**Supplementary Table 1.** Summary of the most relevant preclinical studies evaluating the therapeutic effects of ISL administration to animals and cells subjected to diverse diseases.

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Disease** | **Animals** | **Dose range** | **Cell line** | **Dose range** | **Targets and other pathways** | **Outcomes of study** | **References** |
| ALI | C57BL/6 mice  | ISL (30 mg/kg), intraperitoneally, pretreatment for 1h | RAW 264.7 cells | ISL (5, 10, 20 µM), for 18 h | Nrf2, HO-1, glutamate-cysteine ligase catalytic subunit(GCLC), GCLM, NQO1, AMPK, inducible nitric oxidesynthzase(iNOS), cyclooxygenase-2(COX-2), NOD-like receptor thermal protein domain associated protein 3 (NLRP3), I-kappa-B-alpha(IκBα), p65, GSK-3β, IL-1β | ISL significantly alleviated lung injury, ameliorated inflammatory cell exudation and the production of ROS, and increased the expression of GSH and SOD by activating AMPK/Nrf2/ARE signaling. | (Liu et al., 2017) |
| COPD | C57BL/6N male mice, 6-8 weeks | ISL (10,20,30 mg/kg), orally, 1h before CS administration, twice a day for 4 weeks | —— | —— | p65, IκB, Nrf2, HO-1, MPO, MDA, IL-1β, TNF-α | ISL protected against CS-induced COPD by reducing inflammation and oxidative stress by regulating Nrf2 and NF-κB signaling. | (Yu et al., 2018) |
| Carrageenan-induced pleurisy | Female BALB/c mice | ISL (30 mg/kg), intraperitoneally, twice (each time interval of 12 h) for 24h  | —— | —— | Nrf2, Keap1, HO-1, NQO1, GCLC, GCLM, iNOS, COX-2, VCAM-1, JNK, ERK, p38, p65, IκBα, NLRP3, IL-1β, NADPH Oxidase 2(NOX2), NOX4 | ISL exerts protective effects on carrageenan-induced pleurisy and lung injury by exerting antioxidative activities through Nrf2 and anti-inflammatory actions through NLRP3. | (Gao et al., 2020) |
| Doxorubicin-induced hepatic damage | Adult male rats, 10 weeks | ISL (25 mg/kg), orally, given daily but 10 days prior and 10 days after doxorubicin treatment | Alpha mouse liver 12 cells | ISL (10 µM), for 24h prior post-treatment with doxorubicin  | MDA, GSH, SOD, IL-6, TNF-α, silent information regulator 1 (SIRT1), NF-κB, Nrf2, B-cell lymphoma-2(Bcl-2), Bcl-2-associated X(Bax), Caspase-3/8/9  | ISL prevented hepatocyte damage, decreased TNF-α, IL-6 as well as Bax levels, and increased GSH levels, SOD, and catalase mainly through stimulating the Nrf2 axis and suppressing NF-κB pathway. | (Al-Qahtani et al., 2022) |
| Triptolide-induced hepatotoxicity | Healthy male ICR mice | ISL (25, 50 mg/kg), orally,once daily for 7 days  | Human normal L02 hepatoc-ytes  | ISL (2.5, 5.5, 7.5 µM), for 12h  | Nrf2, NQO1, multidrug resistance protein 4 (MRP4), MRP2 | ISL effectively alleviated triptolide-induced hepatotoxicity and reduce the hepatic oxidative stress and hepatic accumulations of both endogenous bile acids and exogenous Triptolide through increasing Nrf2, NQO1, and hepatic influx and efflux transporters expression. | (Hou et al., 2018) |
| Triptolide-induced hepatotoxicity | —— | —— | Human hepatocarcinoma cell line | ISL (5, 10, 20 µM), for 12h  | ROS, GSH, Nrf2, HO-1, NQO1, MRP2 | ISL decreased ROS level, enhanced intracellular GSH content, and increased the MRP2, HO-1 and NQO1 expression possibly via activating Nrf2. | (Cao et al., 2016b) |
