses; inflammation; neurotrauma; immune signaling; PI3K

ID CONTROLLED CORTICAL IMPACT; INTERLEUKIN-1 RECEPTOR; CELL-SURVIVAL;

ALZHEIMERS-DISEASE; IMMUNE-RESPONSE; DEFICIENT MICE; WHITE-MATTER;

HEAD-INJURY; INFLAMMATION; ACTIVATION

AB IntroductionSevere traumatic brain injury (TBI) is the world's leading cause of permanent neurological disability in children. TBI-induced neurological deficits may be driven by neuroinflammation post-injury. Abnormal activity of SH2 domain-containing inositol 5 ' phosphatase-1 (SHIP-1) has been associated with dysregulated immunological responses, but the role of SHIP-1 in the brain remains unclear. The current study investigated the immunoregulatory role of SHIP-1 in a mouse model of moderate-severe pediatric TBI.MethodsSHIP-1+/- and SHIP-1-/- mice underwent experimental TBI or sham surgery at post-natal day 21. Brain gene expression was examined across a time course, and immunofluorescence staining was evaluated to determine cellular immune responses, alongside peripheral serum cytokine levels by immunoassays. Brain tissue volume loss was measured using volumetric analysis, and behavior changes both acutely and chronically post-injury.ResultsAcutely, inflammatory gene expression was elevated in the injured cortex alongside increased IBA-1 expression and altered microglial morphology; but to a similar extent in SHIP-1-/- mice and littermate SHIP-1+/- control mice. Similarly, the infiltration and activation of CD68-positive macrophages, and reactivity of GFAP-positive astrocytes, was increased after TBI but comparable between genotypes. TBI increased anxiety-like behavior acutely, whereas SHIP-1 deficiency alone reduced general locomotor activity. Chronically, at 12-weeks post-TBI, SHIP-1-/- mice exhibited reduced body weight and increased circulating cytokines. Pro-inflammatory gene expression in the injured hippocampus was also elevated in SHIP-1-/- mice; however, GFAP immunoreactivity at the injury site in TBI mice was lower. TBI induced a comparable loss of cortical and hippocampal tissue in both genotypes, while SHIP-1-/- mice showed reduced general activity and impaired working memory, independent of TBI.ConclusionTogether, evidence does not support SHIP-1 as an essential regulator of brain microglial morphology, brain immune responses, or the extent of tissue damage after moderate-severe pediatric TBI in mice. However, our data suggest that reduced SHIP-1 activity induces a greater inflammatory response in the hippocampus chronically post-TBI, warranting further investigation.

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TI Microglia moonlighting after traumatic brain injury: aging and

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SO TRENDS IN NEUROSCIENCES

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ID ALZHEIMERS-DISEASE; AGED MICE; COGNITIVE DECLINE; SICKNESS BEHAVIOR;

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AB Most of the individuals who experience traumatic brain injury (TBI) develop neuropsychiatric and cognitive complications that negatively affect recovery and health span. Activation of multiple inflammatory pathways persists after TBI, but it is unclear how inflammation contributes to long-term behavioral and cognitive deficits. One outcome of TBI is microglial priming and subsequent hyper-reactivity to secondary stressors, injuries, or immune challenges that further augment complications. Additionally, microglia priming with aging contributes to exaggerated glial responses to TBI. One prominent inflammatory pathway, interferon (IFN) signaling, is increased after TBI and may contribute to microglial priming and subsequent reactivity. This review discusses the contributions of microglia to inflammatory processes after TBI, as well as the influence of aging and IFNs on microglia reactivity and chronic inflammation after TBI.

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TI IL-1R1 signaling in TBI: assessing chronic impacts and neuroinflammatory

dynamics in a mouse model of mild closed-head injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Neuroinflammation; Interleukin-1; Interleukin-1 receptor-1; Astrocyte;

Microglia; Traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; INTERLEUKIN-1 RECEPTOR; DAMAGE; NEUTRALIZATION;

