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Research Article

# Metabolic Roles of the M<sub>3</sub> Muscarinic Acetylcholine Receptor Studied with M<sub>3</sub> Receptor Mutant Mice: A Review

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### **Abstract**

The M<sub>3</sub> muscarinic acetylcholine (ACh) receptor (M<sub>3</sub> mAChR) is expressed in many central and peripheral tissues. It is a prototypic member of the superfamily of G protein-coupled receptors and preferentially activates G proteins of the  $G_{\alpha}$  family.

Recent studies involving the use of newly generated mAChR mutant mice have

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induced obesity and obesity-associated metabolic deficits. Under all experimental conditions tested, M<sub>3</sub> receptor-deficient mice showed greatly ameliorated impairments in glucose homeostasis and insulin sensitivity, reduced food intake, and a significant elevation in basal and total energy expenditure, most likely due to increased central sympathetic outflow and increased rate of fatty acid oxidation. These findings are of potential interest for the development of novel therapeutic approaches for the treatment of obesity and associated metabolic disorders.

Q Key Words: : Glucose homeostasis Insulin Knockout mice Muscarinic receptor Transgenic mice

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