| Emodin-induced hepatotoxicity | Zebrafish embryo | ISL (1, 2, 4µM), for 72h | Human normal L-02 hepatocytes | ISL (10, 15, 20 µM), for 48h | Nrf2, Bax, Bcl-2, GSH, SOD, MDA, ERK, p38, Keap1, HO-1, NQO1, GST, MRP2, MRP4 | ISL effectively protected EMO-induced liver injury, reduced ROS generation and MDA generation, enhanced the levels of GSH and SOD via activating protein kinases MAPKs and Keap1-Nrf2 signaling pathway. | (Ni et al., 2022) |
| t-BHP-induced hepatocyte damage | Sprague-Dawley male rats, 6 weeks  | ISL (5, 20 mg/kg), orally, for 4 consecutive days | HepG2 cells | ISL (10, 30 µM)), for 12 h | Nrf2, ERK, HO-1, GCLC, NQO1, HO-1  | ISL significantly reduced hepatic damage, inhibited apoptosis, mitochondrial damage and ROS production by activating ERK-mediated Nrf2 pathway. | (Park et al., 2016) |
| Acute liver failure (ALF) | C57BL/6 male mice, 8 weeks | ISL (10, 20, 40 mg/kg), orally, once a day for 3 successive days. | Mouse primary hepatocytes | ISL (5, 10, 20, 40 μM), for 12, 24, and 48 h. | PGC-1α, Nrf2, HO-1, NQO1, Keap1, TNF-α, IL-6, IL-1β, iNOS, NLRP3, Caspase-1/3, Bax, Bcl-2 | ISL improves the ability of anti-oxidative stress, alleviates inflammatory reaction, apoptosis, and inhibits NLRP3 inflammasome to protect (LPS/D-GalN)-induced ALF via the PGC-1α/Nrf2 pathway. | (Wang et al., 2021) |
| Triptolide-induced hepatotoxicity | ICR maleMice, 6-8 weeks  | ISL (12.5, 25, 50mg/kg), daily for 7 days consecutively. | —— | —— | Nrf2, GSH, glutathione peroxidase (GSH-PX), SOD, catalase (CAT), MDA | ISL could protect against triptolide-induced hepatotoxicity, inhibiting MDA and restoring the levels of GSH, GSH-PX, SOD and CAT via activation of the Nrf2 pathway. | (Cao et al., 2016a) |
| Diabetes mellitus-induced aortic injury | Sprague-Dawley male rats, 6-8 weeks  | ISL(20 mg/kg) , orally, for 8 weeks. | —— | —— | Nrf2, Keap1, HO-1, IL-6, IL-10, TNF-α, Caspase-3, MDA, GSH  | ISL modulated Nrf2 /Keap1 to restore oxidative/antioxidative stress homeostasis in aorta, suppressed lipid deposition, and restored an almost normal aortic thickness. | (Alzahrani et al., 2021) |
| Ischemia-inducedmyocardial injury | C57BL/6J male mice, aged 8-10 weeks | ISL (100 mg/kg), intraperitoneally, for 3 days | —— | —— | Nrf2, HO-1, ROS, MDA, SOD, GSH-Px, HO-1, p65, IκBα, IL-6, IL-1, TNF-α | ISL significantly reduced the myocardial infarction area, enhanced the function of the heart, and alleviated myocardial oxidative stress and inflammation by activating Nrf2 /HO-1 pathway. | (Yao et al., 2022) |
| Diabetic cardiomyopathy | Male C57BL/6 mice, 8 weeks of age | ISL(10, 20mg/kg),orally, every other day for 12 weeks | Embryonic rat heart-derived H9c2 cells | ISL (2.5, 5, 10, 20, 40 μM), for 24 h  | Nrf2, HO-1, NQO1, JNK, p38, ERK, TNF-α, IL-6, IL-1β, vascular cell adhesion molecule-1(VCAM-1), Collagen-1, Bcl-2, Bax | ISL protected the cardiac function, exhibited anti-inflammatory and antioxidant stress activities via the inhibition of MAPKs and induction of Nrf2 signaling pathway respectively. | (Gu et al., 2020) |
| Acute myocardial-infarction | C57BL6J mice  | ISL(100mg/kg), intraperitoneally, pretreatment daily for 3 days | Neonatal mouse cardiomyocytes | ISL(5,25,50μmol/L), for 48h | Nrf2, ACSL4, GPX4, HO-1, 4-hydroxynonenal (4-HNE), solute carrier family 7 member 11(SLC7A11) | ISL reduced myocardial damage through the activation of the Nrf2/HO-1/SLC7A11/GPX4 pathway, which served to inhibit acute myocardial infarction by mitigating oxidative stress, mitochondrial injury, and cardiomyocyte death. | (Yao et al., 2024) |
