Wan-Rou Shih, Metabolomics 2016, 6:2(Suppl)
http://dx.doi.org/10.4172/2153-0769.C1.032

Fenofibrate regulates cell energy metabolism by restricting hypoxia-induced factor expression in human glioma cells

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Fenofibrate is a lipid-lowering agent that has been shown to suppress tumor progression in many cancers. In the present study, we demonstrated that treatment of human U87 glioma cells with fenofibrate decreased the protein levels of HIF-1α under normoxia and hypoxia conditions. The suppression of HIF-1α was reversed by pretreatment with proteosome inhibitor. The reduction of HIF-1α level was associated with decreased expression levels of glucose transporter Glut-1, and lactate dehydrogenase A. Treatment with fenofibrate also decreased the expression of pyruvate dehydrogenase kinase-1 and pyruvate dehydrogenase phosphorylation which subsequently reactivated mitochondrial oxidative phosphorylation and increased mitochondrial ATP production in U87 cells. Treatment of cells with fenofibrate decreased cell viability, which can be prevented by pretreatment with l-N-aceytylcystein, an anti-oxidant. These data suggest that reactivation of mitochondrial energy metabolism by ferrous glycinate may increase ROS accumulation, and the excessive ROS cause mitochondrial damage and subsequently results in cell death.

Biography

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