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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<sub>q</sub> family. Recent studies involving the use of newly generated mAChR mutant mice have revealed that the M<sub>3</sub> mAChR plays a key role in regulating many important metabolic functions. Phenotypic analyses of mutant mice that either selectively lacked or overexpressed M<sub>3</sub> receptors in pancreatic  $\beta$  -cells indicated that  $\beta$  -cell M<sub>3</sub> mAChRs are essential for maintaining proper insulin release and glucose homeostasis. The experimental data also suggested that strategies aimed at enhancing signaling through

$\beta$ -cell  $M_3$  mAChRs might be beneficial for the treatment of type 2 diabetes. Recent studies with whole body  $M_3$  mAChR knockout mice showed that the absence of  $M_3$  receptors protected mice against various forms of experimentally or genetically induced obesity and obesity-associated metabolic deficits. Under all experimental conditions tested,  $M_3$  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.

Key Words: :

Glucose homeostasis

Insulin

Knockout mice

Muscarinic receptor

Transgenic mice

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