Cellular calcium handling in diabetes

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Tight regulation of calcium levels in the endoplasmic reticulum (ER) – a cellular organelle with multiple functions – contributes to insulin secretion by pancreatic beta cells. Although ER calcium handling is perturbed in diabetes, the molecular determinants of ER calcium balance are not clear.

David Jacobson, Ph.D., and colleagues have now demonstrated that TALK-1 potassium channels located in the ER membrane facilitate calcium release from the ER in mouse and human beta cells.

Mice lacking TALK-1 had reduced cytosolic and increased ER calcium concentrations. When fed a high-fat diet, these mice had reduced signs of ER stress, which contributes to beta cell loss in diabetes, compared to normal mice.

The findings, reported Sept. 19 in *Science Signaling*, suggest that defects in TALK-1 channel activity can perturb ER function and contribute to islet dysfunction in diabetes. TALK-1 and similar ER-localized potassium channels may offer new therapeutic targets to reduce calcium handling defects and ER stress during the pathogenesis of diabetes.

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More information: TALK-1 channels control β cell endoplasmic reticulum Ca2+ homeostasis. *Science Signaling*. doi.org/10.1126/scisignal.aan2883

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