Abstract

The regulation of pancreatic hormone secretion, specifically of insulin and glucagon, is a fundamental mechanism by which the body maintains a stable supply of energy substrates to our cells. Glucose acts as the primary trigger for insulin release, though other nutrients also influence this endocrine response. Oxidation of glucose leads to the production of ATP, which ensures depolarization of the cytoplasmic membrane, rise in calcium ion concentration in the cytosol, and subsequent exocytosis of insulin secretory vesicles from beta cells into the systemic circulation. Recent findings highlight the significance of redox signaling in this process, particularly the role of H_2O_2 as an essential factor for insulin secretion, which is produced by the activity of the enzyme NADPH oxidase 4. Insulin then influences target cells such as myocytes, hepatocytes, and adipocytes to promote the synthesis of energy-rich molecules. Dysregulation of this process can lead to the onset of diabetes mellitus. The incretin glucagon-like peptide 1 is secreted from enteroendocrine L-cells after food intake. Its direct effect on beta cells, along with other mechanisms, effectively reduces postprandial hyperglycemia, making its synthetic analogs an important therapeutic tool for diabetic patients. Moreover, these agents facilitate weight loss in overweight individuals and are therefore also used in the management of obesity.

Keywords

GLP-1, glucose-stimulated insulin secretion, pancreatic beta cells, redox signaling, type 2 diabetes