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MEDICINE AND PHARMACY

Pancreatic hormones

Mohandass Keerthickdass[1], Orozbek uulu Tursunbek[2]

[1] 1st year student;
International Medical Faculty of Osh State University; Republic of Kyrgyzstan

[2] Professor, Department of physiology;
International Medical Faculty of Osh State University; Republic of Kyrgyzstan

Abstract. Pancreatic hormones play a crucial role in maintaining blood glucose levels, regulating metabolism, and ensuring overall energy balance in the body. The pancreas, through its endocrine function, secretes hormones that are essential for glucose homeostasis. Among the most important of these hormones are insulin and glucagon, which work in coordination to maintain stable blood sugar levels. Disorders in this hormonal balance, especially insulin deficiency or resistance, lead to diabetes mellitus — a major global health concern affecting millions of people worldwide. This article highlights the secretion, role, and regulation of pancreatic hormones.

Keywords: *Pancreatic hormones, insulin, glucagon, somatostatin, diabetes mellitus, islets of Langerhans, endocrine pancreas.*

Introduction:

The pancreas is a unique organ that functions as both an exocrine and endocrine gland. Its exocrine part secretes digestive enzymes into the duodenum, aiding in the breakdown of carbohydrates, proteins, and fats. Meanwhile, the endocrine portion, made up of the islets of Langerhans, is responsible for producing vital hormones such as insulin, glucagon, somatostatin, and pancreatic polypeptide.

Each type of cell within the islets plays a specific role:

Beta cells secrete insulin, Alpha cells produce glucagon, Delta cells release somatostatin and PP cells produce pancreatic polypeptide.

These hormones together maintain glucose balance and metabolic stability. Understanding the physiology of pancreatic hormones is essential for comprehending the pathophysiology of diabetes mellitus — one of the most widespread and burdensome endocrine disorders globally. Better knowledge of their secretion and regulation is critical for the prevention and management of such conditions.

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Materials and Methods:

This study was conducted through both theoretical and practical approaches. Reference was taken from standard human physiology textbooks for a detailed understanding of pancreatic hormone functions. The anatomy lab session involved direct observation and identification of the pancreas, focusing on its anatomical structure and position in the abdominal cavity. In the histology lab, prepared pancreatic tissue slides were examined using a compound microscope. Special attention was given to the Islets of Langerhans, which appeared as lighter-stained clusters compared to the darker surrounding exocrine tissue. Within these islets, various cell types such as alpha (α), beta (β), delta (δ), and PP cells were identified and studied based on their position and staining characteristics.

Data Recording and Analysis

Observational data was manually recorded during the lab sessions. In the anatomy lab, the pancreas was identified as a retroperitoneal organ with both exocrine and endocrine functions. In histological analysis, the size, number, and distribution of islets were noted. Each islet was found to contain distinct hormone-secreting cells.

Beta cells were observed predominantly in the central part of the islet, assumed responsible for insulin secretion. Alpha cells were mostly found peripherally, related to glucagon secretion. Delta and PP cells were less numerous but present and structurally distinguishable.

This microscopic examination helped correlate cell types with their specific hormonal outputs. Findings were cross-checked with theoretical information for accuracy. Data was not statistically analyzed as this was a qualitative observational study based on lab sessions.

Results and discussion:

i) Anatomy of Pancreas:

The pancreas is a retroperitoneal organ located posterior to the stomach. The head of the pancreas is nestled within the curvature of the duodenum, while the pancreas extends transversely with its tail lying near the spleen. Structurally, the pancreas is divided into lobules, each composed of exocrine acini that secrete digestive enzymes.

The endocrine part of the pancreas consists of the islets of Langerhans, which contain different types of hormone-secreting cells:

β (beta) cells secrete insulin, making up about 70% of

the islet cells. α (alpha) cells secrete glucagon, accounting for approximately 20%. δ (delta) cells secrete somatostatin, representing about 5-10%. F or PP cells secrete pancreatic polypeptide, comprising around 1-2%. ε (epsilon) cells secrete ghrelin, which make up less than 1% of the islet cells.

ii) Insulin:

Source:

Insulin is secreted by the β (beta) cells in the islets of Langerhans of the pancreas. It is the principal anabolic hormone of the body and plays a central role in carbohydrate, fat, and protein metabolism.

