Suppression of high-fat diet-induced obesity-associated liver mitochondrial dysfunction by docosahexaenoic acid and hydroxytyrosol co-administration

Por: Ortiz, M (Ortiz, Macarena)[1]; Soto-Alarcon, SA (Soto-Alarcon, Sandra A.)[2]; Orellana, P (Orellana, Paula)[2]; Espinosa, A (Espinosa, Alejandra)[3]; Campos, C (Campos, Cristian)[3]; Lopez-Arana, S (Lopez-Arana, Sandra)[2]; Rincon, MA (Rincon, Miguel A.)[4]; Illesca, P (Illesca, Paola)[5]; Valenzuela, R (Valenzuela, Rodrigo)[2]; Videla, LA (Videla, Luis A.)[6]

Ver número de ResearcherID y ORCID de Web of Science

DIGESTIVE AND LIVER DISEASE
Volumen: 52
Número: 8
Páginas: 895-904
DOI: 10.1016/j.dld.2020.04.019
Fecha de publicación: AUG 2020
Tipo de documento: Article

Ver impacto de la revista

Abstract

Objective: Obesity-induced by high-fat diet (HFD) is associated with liver steatosis, oxidative stress and mitochondrial dysfunction, which can be eluded by the co-administration of the lipid metabolism modulator docosahexaenoic acid (DHA) and the antioxidant hydroxytyrosol (HT).

Methods: C57BL/6J mice fed a HFD were orally administered either with vehicle, DHA, HT or DHA+ HT for 12 weeks. We measured parameters related to insulin resistance, serum lipid levels, liver fatty acid (FA) content and steatosis score, concomitantly with those associated with mitochondrial energy functions modulated by the transcriptional coactivator PGC-1a.

Results: HFD induced insulin resistance, liver steatosis with n-3 FA depletion, and loss of mitochondrial respiratory functions with diminished NAD(+) /NADH ratio and ATP levels compared with CD, with the parallel decrease in the expression of the components of the PGC-1 alpha cascade, namely, PPAR-alpha, FGF21 and AMPK, effects that were not observed in mice subjected to DHA and HT co-administration.

Conclusions: Data presented indicate that the combination of DHA and HT prevents the development of liver steatosis and the associated mitochondrial dysfunction induced by HFD, thus strengthening the significance of this protocol as a therapeutic strategy avoiding disease evolution into more irreversible forms characterised by the absence of adequate pharmacological therapy in human obesity. (c) 2020 Published by Elsevier Ltd on behalf of Editrice Gastroenterologica Italiana S.r.l.

Palabras clave

Palabras clave de autor: High-fat diet; Liver steatosis; Mitochondrial dysfunction; Docosahexaenoic acid; Hydroxytyrosol
KeyWords Plus:NF-KAPPA-B; INDUCED HEPATIC STEATOSIS; PPAR-ALPHA; OXIDATIVE STRESS; NONALCOHOLIC STEATOHEPATITIS; DNA-BINDING; N-3 LCPUFA; RECEPTOR; DISEASE; OIL

Información del autor

Dirección para petición de copias:
Universidad de Chile Univ Chile, Fac Med, Nutr Dept, Independencia 1027, Santiago 70000, Chile.
Dirección correspondiente: Valenzuela, R (corresponding author)

+ Univ Chile, Fac Med, Nutr Dept, Independencia 1027, Santiago 70000, Chile.

Direcciones:

+ [1] Catholic Univ Maule, Fac Hlth Sci, Nutr & Dietet Sch, Curico, Chile
+ [2] Univ Chile, Fac Med, Nutr Dept, Independencia 1027, Santiago 70000, Chile
+ [3] Univ Chile, Fac Med, Dept Med Technol, Santiago, Chile
+ [4] Univ Chile, Inst Nutr & Food Technol, Santiago, Chile
+ [5] Univ Litoral, Fac Biochem, Biochem Dept, Santa Fe, Argentina
+ [6] Univ Chile, Fac Med, Inst Biomed Sci, Mol & Clin Pharmacol Program, Santiago, Chile

Direcciones de correo electrónico:rvalenzuelab@med.uchil.ecl

Financiación

Entidad financiadora	Número de concesión
	11140174

Ver texto de financiación

Editorial

ELSEVIER SCIENCE INC, STE 800, 230 PARK AVE, NEW YORK, NY 10169 USA

Información de la revista

- **Impact Factor:** Journal Citation Reports

Categorías / Clasificación

Áreas de investigación: Gastroenterology & Hepatology
Categorías de Web of Science: Gastroenterology & Hepatology

Información del documento

Idioma: English

Número de acceso: WOS:000551553000019
ID de PubMed: 32620521
ISSN: 1590-8658
eISSN: 1878-3562