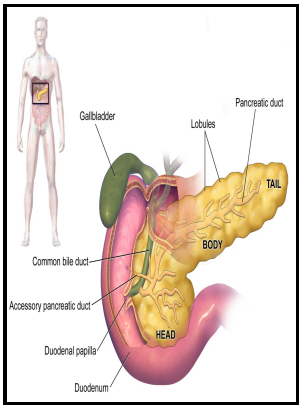
Control of the endocrine pancreas by gastrointestinal peptides.

Aston University. Department of Pharmaceutical Sciences - Physiology, Gastrointestinal Hormonal Control Article



Description: -
-Control of the endocrine pancreas by gastrointestinal peptides.
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14.9 The Endocrine Pancreas

These two hormones regulate the rate of glucose metabolism in the body. Abbreviations: ACh, acetylcholine released from cholinergic enteric neurons; CaSR, calcium-sensing receptor; CCK2, cholecystokinin type 2 receptors; D, D cell; ENS, enteric nervous system; ECL, enterochromaffin-like cell; G, G cell; GRP, gastrin-releasing peptide released from enteric peptidergic neurons; GRPR, GRP receptor; H2, histamine type 2 receptor; M, mucous cell; P, parietal cell; SST, somatostatin; M3, muscarinic type 3 receptor; SST2, and SST type 2 receptor. Glucagon derives from a 180-amino acid precursor peptide called proglucagon, which undergoes tissue-specific post-translation processing to produce different peptides in different cell types.

Endocrine Control

Precisely how insulin facilitates glucose uptake is not entirely clear. Early pancreatic development requires the vertebrate suppressor of hairless RBPJ in the PTF1 bHLH complex. Not much is known about this peptide, except for this essential function.

Hormonal control of avian pancreas by gastrin

In contrast to acinar cells, which remain at the distal tips, endocrine and duct progenitors segregate from their acinar counterparts to localize to epithelial cords. Pancreatic beta cell dedifferentiation as a mechanism of diabetic beta cell failure. Gastrin releasing peptide GRP was also studied.

Control of Cell Identity in Pancreas Development and Regeneration

The secretion of insulin is regulated through a negative feedback mechanism. Histamine then binds H2 receptors on nearby parietal cells, which further stimulates secretion of hydrogen ions. Ghrelin treatments for obesity are still under intense scrutiny and no conclusive evidence has been reached.

Endocrine Control

Finally, insulin promotes triglyceride and protein synthesis. Since H2 blockers and PPIs inhibit secretion of hydrogen ions by parietal cells in the stomach, patients who chronically take these medications will experience secondary hypergastrinemia in response to lack of negative feedback from gastric acid production. Novel small-molecule nonpeptide GLP-1 agonists are becoming available.

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Insulin triggers the rapid movement of a pool of glucose transporter vesicles to the cell membrane, where they fuse and expose the glucose transporters to the extracellular fluid. Kopp JL, Dubois CL, Schaffer AE, et al. Cells in both regions express the Pdx1 transcription factor whereas cells located in the ventral foregut patch also express the Sox17 transcription factor.

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