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

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KEYWORDS

IBD - inflammatory bowel disease, intracellular cholesterol accumulation, mitochondrial dysfunction, inflammasome, NLRP3 inflammasome, diet phytochemicals

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]

In the published article, there was an error in the Funding statement. This work was funded by the Agencia Nacional de Investigación y Desarrollo (ANID)/PhD fellowship #21220889 (JA), and #21200669 (NG), FONDECYT Grants #1120702 (MAH), #11201322 (FAU), MiBi: interdisciplinary group on mitochondrial targeting and bioenergetics ACT 210097 (FAU); FONDECYT postdoctoral grant #3210367 (KD-C). The correct Funding statement appears below.

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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.

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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.	Nrf2 activation: ↑Oxygenase-1 (HO-1) mRNA, ↓ROS production, and ↑PPAR-γ accumulation.	cytokine-stimulated (IL-1α, TNF-α, IFN-γ) HT-29 cells	(147)
	Anti-inflammatory effects.	↑Nrf2, ↓IL-1β, and ↑IL-11.	*In vitro* UC model in Caco-2 cells challenged with TNF-α.	(148)
	Protective effects against the alterations of mitochondrial function and oxidative stress.	↑Intracellular ATP, protective effects against ↓Δψm induced by INDO.	Intestinal epithelial Caco-2 cells induced by indomethacin (INDO).	(149)
	↓Disease activity and ↑Quality of life in UC patients at least partially through the ↓Oxidative stress.	-	Prospective, randomized, double-blind, placebo-controlled study in UC patients. Supplements (containing 500 mg trans-resveratrol) or placebo capsules.	(150)
	Anti-atherosclerotic effects: ↓FA and MAG intestinal accumulation. Restoration of succinate and lactic acid levels.		*ApoE* null mice fed with a high-fat diet (HFD) (AS) and resveratrol intervention.	(151)
		Abolishes oleate-triggered lipid, total cholesterol, and esterified cholesterol accumulation by activating PPAR-α and PPAR-γ signaling.	RAW 264.7 mice Mø treated with oleate and resveratrol.	
Quercetin (onion, apple grape, and citrus fruits)	Protective effects against the alterations of mitochondrial function and oxidative stress.	↑Intracellular ATP, protective effects against ↓Δψm induced by INDO, and inhibition of the inhibitory effects of INDO and rotenone on complex I.	Intestinal epithelial Caco-2 cells induced by INDO.	(149)
	Mitochondrial protective effects against and maintenance of gastrointestinal mucosal renewing regulating apoptosis.	Prevents Ca^{2+} mobilization induced by INDO and its consequences, including ↑Caspase-3 and caspase-9 activation and cytochrome C release.	Intestinal epithelial Caco-2 cells induced by INDO.	(152)
	↓NLRP3 inflammasome activation and ↓Mitochondrial damage.	↓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.	Caco-2 cells infected by *Escherichia coli* O157:H7	(153)
		↓TNF-α, IFN-γ, and IL-6. Nuclear Nrf2 accumulation ↑ HO-1 expression in colonic Mø.		
	Intestinal anti-inflammatory effects via Nrf2/HO-1		T cell-dependent colitis model induced by the adoptive transfer of *naive* T cells into *Rag1* null mice and DSS-induced colitis mice model.	(154)
Sulforaphane (cruciferous vegetables)	Antioxidant and anti-inflammatory effects, ↑ Mitochondrial bioenergetic function upon cholesterol-induced pancreatic β-cell dysfunction.	Improving ATP turnover, spare capacity, and impairment of the electron flow at complexes I, II, and IV. ↓NFκB pathway.	Min6 cells, a β-cell line exposed to high concentration of cholesterol.	(155)
	↓Intestinal permeability upon LPS, ↓Oxidative stress, ↓Inflammation, and ↓apoptosis.	↑SIRT1 and ↑PGC-1α expression. ↑ Antioxidant enzymes of the Nrf2 pathway and ↓Lipid peroxidation induced by cholesterol.	LPS-induced Caco-2 *in vitro* model.	(156)

(Continued)

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TABLE 1 Continued

Phytochemical (Source)	Outcome	Proposed pathway	Model	Reference
		Activating the AMPK/SIRT1/PGC-1 pathway.		
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.	↓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_2O_2 and SOD2 protein expression and ↑8-oxoguanine DNA glycosylase 1 (OGG1) expression. ↑CPT-1 and ACADL, ATP production, and PGC-1α. ↓NF-kB 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.	↑ Nrf2 and ↓iNOS, IL-6, and TNF-α expression.	DSS-induced UC C57BL/6 mouse model.	(159)
	Anti-inflammatory effects.	↓NLRP3 expression via disruption of IL-17A signaling.	DSS-induced colitis C57BL/6 mice model.	(160)
	↓Lipid accumulation.	↓LXR-dependent SREBP-1c expression and intracellular lipid levels. ↓LXR-induced ABCA1 expression in Mø.	HepG2 cells and RAW264.7 Mø stimulated LXRα/β agonist (T0901317).	(161)
	Reparative effects of intestinal barrier injury.	↓MAPK/NF-κB/MLCK t activating indirectly Nrf2 signaling pathways.	Ethanol-induced intestinal barrier damage in a Caco-2 cell monolayer model.	(162)
Sesamin (*Sesamum indicum* seeds)	Anti-atherosclerotic effects: ↓oxLDL-elicited lipid accumulation and ↑ HDL-mediated cholesterol efflux.	↑ PPARγ-dependent ABCG1 mRNA levels.	RAW264.7 Mø stimulated with oxLDL and sesamin.	(163)
	Antioxidants and anti-inflammatory effects.	Cytoprotective effect via Glutathione-S-transferase (GSH)-mediated ROS scavenger.	Caco-2 cells stimulated by H_2O_2	(164)
		Nrf2/ARE signaling activation dependent on ERK and AKT activation.		
	↓Cholesterol absorption by enterocytes. ↓ Hepatic lipogenic genes expression. Antagonist ligand of LXRα.	↑LXR-induced ABCA1/G1 expression.	LS174T colonic epithelial cells with LXRα agonist (T090) treatment.	(165)

[↑ increase; ↓ decrease]

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