**Diabetes and pancreatic islet crosstalk**

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**Abstract**

Pancreatic beta cells are the only cell type in our body capable of producing and secreting insulin to instruct the insulin-sensitive cells and tissues of our bodies to absorb nutrients after a meal. Accurate control of insulin release is of critical importance; too little insulin leads to diabetes, while an excess of insulin can cause potentially fatal hypoglycaemia. Yet, the pancreas of most people will control insulin secretion safely and effectively over decades and in response to glucose excursions driven by tens of thousands of meals. Because we only become aware of the important contributions of the pancreas when it fails to maintain glucose homeostasis, it is easy to forget just how well insulin release from a healthy pancreas is matched to insulin need to ensure stable blood glucose levels. Beta cells achieve this feat by extensive crosstalk with the rest of the endocrine cell types in the islet, notably the glucagon-producing alpha cells and somatostatin-producing delta cells. Here I will review the important paracrine contributions that each of these cells makes to the stimulation and subsequent inhibition of insulin release in response to a transient nutrient stimulation, and make the case that a breakdown of this local crosstalk contributes to the pathophysiology of diabetes.