Adipose Tissue-specific PTEN Knockout Mice

Tech ID: 20189 / UC Case 2004-644-0

BACKGROUND

Aberrant glucose uptake due to insulin resistance is a key pathogenic feature of type 2 diabetes. Insulin signals through its cell surface receptor (IR). The binding of insulin to IR leads to the activation of the PI3-kinase pathway. A phosphatase and tensin homolog deleted from chromosome 10 (PTEN) is a negative regulator of the PI3-kinase/AKT pathway and is hypothesized to inhibit the metabolic effects of insulin. Understanding insulin signaling will lead to new targets for interventions aimed at reversing insulin resistance.

INNOVATION

UCLA researchers have developed a mouse model to explore the mechanisms by which insulin signaling in adipose tissue regulates systemic insulin sensitivity. A targeted deletion of PTEN in adipose tissue was accomplished by utilizing the Cre-lox system under control of the aP2 adipose specific promoter. Loss of PTEN results in improved systemic glucose tolerance and insulin sensitivity. In addition, mutant animals exhibit increased insulin signaling and AMP kinase activity in the liver.

RELATED MATERIALS

- Insulin hypersensitivity and resistance to streptozotocin-induced diabetes in mice lacking PTEN in adipose tissue. *Mol Cell Biol.* (2005)

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OTHER INFORMATION

KEYWORDS

research tools mouse model, PTEN, oncology, diabetes, adipose tissue, conditional

CATEGORIZED AS

- **Research Tools**
  - Animal Models

RELATED CASES

2004-644-0

ADDITIONAL TECHNOLOGIES BY THESE INVENTORS

- PTEN Null Cell Lines
- Genetically Engineered Mice Lacking a Tumor Suppressor Gene in the CNS
- Murine PTEN Null Prostate Cancer Model
- A Method for In Vivo Visualization of Mutated Mouse Cells

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