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Study characterizes insulin secretion in response to metabolic stress

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The development of type 2 diabetes is linked to persistent inflammation as a consequence of metabolic stress. Prolonged exposure to the proinflammatory molecule IL-1β is associated with reduced insulin secretion by pancreatic β cells, while short-term exposure to IL-1β has been shown to increase insulin levels. Reducing IL-1 signaling in patients with type 2 diabetes has had mixed success in clinical studies, suggesting multiple effects of IL-1β in insulin secretion.

A new study in *JCI Insight* reveals that IL-1 signaling is an important mediator of islet compensation to metabolic stress. Patrick MacDonald and colleagues at the University of Alberta determined that IL-1β amplifies insulin secretion in healthy human islets. Islets from obese individuals were particularly sensitive to IL-1β stimulation; however, islets from obese subjects with type 2 diabetes were not responsive to IL-1β. In mice, inhibition of IL-1 signaling resulted in symptoms of type 2 diabetes, including glucose intolerance and impaired insulin secretion in response to metabolic stress. Additionally, the authors determined that IL-1β directly promotes insulin secretion by enhancing release of insulin-containing granules.

The results of this study demonstrate that IL-1R signaling is important for glucose homeostasis.

More information: Catherine Hajmrle et al. Interleukin-1 signaling contributes to acute islet compensation, *JCI Insight* (2016). DOI: 10.1172/jci.insight.86055