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# Beta Cells in Pancreas

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## Editorial

Beta cells (β-cells) are a type of cell found in pancreatic islets that synthesize and secrete insulin and amylin. Beta cells make up 50-70% of the cells in human islets. In patients with type 1 diabetes, beta-cell mass and function are diminished, leading to insufficient insulin secretion and hyperglycemia. The primary function of a beta cell is to produce and release insulin and amylin. Both are hormones which reduce blood glucose levels by different mechanisms [1]. Beta cells can respond quickly to spikes in blood glucose concentrations by secreting some of their stored insulin and amylin while simultaneously producing more. Beta cells are the only site of insulin synthesis in mammals. As glucose stimulates insulin secretion, it simultaneously increases proinsulin biosynthesis, mainly through translational control. The insulin gene is first transcribed into mRNA and translated into preproinsulin. After translation, the preproinsulin precursor contains an N-terminal signal peptide that allows translocation into the rough endoplasmic reticulum. Inside the RER, the signal peptide is cleaved to form proinsulin. Then, folding of proinsulin occurs forming three disulfide bonds. Subsequent to protein folding, proinsulin is transported to the Golgi apparatus and enters immature insulin granules where proinsulin is cleaved to form insulin and C-peptide. After maturation, these secretory vesicles hold insulin, C-peptide, and amylin until calcium triggers exocytosis of the granule contents. In beta cells, insulin release is stimulated primarily by glucose present in the blood. As circulating glucose levels rise such as after ingesting a meal, insulin is secreted in a dose-dependent fashion. This system of release is commonly referred to as glucose-stimulated insulin secretion [2]. There are four key pieces to the "Consensus Model" of GSIS: GLUT2 dependent glucose uptake, glucose metabolism, KATP channel closure, and the opening of voltage gated calcium channels causing insulin granule fusion and exocytosis. Voltage-gated calcium channels and ATP-sensitive potassium ion channels are embedded in the plasma membrane of beta cells. These ATP-sensitive potassium ion channels are normally open and the calcium ion channels are normally closed. Potassium ions diffuse out of the cell, down their concentration gradient, making the inside of the cell more negative with respect to the outside (as potassium ions carry a positive charge) [3]. At rest, this creates a potential difference across the cell surface membrane of -70mV. When the glucose concentration outside the cell is high, glucose molecules move into the cell by facilitated diffusion, down its concentration gradient through the GLUT2 transporter. Since beta cells use glucokinase to catalyze the first step of glycolysis, metabolism only occurs around physiological blood glucose levels and above [4]. Metabolism of the glucose produces ATP, which increases the ATP to ADP ratio. The ATP-sensitive potassium ion channels close when this ratio rises. This means that potassium ions can no longer diffuse out of the cell. As a result, the potential difference across the membrane becomes more positive (as potassium ions accumulate inside the cell) [5]. This change in potential difference opens the voltage-gated calcium channels, which allows calcium ions from outside the cell to diffuse in down their concentration gradient. When the calcium ions enter the cell, they cause vesicles containing insulin to move to, and fuse with, the cell surface membrane, releasing insulin by exocytosis into the hepatic portal vein.

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