

Novel mechanism of insulin resistance in type 2 diabetes

September 17 2015



Credit: Darren Lewis/public domain

Insensitivity to insulin, also called insulin resistance, is associated with type 2 diabetes and affects several cell types and organs in the body. Now, scientists from Sweden's Karolinska Institutet have discovered a mechanism that explains how insulin-producing cells can be insulin resistant and insulin sensitive at the same time.

The findings are being published in the journal *Cell Reports*, and may lead to future novel [treatment strategies](#) for type 2 diabetes.

Insulin is critical in lowering blood glucose concentration. Individuals with type 2 diabetes suffer from [insulin resistance](#) and this means that their cells/organs are insensitive to insulin. In [type 2 diabetes](#) the body tries to compensate by producing more insulin, and also by increasing the number of [insulin-producing cells](#). Finding new treatment strategies is only possible by gaining a greater understanding of what happens in the body of a diabetic patient. One scientific challenge is to explain how a cell/organ at the same time can be insulin resistant in one biological function and insulin sensitive in another.

Drs Barbara Leibiger and Ingo Leibiger, both members of Professor Per-Olof Berggren's research group at the Department of Molecular Medicine and Surgery, Karolinska Institutet, are particularly interested in the insulin-producing beta cells.

"The beta cell must have insulin to work properly", says Barbara Leibiger, PhD, Associate Professor, and lead author of the current study. "In a person with diabetes, the beta cells become insensitive to insulin."

The researchers have previously shown that the beta cell has two receptors with different biological functions, insulin receptor A and insulin receptor B. In the current study, they found that under diabetic conditions, even though [insulin receptor B](#) is insulin insensitive for one signalling pathway, insulin can under these conditions instead activate a different signalling pathway, leading to beta cell proliferation. The researchers also identified the factor, PI3K-C2 α , that caused the switch from one signalling pathway to another.

"The results are important since it explains how the beta cell can go from a differentiated state to a proliferative state", says Ingo Leibiger, PhD, Associate Professor, who co-supervised the current study with Professor Berggren. "This means that the cells change from being glucose-responsive to instead increase in number."

According to the study authors, also including researchers from the Pohang University of Science and Technology, Republic of Korea, factors involved in the re-routing of the [insulin](#) signal represent tentative therapeutic targets in the treatment of [diabetes](#).

More information: 'PI3K-C2 α Knockdown Results in Rerouting of Insulin Signaling and Pancreatic Beta Cell Proliferation', Barbara Leibiger, Tilo Moede, Meike Paschen, Na-Oh Yunn, Jong Hoon Lim, Sung Ho Ryu, Teresa Pereira, Per-Olof Berggren, and Ingo B. Leibiger, *Cell Reports*, October 06, 2015 paper issue, online first September 17, 2015.

Provided by Karolinska Institutet

Citation: Novel mechanism of insulin resistance in type 2 diabetes (2015, September 17)
retrieved 24 April 2024 from
<https://medicalxpress.com/news/2015-09-mechanism-insulin-resistance-diabetes.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.