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Corrigendum: The role of cholesterol and mitochondrial bioenergetics in activation of the inflammasome in IBD (Front. Immunol., (2022), 13, 1028953, 10.3389/fimmu.2022.1028953)

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Published in:
Frontiers in Immunology

DOI:
[10.3389/fimmu.2024.1384162](https://doi.org/10.3389/fimmu.2024.1384162)

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2024

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Citation for published version (APA):

Astorga, J., Gasaly, N., Dubois-Camacho, K., De la Fuente, M., Landskron, G., Faber, K. N., Urra, F. A., & Hermoso, M. A. (2024). Corrigendum: The role of cholesterol and mitochondrial bioenergetics in activation of the inflammasome in IBD (Front. Immunol., (2022), 13, 1028953, 10.3389/fimmu.2022.1028953). *Frontiers in Immunology*, 15, Article 1384162. <https://doi.org/10.3389/fimmu.2024.1384162>

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Frontiers Media SA, Switzerland

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RECEIVED 08 February 2024

ACCEPTED 12 February 2024

PUBLISHED 26 February 2024

CITATION

Astorga J, Gasaly N, Dubois-Camacho K, De la Fuente M, Landskron G, Faber KN, Urra FA and Hermoso MA (2024) Corrigendum: The role of cholesterol and mitochondrial bioenergetics in activation of the inflammasome in IBD.

Front. Immunol. 15:1384162.

doi: 10.3389/fimmu.2024.1384162

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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

By Astorga J, Gasaly N, Dubois-Camacho K, De la Fuente M, Landskron G, Faber KN, Urra FA and Hermoso MA (2022) *Front. Immunol.* 13:1028953. doi: 10.3389/fimmu.2022.1028953

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.

FUNDING

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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.	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.	<i>In vitro</i> 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.	Abolishes oleate-triggered lipid, total cholesterol, and esterified cholesterol accumulation by activating PPAR-α and PPAR-γ signaling.	<i>ApoE</i> null mice fed with a high-fat diet (HFD) (AS) and resveratrol intervention. RAW 264.7 mice Mø treated with oleate and resveratrol.	(151)
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 <i>Escherichia coli</i> O157:H7	(153)
	Intestinal anti-inflammatory effects via Nrf2/HO-1	↓TNF-α, IFN-γ, and IL-6. Nuclear Nrf2 accumulation ↑ HO-1 expression in colonic Mø.	T cell-dependent colitis model induced by the adoptive transfer of naive T cells into <i>Rag1</i> 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 <i>in vitro</i> model.	(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.	<i>In vivo</i> model of DSS-induced colitis in male C57BL/6 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. <i>In vivo</i> 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 (<i>Sesamum indicum</i> 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. Nrf2/ARE signaling activation dependent on ERK and AKT activation.	Caco-2 cells stimulated by H ₂ O ₂	(164)
	↓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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