A combined docosahexaenoic acid-thyroid hormone protocol upregulates rat liver beta-Klotho expression and downstream components of FGF21 signaling as a potential novel approach to metabolic stress conditions

Por: Vargas, R (Vargas, R.)[1]; Riquelme, B (Riquelme, B.)[1]; Fernandez, J (Fernandez, J.)[1]; Videla, LA(Videla, L. A.)[1]
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Resumen

Liver preconditioning by a docosahexaenoic acid (DHA) and triiodothyronine (T-3) combined protocol underlies peroxisome-proliferator activated receptor alpha (PPAR alpha)-fibroblast growth factor 21 (FGF21) upregulation, the study of the regulatory mechanisms involved being the aim of this work. Combined DHA (daily doses of 300 mg kg(-1) for 3 days)-T-3 (0.05 mg kg(-1) at the fourth day) administration elicited higher levels of liver DHA and serum T-3, with enhanced hepatic nuclear/cytosolic PPAR alpha ratios, upregulation of FGF21 and beta-Klotho expression, and a small reduction in that of FGF receptor 1 (FGFR1), compared with the respective controls. Concomitantly, the components of the FGF21 cascade extracellular-signal-regulated kinase 1/2 (ERK1/2), FGF receptor substrate 2 alpha (FRS2 alpha), cFos, ribosomal S6 kinase 1 (RSK1), liver kinase B1 (LKB1), and AMP-activated protein kinase (AMPK) were activated. The upregulation of liver PPAR alpha-FGF21-AMPK signaling by the combined DHA-T-3 protocol resulted in values significantly higher than those elicited by the addition of the data obtained for DHA and T-3 alone. It is concluded that combined DHA-T-3 supplementation achieves synergistic effects on liver PPAR alpha-FGF21-AMPK signaling, which may result in significant metabolic changes associated with energy expenditure that are of importance in the treatment of obesity and other metabolic disorders.

Palabras clave

KeyWords Plus:GROWTH-FACTOR 21; HIGH-FAT DIET; PPAR-ALPHA; ENERGY-METABOLISM; RSK FAMILY; IN-VIVO; PROTEIN; ACTIVATION; FIBROBLAST-GROWTH-FACTOR-21; DEPENDENCY

Información del autor

Dirección para petición de copias: Videla, LA (autor para petición de copias)

+ Univ Chile, Fac Med, Inst Biomed Sci, Mol & Clin Pharmacol Program, Santiago, Chile.

Direcciones:

+ [1] Univ Chile, Fac Med, Inst Biomed Sci, Mol & Clin Pharmacol Program, Santiago, Chile

Direcciones de correo electrónico:lvidela@med.uchilecl

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