LIPOCALIN-2; ACTIVATION; IL-1-BETA; MICROGLIA; CYTOKINES; BLOCKADE

AB Neuroinflammation contributes to secondary injury cascades following traumatic brain injury (TBI), with alternating waves of inflammation and resolution. Interleukin-1 (IL-1), a critical neuroinflammatory mediator originating from brain endothelial cells, microglia, astrocytes, and peripheral immune cells, is acutely overexpressed after TBI, propagating secondary injury and tissue damage. IL-1 affects blood-brain barrier permeability, immune cell activation, and neural plasticity. Despite the complexity of cytokine signaling post-TBI, we hypothesize that IL-1 signaling specifically regulates neuroinflammatory response components. Using a closed-head injury (CHI) TBI model, we investigated IL-1's role in the neuroinflammatory cascade with a new global knock-out (gKO) mouse model of the IL-1 receptor (IL-1R1), which efficiently eliminates all IL-1 signaling. We found that IL-1R1 gKO attenuated behavioral impairments 14 weeks post-injury and reduced reactive microglia and astrocyte staining in the neocortex, Ncorpus callosum, and hippocampus. We then examined whether IL-1R1 loss altered acute neuroinflammatory dynamics, measuring gene expression changes in the neocortex at 3, 9, 24, and 72 h post-CHI using the NanoString Neuroinflammatory panel. Of 757 analyzed genes, IL-1R1 signaling showed temporal specificity in neuroinflammatory gene regulation, with major effects at 9 h post-CHI. IL-1R1 signaling specifically affected astrocyte-related genes, selectively upregulating chemokines like Ccl2, Ccl3, and Ccl4, while having limited impact on cytokine regulation, such as Tnf alpha. This study provides further insight into IL-1R1 function in amplifying the neuroinflammatory cascade following CHI in mice and demonstrates that suppression of IL-1R1 signaling offers long-term protective effects on brain health.

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TI Repeated mild traumatic brain injury causes sex-specific increases in

cell proliferation and inflammation in juvenile rats

SO JOURNAL OF NEUROINFLAMMATION

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DT Article

DE Concussion; Dentate gyrus; Awake closed-head injury; Microglia;

Sub-granular zone; Development; mTBI; Mild traumatic brain injury

ID MEDIAL PREFRONTAL CORTEX; ADULT DENTATE GYRUS; CHRONIC STRESS; SYNAPTIC

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PERFORMANCE; HIPPOCAMPAL NEUROGENESIS; POSTCONCUSSION SYNDROME; AXONAL

INJURY

AB Childhood represents a period of significant growth and maturation for the brain, and is also associated with a heightened risk for mild traumatic brain injuries (mTBI). There is also concern that repeated-mTBI (r-mTBI) may have a long-term impact on developmental trajectories. Using an awake closed head injury (ACHI) model, that uses rapid head acceleration to induce a mTBI, we investigated the acute effects of repeated-mTBI (r-mTBI) on neurological function and cellular proliferation in juvenile male and female Long-Evans rats. We found that r-mTBI did not lead to cumulative neurological deficits with the model. R-mTBI animals exhibited an increase in BrdU + (bromodeoxyuridine positive) cells in the dentate gyrus (DG), and that this increase was more robust in male animals. This increase was not sustained, and cell proliferation returning to normal by PID3. A greater increase in BrdU + cells was observed in the dorsal DG in both male and female r-mTBI animals at PID1. Using Ki-67 expression as an endogenous marker of cellular proliferation, a robust proliferative response following r-mTBI was observed in male animals at PID1 that persisted until PID3, and was not constrained to the DG alone. Triple labeling experiments (Iba1+, GFAP+, Brdu+) revealed that a high proportion of these proliferating cells were microglia/macrophages, indicating there was a heightened inflammatory response. Overall, these findings suggest that rapid head acceleration with the ACHI model produces an mTBI, but that the acute neurological deficits do not increase in severity with repeated administration. R-mTBI transiently increases cellular proliferation in the hippocampus, particularly in male animals, and the pattern of cell proliferation suggests that this represents a neuroinflammatory response that is focused around the mid-brain rather than peripheral cortical regions. These results add to growing literature indicating sex differences in proliferative and inflammatory responses between females and males. Targeting proliferation as a therapeutic avenue may help reduce the short term impact of r-mTBI, but there may be sex-specific considerations.

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TI Efferocytosis is restricted by axon guidance molecule EphA4 via

ERK/Stat6/MERTK signaling following brain injury

SO JOURNAL OF NEUROINFLAMMATION

LA English

DT Article

DE Efferocytosis; Neuroinflammation; Traumatic brain injury; Apoptosis;

