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 bene ts in metabolism and health. Regulation of lipid metabolism is very signi cant to alleviate type-2 diabetes and obesity. e 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: 5-6 weeks old male Wistar rats were used in this study. Western blot was performed to assess the protein expression o Carnitine palmitoyltransferase (CPT)-1 and Acetyl-CoA carboxylase (ACC)-1. Cpt-1 and Acc-1 gene expressions were assesse using Quantitative real time PCR (qPCR).

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

Conclusions: is 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.