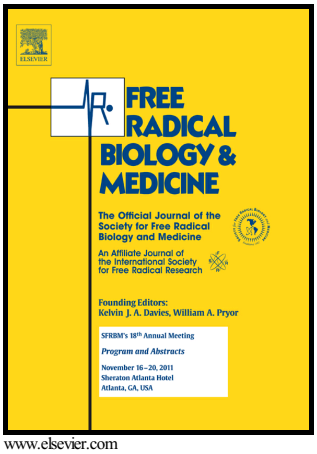
Procyanidin B2 ameliorates free fatty acids-induced hepatic steatosis through regulating TFEB-mediated lysosomal pathway and redox state

Hongming Su, Yuting Li, Dongwen Hu, Lianghua Xie, Huihui Ke, Xiaodong Zheng, Wei Chen



www.elsevier.com

PII: S0891-5849(18)31439-4
DOI: https://doi.org/10.1016/j.freeradbiomed.2018.08.024
Reference: FRB13888

To appear in: *Free Radical Biology and Medicine*

Received date: 29 April 2018
Revised date: 19 August 2018
Accepted date: 20 August 2018

Cite this article as: Hongming Su, Yuting Li, Dongwen Hu, Lianghua Xie, Huihui Ke, Xiaodong Zheng and Wei Chen, Procyanidin B2 ameliorates free fatty acids-induced hepatic steatosis through regulating TFEB-mediated lysosomal pathway and redox state, *Free Radical Biology and Medicine,* https://doi.org/10.1016/j.freeradbiomed.2018.08.024

Procyanidin B2 ameliorates free fatty acids-induced hepatic steatosis through regulating TFEB-mediated lysosomal pathway and redox state

Hongming Su, Yuting Li, Dongwen Hu, Lianghua Xie, Huihui Ke, Xiaodong Zheng, Wei Chen*

Department of Food Science and Nutrition, National Engineering Laboratory of Intelligent Food Technology and Equipment, Zhejiang Key Laboratory for Agro-Food Processing, Zhejiang University, Hangzhou 310058, China.

*Corresponding author. Wei Chen, Ph.D. Department of Food Science and Nutrition, Zhejiang University, No.866 Yuhangtang Road, Xihu District, Hangzhou 310058, China. Tel.: +86 571 88982861; fax: +86 571 88982191. zjuchenwei@zju.edu.cn

Abstract

Procyanidin B2, a naturally occurring phenolic compound, has been reported to exert multiple beneficial functions. However, the effect of procyanidin B2 on free fatty acids (FFAs)-induced hepatic steatosis remains obscure. The present study is therefore aimed to elucidate the protective effect of procyanidin B2 against hepatic steatosis and its underlying mechanism. Herein, we reported that procyanidin B2 attenuated FFAs-induced lipid accumulation and its associated oxidative stress by scavenging excessive ROS and superoxide anion radicals, blocking loss of mitochondrial