

New mechanism regulating insulin secretion may explain genetic susceptibility to diabetes

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New Zealand research revealing a new mechanism for how glucose stimulates insulin secretion may provide a new explanation for how a gene that makes people more susceptible to diabetes – called TCF7L2 – actually contributes to the disease.

"It has long been known that insulin is secreted from beta-cells in the pancreas, in response to rising [blood glucose levels](#), and that the insulin in turn controls glucose levels," explains team leader Professor Peter Shepherd from the Maurice Wilkins Centre for Molecular Biodiscovery and The University of Auckland. "However the mechanisms controlling insulin secretion have not been fully understood."

The latest research in Professor Shepherd's laboratory has revealed the missing link in a series of [chemical signals](#) by which glucose stimulates insulin secretion from beta-cells. The scientists found that a signalling molecule called cyclic-AMP acts to stabilise beta-catenin, a protein they show has an important role in regulating beta-cell function, including the release of insulin in response to glucose.

"This is important as Type-2 diabetes is increasing to [epidemic proportions](#) worldwide. It is caused by defective [insulin release](#) from beta-cells, and the resulting failure to control blood glucose levels. In order to understand the disease it's important to learn about the mechanism that control insulin secretion," says lead researcher Dr Emmanuelle Cognard, also from The University of Auckland.

This newly discovered signalling pathway may explain how one of the major diabetes susceptibility genes, called TCF7L2, can impair insulin secretion, as TCF7L2 redirects beta-catenin away from the cell surface and so would reduce the effect of beta-catenin on [insulin secretion](#). The research is likely to influence the way new drugs to treat Type-2 diabetes are designed.

The research, which was funded by the Health Research Council of New Zealand, has been published in the February 2013 issue of the *Biochemical Journal*.

Provided by University of Auckland

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