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."

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