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## Exercise-induced calmodulin dependent protein kinase (CaMK) II activation regulates saturated and unsaturated fatty acids in rat skeletal muscle

**Sandile Fuku**  
North West University, South Africa

**Introduction:** CaMKII regulates many pathways involved in the regulation of various cellular and molecular mechanisms that result in myriad health benefits. Exercise is a key activator of CaMKII, and shown to improve many functional activities in individuals who exercise compared with those who do not exercise, however the mechanism involved not yet fully elucidated, which became the objective of this study.

**Methods:** In this study using rats, we investigated various lipids metabolism for both saturated and non-saturated fatty acids in rats that exercised, non-exercised and exercised+KN93 (CaMKII inhibitor). Lipids were analyzed using GC $\times$ GC TOFMS. Palmitoleic acid and oleic acid which are mono unsaturated fatty acids known to promote insulin sensitivity and improve glycaemic control were investigated. Levels of the exercise group showed ~2.0 fold increase compared with the non-exercise (control) group. Abolishing CaMKII activity by administration of KN93 significantly decreased exercise-induced palmitoleic acid levels. Oleic acid levels of the exercise group were ~4.1 folds higher than the non-exercise group and followed the same pattern as palmitoleic acid.

**Results:** Lauric acid is a saturated fatty acid, which increases fatty acid needed for better health. The exercise group showed ~ 8.7 fold increase compared with the non-exercise group of lauric acid. The exercise +KN93 group significantly reduced induction by ~2.5 fold compared with the exercise group. On the other hand, myristic acid and palmitic acid which are saturated fatty acids known to increase risk factors of metabolic syndrome. The myristic acid level of the exercise group decreased by ~3.4 fold compared with the control group, whereas the exercise +KN93 group significantly increased by ~4.3 compared with the exercise group.

**Conclusion:** CaMKII can reduce the risk factors of metabolic syndrome and type 2 diabetes.

### Biography

Sandile Fuku 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 and phytochemistry.

[fukusl@yahoo.com](mailto:fukusl@yahoo.com)

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