Peripheral-derived macrophages; Microglia; Eph; Ephrin; MERTK

ID PHAGOCYTOSIS; ACTIVATION; CELLS; STAT6; IL-4; RAS

AB BackgroundEfferocytosis is a process that removes apoptotic cells and cellular debris. Clearance of these cells alleviates neuroinflammation, prevents the release of inflammatory molecules, and promotes the production of anti-inflammatory cytokines to help maintain tissue homeostasis. The underlying mechanisms by which this occurs in the brain after injury remain ill-defined.MethodsWe used GFP bone marrow chimeric knockout (KO) mice to demonstrate that the axon guidance molecule EphA4 receptor tyrosine kinase is involved in suppressing MERTK in the brain to restrict efferocytosis of resident microglia and peripheral-derived monocyte/macrophages.ResultsSingle-cell RNAseq identified MERTK expression, the primary receptor involved in efferocytosis, on monocytes, microglia, and a subset of astrocytes in the damaged cortex following brain injury. Loss of EphA4 on infiltrating GFP-expressing immune cells improved functional outcome concomitant with enhanced efferocytosis and overall protein expression of p-MERTK, p-ERK, and p-Stat6. The percentage of GFP+ monocyte/macrophages and resident microglia engulfing NeuN+ or TUNEL+ cells was significantly higher in KO chimeric mice. Importantly, mRNA expression of Mertk and its cognate ligand Gas6 was significantly elevated in these mice compared to the wild-type. Analysis of cell-specific expression showed that p-ERK and p-Stat6 co-localized with MERTK-expressing GFP + cells in the peri-lesional area of the cortex following brain injury. Using an in vitro efferocytosis assay, co-culturing pHrodo-labeled apoptotic Jurkat cells and bone marrow (BM)-derived macrophages, we demonstrate that efferocytosis efficiency and mRNA expression of Mertk and Gas6 was enhanced in the absence of EphA4. Selective inhibitors of ERK and Stat6 attenuated this effect, confirming that EphA4 suppresses monocyte/macrophage efferocytosis via inhibition of the ERK/Stat6 pathway.ConclusionsOur findings implicate the ERK/Stat6/MERTK axis as a novel regulator of apoptotic debris clearance in brain injury that is restricted by peripheral myeloid-derived EphA4 to prevent the resolution of inflammation.

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TI Neuroinflammation-Modulating Agent SB1617 Enhances LC3-Associated

Phagocytosis to Mitigate Tau Pathology

SO ACS CHEMICAL NEUROSCIENCE

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DT Article

DE LC3-associated phagocytosis; microglial polarization; neuroinflammation;

proteostasis; tau pathology; traumatic brain injury

ID TRAUMATIC BRAIN-INJURY; ALZHEIMERS-DISEASE; AUTOPHAGY; STRESS;

PROGRANULIN; ACTIVATION; MICROGLIA; RUBICON; PROTEIN; ROLES

AB Tau protein aggregation and propagation in neurons and surrounding microglia are well-known risk factors for neurodegenerative diseases. Therefore, emerging therapeutic strategies that target neuroinflammatory activity in microglia have the potential to prevent tauopathy. Here, we explored the microglia-mediated neuroprotective function of SB1617 against tau aggregation. Our study revealed that SB1617-inactivated pathogenic M1-like microglia, reduced the secretion of pro-inflammatory cytokines via translational regulation, and induced microglial polarization toward the M2 phenotype and phagocytic function. Furthermore, we observed that extracellular pathogenic tau aggregates were eliminated via LC3-associated phagocytosis. The in vivo efficacy of SB1617 was confirmed in mice with traumatic brain injury in which SB1617 exerted neuroprotective effects by reducing pathogenic tau levels through microglia-mediated anti-inflammatory activity. Our results indicated that SB1617-mediated microglial surveillance with LC3-associated phagocytosis is a critical molecular mechanism in the regulation of tau proteostasis. This study provides new insights into tauopathies and directions for developing novel therapies for neurodegenerative diseases.

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WC Biochemistry & Molecular Biology; Chemistry, Medicinal; Neurosciences

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TI Microglial histone deacetylase 2 is dispensable for functional and

histological outcomes in a mouse model of traumatic brain injury

SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM

LA English

DT Article; Early Access

DE Conditional gene knockout; HDAC2; long-term sensorimotor outcomes;

microglia; neuroinflammation

ID WHITE-MATTER; MICROGLIA/MACROPHAGE POLARIZATION; CORPUS-CALLOSUM;

INHIBITORS; VULNERABILITY; ACTIVATION; CONDUCTION; EVOLUTION; RECOVERY;

AXONS

AB The Class-I histone deacetylases (HDACs) mediate microglial inflammation and neurological dysfunction after traumatic brain injury (TBI). However, whether the individual Class-I HDACs play an indispensable role in TBI pathogenesis remains elusive. HDAC2 has been shown to upregulate pro-inflammatory genes in myeloid cells under brain injuries such as intracerebral hemorrhage, thereby worsening outcomes. Thus, we hypothesized that HDAC2 drives microglia toward a pro-inflammatory neurotoxic phenotype in a murine model of controlled cortical impact (CCI). Our results revealed that HDAC2 expression was highly induced in CD16/CD32+ pro-inflammatory microglia 3 and 7d after TBI. Surprisingly, microglia-targeted HDAC2 knockout (HDAC2 miKO) mice failed to demonstrate a beneficial phenotype after CCI/TBI compared to their wild-type (WT) littermates. HDAC2 miKO mice exhibited comparable levels of grey and white matter injury, efferocytosis, and sensorimotor and cognitive deficits after CCI/TBI as WT mice. RNA sequencing of isolated microglia 3d after CCI/TBI indicated the elevation of a panel of pro-inflammatory cytokines/chemokines in HDAC2 miKO mice over WT mice, and flow cytometry showed further elevated brain infiltration of neutrophils and B cells in HDAC2 miKO mice. Together, this study does not support a detrimental role for HDAC2 in microglial responses after TBI and calls for investigation into alternative mechanisms.