| Acute pancreatitis | Male ICR mice, 8 weeks of age | ISL (50, 100, 200mg/kg), intraperitoneally, at the beginning of the experiment | —— | —— | Nrf2, HO-1, SOD, MDA, GSH, IL-1β, IL-6 | ISL mitigated the severity of pancreatic tissue injury and pancreatitis-associated lung injury, decreased serum amylase and lipase levels, and reduced oxidative stress injury by modulating the Nrf2 /HO-1 pathway. | (Liu et al., 2018) |
| SAP | Male C57BL/6 mice | —— | —— | —— | Nrf2, HO-1, NQO1, MDA, SOD, IL-6, TNF-α, NF-κB, IκB, Caspase-3 | ISL reduced pancreatic and intestinal injury, repaired the intestinal barrier damage, suppressed oxidative stress and the inflammatory responses via regulation of the Nrf2 /NF-κB pathway. | (Zhang et al., 2018b) |
| Inflammatory bowel disease  | —— | —— | HT-29 cell lines  | ISL (1, 5, 10, 20 μM), for 1, 3, 6, 12, 24h | IL-8, IL-1β, COX-2, Nrf2 , HO-1, NQO1, high mobility group box 1 protein (HMGB1), histone deacetylase (HDAC), NF-κB, p65, IκBα | ISL exerted Anti-inflammatory effects by suppressing the expression of inflammatory molecules such as HMGB1, IL-8, IL-1β, which may be related with the induction of HDAC activity and induced activation of Nrf2 and expression of its target genes, such as HO-1 and NQO1. | (Chi et al., 2017) |
| Colitis | C57BL/6mice  | ISL (20 mg/kg), orally, treatment for 14 days | —— | —— | Nrf2, NQO1, HO-1, TNF-α, IL-1β, IL-6, GSH-PX, MPO, MDA, SOD | ISL and SFN supplementation regulated oxidative stress and inflammation responses via activating the Keap1–Nrf2 pathway to alleviate colitis. | (Cheng et al., 2022) |
| Hypertensiverenal injury | —— | —— | Human HK-2 proximal tubule epithelial cells | ISL(2.5, 5, 10 mM), for 24, 48h | Bax, Bcl-2, cleaved Caspase-3, cleaved Caspase-9, SOD, MDA, Nrf2, HO-1, NQO1, p65, IκBα, IL-1β, TNF-α | ISL ameliorated hypertensive renal injury induced by Ang II through suppressing inflammation cytokines, excessive deposition of extracellular matrix and oxidative stress induced apoptosis via Nrf2 and NF-κB pathways. | (Xiong et al., 2018a) |
| Diabetic nephropathy | C57BL/6 mice, 6-8 weeks | ISL(10,20mg/kg), orally, every 2 days for 12 weeks | Rat renal tubular epithelial cell line | ISL (10,20 μM), pre-treatment for 1h | Nrf2, HO-1, p38, ERK, JNK, transforming growth factor-β (TGF-β), Bax, Bcl-2 | ISL prevented diabetic nephropathy, ameliorated the inflammatory reactions and oxidative stress, and regulated p38 MAPK and Nrf2 pathways downstream of SIRT1 . | (Huang et al., 2020) |
| Alzheimer’s disease  | —— | —— | BV2 cells and mouse neuroblastoma(N2a) cells | ISL (1, 5, 10, and 20 µM), for 24 h | IL-1β, IL-6, TNF-α, COX-2, iNOS, Nrf2, HO-1, NQO1, NF-κB | ISL suppresses AβO-induced inflammation and oxidative stress in BV2 cells via regulating the Nrf2 /NF-κB signaling. | (Fu and Jia, 2021) |
| PD | Male C57BL/6J mice | ISL (20mg/kg), intraperitoneally, for 14 days | —— | —— | Iba-1, IL-1β, IL-6, TNF-α, Nrf2, NQO1 | ISL significantly attenuated neurological deficits in PD, reduced neuroinflammation through the activation of the Nrf2 /NQO1 signaling pathway. | (Huang et al., 2022) |
| TBI | Male C57BL/6 mice | ISL (20mg/kg), intraperitoneally, at 1 h post-TBI | SH-SY5Y cells | ISL (20 µM), added 2 h before the cells were placed in an anaerobic chamber for 6 h. | Aquaporin4, cleaved-Caspase-3, GSH-PX, SOD, MDA, HO-1, NQO1 | ISL protect the brain injury against TBI through reducing Garcia neuro score, injury histopathology, cerebral vascular permeability and inhibiting oxidative stress via Nrf2 /HO-1 pathway. | (Zhang et al., 2019b) |
