

## New insight into the biology of insulin release

July 18 2019, by Ziba Kashef

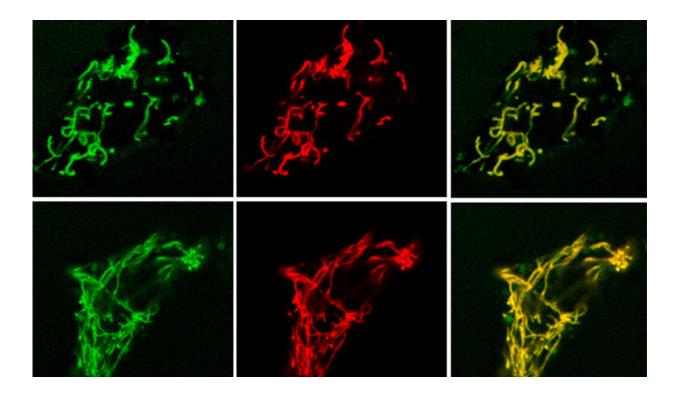


Image shows the role of GTP-limiting or -producing enzymes (green) in beta-cell mitochondria (red), leading to altered mitochondria biology (yellow), which affects insulin. Credit: Yale University

In a new study, Yale researchers challenge a long-held assumption about how insulin-producing cells in the pancreas sense and respond to glucose. Their findings could lead to changes in the way that scientists approach the treatment of diabetes, the authors said.



To evaluate exactly how beta cells, which secrete insulin, detect a rise in glucose, associate professor of medicine and corresponding author Dr. Richard Kibbey and his collaborators studied a rare form of hypoglycemia as a model. They conducted experiments in <u>cell cultures</u> and in mice to examine different mechanisms for how metabolism in beta cells might affect insulin production and glucose levels.

The researchers found that a <u>chemical compound</u> in beta cells called mitochondrial GTP, or mtGTP, is a key factor that allows the cells to secrete insulin. Specifically, when mtGTP is produced from a particular enzyme, it serves as a signal to stimulate the release of insulin. The research team also revealed that mtGTP has other effects that promote the health of beta cells more broadly.

This discovery shows that the established understanding of how <u>beta</u> <u>cells</u> function and respond to glucose is not the whole story, said the Yale scientists. The study finding illuminates a novel <u>metabolic pathway</u> that researchers can plan to target with drug therapy to improve beta cell health, insulin production, and the balance of blood sugar, they noted.

The study is published in *Cell Reports*.

**More information:** Sean R. Jesinkey et al. Mitochondrial GTP Links Nutrient Sensing to β Cell Health, Mitochondrial Morphology, and Insulin Secretion Independent of OxPhos, *Cell Reports* (2019). DOI: 10.1016/j.celrep.2019.06.058

## Provided by Yale University

Citation: New insight into the biology of insulin release (2019, July 18) retrieved 9 April 2024 from <a href="https://medicalxpress.com/news/2019-07-insight-biology-insulin.html">https://medicalxpress.com/news/2019-07-insight-biology-insulin.html</a>



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