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Cyanidin-3-O-glucoside ameliorates lipid and glucose accumulation in C57BL/6J mice via activation of PPAR-α and AMPK?

Yaoyao Jia, Ji Hae Lee, Chunyan Wu, Yeonji Kim and **Sung-Joon Lee**
Korea University, Republic of Korea

Cyanidin-3-O-glucoside (C3G) is an anthocyanidin abundant in fruits and vegetables that exhibits potent activities in energy metabolism; however, molecular target(s) and the mode of actions of C3G have been elusive. Surface plasmon resonance and time-resolved fluorescence resonance energy transfer analyses revealed that C3G directly interacted with PPARα ligand-binding domain, thus the C3G induced hepatic fatty acid oxidation and ketogenesis regulating PPARα responsive genes. C3G also bound directly to the α1 subunit of AMPK in cell-free kinase assay, phosphorylated ACC1, decreased hepatic malonyl-CoA concentration, thus reduced hepatic fatty acid synthesis. In high-fat-diet fed C57BL/6J mice orally administrated with C3G for 8 weeks, hepatic and plasma triglycerides were significantly reduced due to the combined effects of PPARα and AMPK activation. Insulin resistance and glucose tolerance were significantly improved with reduction in hepatic gluconeogenesis in C3G mice compared with those of controls. The AMPK activation by C3G led to the phosphorylation inhibition of FoxO1 and CREB to suppress the expressions of key genes in hepatic gluconeogenesis. Body fat accumulation was reduced in C3G fed mice with induction of thermogenic gene expressions in brown adipocytes and energy expenditure was significantly increased in mice fed C3G. Hepatic autophagy pathway was significantly activated in C3G livers, via activation AMPK-mTOR signaling axis and PPARα activation. Autophagy activation may contribute to lowering lipid accumulation in the liver. Collectively, these demonstrate that C3G in diet has profound metabolic roles in regulating lipid metabolism and insulin resistance via activation of both PPARα and AMPK.

Biography

Yaoyao Jia earned her Degree of Doctor of Philosophy from Food Science and Technology Department of Biotechnology Graduate School of Korea University. Her research is mainly focus on effects of natural compounds in cellular lipid metabolism and regulation of PPAR activity.

janciey227@naver.com