| Kainic acid-induced seizures | Male Wistar rats | ISL (20mg/kg), intraperitoneally, at 30 min, 12 h and 24 h prior to KA injection | —— | —— | cleaved-Caspase-3/9, Nrf2, HO-1, NQO1, NLRP3, TNF-α, IL-1β, IL-18 | ISL protected against cognitive impairment, suppressed synaptic dysfunction, neuronal injury and neuroinflammation via regulating the Nrf2 signaling and the NLRP3 inflammasome pathway. | (Zhu et al., 2019b) |
| Experimental intracerebralhemorrhage | Adult male Sprague-Dawley rats  | ISL (10, 20, 40 mg/kg), intraperitoneally, at 30 min, 12 h, 24 h, and 48 h after ICH induction | —— | —— | NF-κB, p-65, NLRP3, Nrf2, HO-1, NQO1, IL-1β, IL-18, Caspase-1 | ISL reduced early brain impairments and neurological deficits, attenuated the ROS production, regulating the NF-κB on the activation of NLRP3 inflammasome pathway through the triggering of Nrf2 activity and Nrf2 induced antioxidant system. | (Zeng et al., 2017) |
| Experimental diabetic neuropathy | Adult male Sprague-Dawley rats | ISL (10, 20 mg/kg), orally, for 2 weeks  | Mouse N2A neuroblastoma cells | ISL (2.5, 5 µM), for 6h | SIRT1, Nrf2, NQO1, PGC-1α, AMPK, Beclin1  | ISL attenuated the experimental diabetic neuropathy injury, reduced oxidative damage and alleviated mitochondrial impairment via SIRT1 activation, facilitating the Nrf2 directed antioxidant signaling. | (Yerra et al., 2017) |
| Viral diseases  | Male C57BL/6 mice aged 6-8 weeks | ISL (25, 50 mg/kg/day), intraperitoneally, for 4 days  | A549 cells,THP1 cells | ISL (50 μM) was incubated for 1.5h, and then ISL was replaced, for another 8.5h incubation  | Nrf2, HO-1, p62, GCLM, SQSTM1  | ISL has antiviral and anti-inflammatory effects in virus infections, which are associated with its ability to activate Nrf2. | (Wang et al., 2023a) |
| Acute myeloid leukemia | —— | —— | Human promyelocytic leukemia cells  | ISL (2.5, 5, 10 μg/mL), for 72 h | ROS, MDA, GSH, CAT, GST, GSH-PX, Nrf2, NQO1  | ISL modulate intercellular redox homeostasis and facilitate differentiation as a differentiation-inducing agent possibly via Nrf2 /ARE pathway. | (Chen et al., 2013a) |
| Gallbladder cancer(GBC) | BALB/c nude mice | ISL(50mg/kg), orally, every 2 days for 28 days | Human GBC SGC996 cell line | ISL(30,60,90mmol/L)for24h,48h,72h | Nrf2, Keap1, P62, gluta thione peroxidase 4 (GPX4), Acyl-CoA synthetase long-chain family member 8 (ACSL8), haem oxygenase-1(HMOX1) | ISL triggered ferroptosis in GBC through activation of p62-Keap1-Nrf2-HMOX1 signaling and downregulation of GPX4. | (Wang et al., 2023b) |
| Rheumatoid arthritis(RA) | Sprague-Dawley rats | ISL (10 mg/kg), local injection, injections into the right knee for 3 days | C20A4 human chondrocyte cell line | ISL (3,5,10 μM), pretreated for 1 h | HO-1, Bax, Bcl-2, Caspase-3, iNOS, PGC-1α, Nrf2, SIRT1  | ISL exerted anti-oxidation, anti-inflammation, anti-apoptosis through activation Nrf2/HO-1 pathway in RA. | (Hung et al., 2024) |
| Chronic prostatitis and chronic pelvic pain syndrome | Nonobese diabetic mice | ISL(50,100,200mg/kg), intraperitoneally, treatment started two days before the subsequent immunization | Mouse macrophage RAW 264.7 cells | ISL (20μmol/ml), for 4 h | Nrf2, HO-1, NLRP3, Caspase -1, IL-1β, Nrf2, SOD, MDA | ISL attenuated experimental autoimmune prostatitis through activation Nrf2 and downregulating the NLRP3 inflammasome pathway. | (Feng et al., 2024) |

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