

A body temperature sensor, TRPM2, promotes insulin secretion

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The research group led by professor Makoto Tominaga and Dr. Kunitoshi Uchida, National institute for Physiological Sciences (NIPS), Japan, found TRPM2 ion channel in pancreatic beta-cells is important for insulin secretion stimulated by glucose and gastrointestinal hormone (incretin) secreted after food intake. Their finding was reported in *Diabetes*.

Diabetes mellitus is a disease caused by lack of insulin secretion from pancreatic cells, or less response to the secreted insulin, which raises the blood glucose levels, and as a result, causes serious disorders. It is said that at least 171 million people worldwide suffer from [diabetes mellitus](#), and its incidence is increasing rapidly. Clarify the mechanisms of insulin secretion is important for the development of [diabetes](#) therapy. Here, this research group focused on TRPM2 acting as a body temperature sensor.

TRPM2 is a temperature-sensitive Ca²⁺-permeable channel and expressed in pancreatic beta-cells. They found that TRPM2-deficient mice have shown the higher blood glucose levels with impaired insulin secretion compared with wild-type mice. Furthermore, TRPM2-deficient pancreatic beta-cells showed smaller intracellular Ca²⁺ increase and lesser insulin secretion stimulated by glucose and incretin.

Professor Makoto Tominaga and Dr. Kunitoshi Uchida said, "TRPM2 may control [insulin secretion](#) levels mainly by modulating intracellular

Ca²⁺ concentrations. Finding the substance which stimulates TRPM2 effectively could lead to the development of a new therapy for diabetes mellitus."

Provided by National Institute for Physiological Sciences

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