Sandile Fuku, Metabolomics 2016, 6:3(Suppl)
http://dx.doi.org/10.4172/2153-0769.C1.034

Calmodulin dependent protein kinase (CaMK) II activation by exercise regulates lipid metabolism in rat skeletal muscle

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Background: Activation of calmodulin dependent protein kinase (CaMK) II by exercise has plethora of benefits in metabolism and health. Regulation of lipid metabolism is very significant to alleviate type 2 diabetes and obesity. The role of CaMKII in the regulation of genes that are involved in lipid metabolism has not been studied yet, which became the focus of this study.

Methods: 5 to 6 weeks old male Wistar rats were used in this study. Western blot was performed to assess the protein expression of carnitine palmitoyltransferase *(CPT)-1* and acetyl-CoA carboxylase *(ACC)-1*. Cpt-1 and Acc-1 gene expressions were assessed using quantitative real time PCR (qPCR).

Results: The results indicate that exercise-induced CaMKII activation increases CPT-1 expression and decreases ACC-1 expression in rat skeletal muscle. Thus, confirming CaMKII activation by exercise and the resultant increase in lipid oxidation. Administration of KN93 (CaMKII inhibitor) reversed all exercise-induced changes.

Conclusions: This study demonstrated that CaMKII activation, by exercise, regulates lipid metabolism genes in rat skeletal muscle. Further, the increase in lipid oxidation and decrease in lipid synthesis are evidence of the regulatory role CaMKII in lipid metabolism. CaMKII is a potential target in designing novel therapeutic drugs in the management and treatment of type 2 diabetes and obesity.

Biography

Sandile Fuku has completed his Doctoral studies in Biomedical Technology from the Central University of Technology and is currently a Post-doctoral fellow in the Department of Biochemistry at North-West University. Currently, his research is on epigenetic regulation in metabolic syndromes, particularly focusing on Diabetics and Cancer. He has published work in Cancer Treatment, Diabetes and Phytochemistry.

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