



The Contributions of Skeletal Muscle PKC Theta to Diet-Induced Obesity

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Abstract

Protein Kinase C- Theta (PKC θ) is a gene predominantly expressed in hematopoietic cells and skeletal muscle. In skeletal muscle, PKC θ regulates fat metabolism and insulin sensitivity. PKC θ activity increases in response to high levels of diacylglycerol in the cell, a common outcome of chronic high fat diet consumption and obesity. PKC θ is associated with skeletal muscle metabolic dysfunction, which may exacerbate weight gain and metabolic disease. The purpose of this study was to test the hypothesis that the selective deletion of PKC θ from skeletal muscle protects against diet-induced obesity.

Mice lacking PKC θ in skeletal muscle were created using Cre-Lox recombination. At weaning, control (PKC $\theta^{\text{SkM}+/+}$) and knockout (PKC $\theta^{\text{SkM}-/-}$) mice were randomly assigned to regular or high fat diet (RD or HFD, respectively) groups. Mouse weights were taken weekly for 15 weeks.

During the 15-week diet intervention, male PKC $\theta^{\text{SkM}+/+}$ mice on a HFD became obese. Male PKC $\theta^{\text{SkM}-/-}$ mice consuming a HFD showed attenuated weight gain, which was similar to mice on a RD. This trend was not present for female mice, in which weight changed to a similar magnitude independent of diet and genotype. In conclusion, PKC- θ in the skeletal muscle may contribute to the regulation of diet-induced obesity. It is unclear whether these affects are sex specific.

Keywords

Protein Kinase C Theta; Obesity; High fat diet



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ABSTRACT

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