EuroSciCon

11th International Conference on

Endocrinology and Diabetology

August 09-10, 2018
Madrid, Spain

Mi Ock Lee et al., J Clin Mol Endocrinol 2018, Volume 3
DOI: 10.21767/2572-5432-C2-006

LIVER-SPECIFIC DELETION OF ROR α AGGRAVATES DIET-INDUCED NONALCOHOLIC STEATOHEPATITIS BY INDUCING MITOCHONDRIAL DYSFUNCTION

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Mitochondrial dysfunction may play a key role in the progression of steatosis to nonalcoholic steatohepatitis (NASH); however, the molecular mechanism that controls the structure and function of mitochondria in NASH is not clearly understood. Here, we demonstrated that RORα is a regulator of expression of BNIP3 and PGC-1α, and thereby enhances mitochondrial quality. First, we observed that liver-specific RORα knockout mice (RORα-LKO) were more susceptible to high-fat diet-induced NASH compared with control, probably due to mitochondrial dysfunction. Concordantly, mitochondrial fission in response to nutrient stimuli was abolished with downregulation of BNIP3 and Phospho-DRP1 in the hepatocytes of RORα-LKO. RORα enhanced oxygen consumption rate and expression of genes associated with mitochondrial quality control. Finally, we observed the positive correlation of the expression levels of BNIP3 and PGC-1α with those of RORα in patients with steatohepatitis. Together, we demonstrated that RORα mediates mitochondrial quality under nutrient-overloaded conditions and propose RORα as a potential therapeutic target in treatment of NASH.

Biography

Mi Ock Lee received her undergraduate and Master's Degree in Pharmacy from Seoul National University (SNU), Republic of South Korea; pursued PhD in Pharmacology from University of Minnesota, USA. She is presently working as a Professor at the SNU- College of Pharmacy. She worked as a Postdoctoral Researcher at Sanford Burnham Prebys Medical Discovery Institute, SanDiego (USA). She also worked as an Assistant Professor at the Yonsei University College of Medicine, Republic of South Korea.

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