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Corrigendum: The role of cholesterol and mitochondrial bioenergetics in activation of the inflammasome in IBD

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A Corrigendum on:

The role of cholesterol and mitochondrial bioenergetics in activation of the inflammasome in IBD

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In the published article, there was an error in the legend for

Table 1 Dietary phytochemicals and their restorative effects on mitochondrial function and lipid homeostasis as published. ↓ increase; ↑ decrease (on table footnotes). The corrected legend appears below.

[↑ increase; ↓ decrease]

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The authors apologize for these errors and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

TABLE 1 Dietary phytochemicals and their restorative effects on mitochondrial function and lipid homeostasis.

Phytochemical (Source)	Outcome	Proposed pathway	Model	Reference
Resveratrol (Grape, wine, peanut, and cranberry)	Anti-inflammatory protection and oxidative stress inhibition against intestinal inflammation. Anti-inflammatory effects. Protective effects against the alterations of mitochondrial function and oxidative stress. ↓Disease activity and ↑Quality of life in UC patients at least partially through the ↓Oxidative stress.	Nrf2 activation: ↑Oxygenase-1 (HO-1) mRNA, ↓ROS production, and ↑PPAR-γ accumulation. ↑Nrf2, ↓IL-1β, and ↑IL-11. ↑Intracellular ATP, protective effects against ↓Δψm induced by INDO.	cytokine-stimulated (IL-1α, TNF-α, IFN-γ) HT-29 cells <i>In vitro</i> UC model in Caco-2 cells challenged with TNF-α.	(147) (148)
	Anti-atherosclerotic effects: ↓FA and MAG intestinal accumulation. Restoration of succinate and lactic acid levels.	-	Intestinal epithelial Caco-2 cells induced by indomethacin (INDO).	(149)
		Abolishes oleate-triggered lipid, total cholesterol, and esterified cholesterol accumulation by activating PPAR-α and PPAR-γ signaling.	Prospective, randomized, double-blind, placebo-controlled study in UC patients. Supplements (containing 500 mg trans-resveratrol) or placebo capsules.	(150)
			<i>ApoE</i> null mice fed with a high-fat diet (HFD) (AS) and resveratrol intervention.	(151)
Quercetin (onion, apple grape, and citrus fruits)	Protective effects against the alterations of mitochondrial function and oxidative stress. Mitochondrial protective effects against and maintenance of gastrointestinal mucosal renewing regulating apoptosis. ↓NLRP3 inflammasome activation and ↓Mitochondrial damage.	↑Intracellular ATP, protective effects against ↓Δψm induced by INDO, and inhibition of the inhibitory effects of INDO and rotenone on complex I. Prevents Ca ²⁺ mobilization induced by INDO and its consequences, including ↑Caspase-3 and caspase-9 activation and cytochrome C release. ↓Activity of caspase-1 and ↓Secretion of IL-1β and ↓IL-18 via NLRP3 inflammasome. Improvement in Δψm, blocking cytochrome C release, ↓O ₂ consumption, ↓ mtDNA cytosolic content, and ↓ ROS level.	Intestinal epithelial Caco-2 cells induced by INDO. Intestinal epithelial Caco-2 cells induced by INDO.	(149) (152)
	Intestinal anti-inflammatory effects via Nrf2/HO-1		Caco-2 cells infected by <i>Escherichia coli</i> O157:H7 ↓TNF-α, IFN-γ, and IL-6. Nuclear Nrf2 accumulation ↑ HO-1 expression in colonic Mø.	(153)
Sulforaphane (cruciferous vegetables)	Antioxidant and anti-inflammatory effects, ↑ Mitochondrial bioenergetic function upon cholesterol-induced pancreatic β-cell dysfunction. ↓Intestinal permeability upon LPS, ↓Oxidative stress, ↓Inflammation, and ↓apoptosis.	Improving ATP turnover, spare capacity, and impairment of the electron flow at complexes I, II, and IV. ↓NFκB pathway. ↑SIRT1 and ↑PGC-1α expression. ↑ Antioxidant enzymes of the Nrf2 pathway and ↓Lipid peroxidation induced by cholesterol.	T cell-dependent colitis model induced by the adoptive transfer of <i>naive</i> T cells into <i>Rag1</i> null mice and DSS-induced colitis mice model. Min6 cells, a β-cell line exposed to high concentration of cholesterol. LPS-induced Caco-2 <i>in vitro</i> model.	(154) (155) (156)

(Continued)

TABLE 1 Continued

Phytochemical (Source)	Outcome	Proposed pathway	Model	Reference
Dried apple peel polyphenols (DAPP)	↓DSS-induced damage, ↓Pro-inflammatory factors, ↓Oxidative markers, and ↓ROS. ↓Mitochondria-dependent cell death, ↑β-oxidation, ↑Mitochondrial bioenergetics, and ↓Alteration in mitochondrial morphology.	Activating the AMPK/SIRT1/PGC-1 pathway. ↓TNF-α, COX-2, and iNOS.	In vivo model of DSS-induced colitis in male C57BL6 mice.	(157>)
Strawberry Ellagitannin-Rich Extract (S-ET)	↓ HFD effects in rats, ↓Body weight, ↓Relative mass of the epididymal pad, ↓Hepatic fat, ↓Oxidized glutathione, ↓TG, ↓Total cholesterol, and ↓Thiobarbituric acid-reactive substances concentrations and improve blood plasma parameters.	↓H ₂ O ₂ and SOD2 protein expression and ↑8-oxoguanine DNA glycosylase 1 (OGG1) expression. ↑CPT-1 and ACADL, ATP production, and PGC-1α. ↓NF-κB and AP-1.	HFD supplemented with S-ET) in Male Wistar rats. In vivo model	(158)
Luteolin (carrot, pepper, celery, spinach, and parsley)	Antioxidants and anti-inflammatory effects. Anti-inflammatory effects. ↓Lipid accumulation.	↑ Nrf2 and ↓iNOS, IL-6, and TNF-α expression. ↓NLRP3 expression via disruption of IL-17A signaling. ↓LXR-dependent SREBP-1c expression and intracellular lipid levels. ↓LXR-induced ABCA1 expression in Mø.	DSS-induced UC C57BL/6 mouse model. DSS-induced colitis C57BL/6 mice model. HepG2 cells and RAW264.7 Mø stimulated LXRx/β agonist (T0901317).	(159) (160) (161)
Sesamin (<i>Sesamum indicum</i> seeds)	Reparative effects of intestinal barrier injury. Anti-atherosclerotic effects: ↓oxLDL-elicted lipid accumulation and ↑ HDL-mediated cholesterol efflux. Antioxidants and anti-inflammatory effects.	↓MAPK/NF-κB/MLCK t activating indirectly Nrf2 signaling pathways. ↑PPARγ-dependent ABCG1 mRNA levels. Cytoprotective effect via Glutathione-S-transferase (GSH)-mediated ROS scavenger. Nrf2/ARE signaling activation dependent on ERK and AKT activation.	Ethanol-induced intestinal barrier damage in a Caco-2 cell monolayer model. RAW264.7 Mø stimulated with oxLDL and sesamin. Caco-2 cells stimulated by H ₂ O ₂	(162) (163) (164)
	↓Cholesterol absorption by enterocytes. ↓ Hepatic lipogenic genes expression. Antagonist ligand of LXRx.	↑LXR-induced ABCA1/G1 expression.	LS174T colonic epithelial cells with LXRx agonist (T090) treatment.	(165)

[↑ increase; ↓ decrease]

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