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TI Early Life Stress Negatively Impacts Spatial Learning Acquisition and

Increases Hippocampal CA1 Microglial Activation After a Mild Traumatic

Brain Injury in Adult Male Rats

SO JOURNAL OF NEUROTRAUMA

LA English

DT Article; Early Access

DE controlled cortical impact; maternal separation; microglia;

neurogenesis; spatial learning

ID MATERNAL SEPARATION; ENVIRONMENTAL ENRICHMENT; MAZE PERFORMANCE; HPA

AXIS; NEUROGENESIS; CORTICOSTERONE; BEHAVIOR; EXPERIENCES; MATURATION;

RECOVERY

AB Early life stress (ELS) affects neurogenesis and spatial learning, and increases neuroinflammation after a pediatric mild traumatic brain injury (mTBI). Previous studies have shown that ELS has minimal effects in juveniles but shows age-dependent effects in adults. Hence, we aimed to evaluate the effects of ELS in adult male rats after an mTBI. Maternal separation for 180 min per day (MS180) during the first 21 post-natal (P) days was used as the ELS model. At P110, the rats were subjected to a mild controlled cortical impact injury (2.6 mm) or sham surgery. Spatial learning was evaluated in the Morris water maze (MWM) 14 days after surgery and both microglial activation and neurogenesis were quantified. The results indicate that MS180 + mTBI, but not control (CONT) + mTBI, rats show deficiencies in the acquisition of spatial learning. mTBI led to comparable increases in microglial activation in both the hilus and cortical regions for both groups. However, MS180 + mTBI rats exhibited a greater increase in microglial activation in the ipsilateral CA1 hippocampus subfield compared with CONT + mTBI. Interestingly, for the contralateral CA1 region, this effect was observed exclusively in MS180 + mTBI. ELS and mTBI independently caused a decrease in hippocampal neurogenesis and this effect was not increased further in MS180 + mTBI rats. The findings demonstrate that ELS and mTBI synergistically affect cognitive performance and neuroinflammation, thus supporting the hypothesis that increased inflammation resulting from the combination of ELS and mTBI could underlie the observed effects on learning.

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J9 J NEUROTRAUM

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PT J

AU Zhang, CH

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TI Deferoxamine Induces Autophagy Following Traumatic Brain Injury via

TREM2 on Microglia

SO MOLECULAR NEUROBIOLOGY

LA English

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DE Traumatic brain injury; Triggering receptor expressed on myeloid cells

2; Autophagy; Microglia; Deferoxamine

ID IRON; NEUROPROTECTION; INFLAMMATION; ACTIVATION; RAPAMYCIN; DEFICITS

AB Previous studies have indicated that iron disorder, inflammation, and autophagy play an important role in traumatic brain injury (TBI). The triggering receptor expressed on myeloid cells 2 (TREM2), an immunoglobulin superfamily transmembrane receptor, is involved in inflammation. However, the role of TREM2 in modulating the microglia response in TBI has been rarely investigated. The present study aimed to investigate if the iron chelator deferoxamine (DFO) could ameliorate TBI through autophagy mediated by the TREM2. TBI was developed by the controlled cortical impact (CCI) mouse model and stretching of individual primary cortical microglia taken from the tissue of the rat brain. DFO was intraperitoneally used for intervention. Western blotting assay, qRT-PCR, TUNEL staining, immunofluorescence staining, confocal microscopy analysis, transmission electron microscopy, H&E staining, brain water content measurement, and the neurobehavioral assessments were performed. TREM2 expression was up-regulated in cortex of TBI mice model and in microglia stretching model, which was attenuated by DFO. After the mice were subjected to CCI, DFO treatment significantly up-regulated the protein levels of autophagy compared with the TBI group at 3 days and caused an increase of autophagic vacuoles. Treatment with DFO reduced TBI-induced cell apoptosis, cerebral edema, neuroinflammation, and motor function impairment in mice, at least partly via the mTOR signaling pathway that facilitates the TREM2 activity. The results indicated that the maintenance of iron homeostasis by DFO plays neuroprotection by modulating the inflammatory response to TBI through TREM2-mediated autophagy. This study suggested that TREM2-mediated autophagy might be a potential target for therapeutic intervention in TBI.

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