Jhong-Huei Jheng, Metabolomics 2016, 6:2(Suppl)
http://dx.doi.org/10.4172/2153-0769.C1.032

Ferrous glycinate regulates cell energy metabolism via suppression of hypoxia-induced factor in human A549 cells

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HIF-1αis a transcription factor and play a crucial role in cancer metabolism, which are characterized with aerobic glycolysis and suppressing of mitochondrial energy metabolism (Warburg effect). In the present study, we demonstrated that incubation of A549 cells with ferrous glycinate decreased the protein levels of HIF-1α under either nomoxia or hypoxia conditions, which was further reversed by pretreatment with proteosome inhibitor, or prolyl hydroxylase inhibitor. The reduction of HIF-1α level was associated with decreased expression levels of glucose transporter, Glut-1, and glycolytic enzymes including hexokinase-2, and lactate dehydrogenase A. On the other hand, treatment with ferrous glycinate reactivated oxidative phosphorylation by decreasing the expression of pyruvate dehydrogenase kinase-1 and pyruvate dehydrogenase phosphorylation that subsequently increased mitochondrial membrane potential and ATP production, suggesting ferrous glycinate regulate energy metabolism in lung adenocarcinoma cells. These results indicate that ferrous glycinate may serve as a therapeutic adjuvant for related human lung cancers.

Biography

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