Synthesis:

Insulin is synthesized in the rough endoplasmic reticulum (RER) of the β-cells. It is initially produced as preproinsulin, which is then cleaved in the endoplasmic reticulum to form proinsulin. In the Golgi apparatus, proinsulin is further cleaved into insulin and C-peptide through a series of enzymatic peptide cleavages. Both insulin and C-peptide are stored in secretory granules and are co-released into the bloodstream.

Function:

The primary function of insulin is to lower blood glucose levels. It does this by:

Increasing glucose uptake into muscle and adipose tissues via GLUT-4 transporters. Promoting glycogenesis — the storage of excess glucose in the liver and muscles in the form of glycogen. Inhibiting glycogenolysis (breakdown of glycogen) and gluconeogenesis (synthesis of new glucose). Enhancing lipogenesis and inhibiting lipolysis. Stimulating protein synthesis and inhibiting protein breakdown. Thus, insulin maintains glucose homeostasis and supports overall energy balance in the body.

Regulation:

Insulin secretion is primarily regulated by blood glucose levels.

When blood glucose levels increase (e.g., after a meal), insulin secretion is stimulated. When glucose levels decrease, insulin secretion is inhibited. Amino acids like arginine and leucine also stimulate insulin secretion. Elevated levels of free fatty acids and ketone bodies can stimulate insulin release to reduce lipolysis and restore energy balance. Parasympathetic (vagal) stimulation increases insulin secretion, while sympathetic stimulation (via

norepinephrine) may reduce it.

iii) Glucagon:

Source:

Glucagon is secreted primarily by the α (alpha) cells of the islets of Langerhans in the pancreas. In addition, smaller amounts are secreted by the A cells of the stomach and L cells of the intestine. It is a catabolic hormone that plays a key role in increasing blood glucose levels, especially during fasting.

Synthesis:

Glucagon is synthesized in the α-cells from a larger precursor molecule called preproglucagon. Preproglucagon is first processed into proglucagon, which is then cleaved by specific enzymes to form glucagon. In intestinal L-cells, proglucagon can also give rise to other peptides like GLP-1 (glucagon-like peptide-1), depending on tissue-specific processing

Functions:

Glucagon acts to increase blood glucose levels, especially during fasting or between meals. Its major functions include:

Stimulating glycogenolysis (breakdown of glycogen into glucose) in the liver. Promoting gluconeogenesis (synthesis of glucose from non-carbohydrate sources like amino acids). Enhancing lipolysis (breakdown of fats) to provide energy. Inhibiting glycolysis and glycogenesis. Acting as a physiological antagonist to insulin, helping maintain glucose homeostasis.

Regulation:

Glucagon secretion is tightly regulated by blood glucose and other metabolic signals:

Low blood glucose levels stimulate glucagon secretion. High blood glucose levels inhibit glucagon release. Certain amino acids such as alanine and arginine stimulate glucagon secretion, especially after a high-protein meal. Exercise and sympathetic stimulation (via catecholamines) also increase glucagon secretion to meet increased energy demands.

iv) Somatostatin:

Source:

Somatostatin is secreted from multiple sites in the body:

The hypothalamus (as growth hormone-inhibiting hormone), The δ (delta) cells of the islets of Langerhans in the pancreas, The D cells of the stomach and upper part of the small intestine.

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It acts both as a hormone and a paracrine regulator, depending on the site of secretion.

Synthesis:

Somatostatin is synthesized from a larger precursor molecule called prosomatostatin. In the pancreatic δ-cells, prosomatostatin is predominantly converted into somatostatin-14, the shorter and more common form. In the intestinal cells, especially in the duodenum, a larger form known as somatostatin-28 is mainly produced. Both forms have similar actions but differ in potency and half-life.

Functions:

Somatostatin is an inhibitory hormone that plays a major role in regulating other hormonal secretions. Its functions include:

Inhibiting insulin and glucagon secretion from the pancreas, thereby helping to prevent extreme fluctuations in blood glucose levels. Inhibiting the release of growth hormone (GH) from the anterior pituitary (when secreted from the hypothalamus). Suppressing gastric acid, pepsin, and digestive enzyme secretion from the stomach and pancreas. Reducing intestinal motility and nutrient absorption, thereby slowing digestion.

Thus, somatostatin helps maintain hormonal balance and digestive control.

Regulation:

Somatostatin secretion is stimulated by several factors, including:

An increase in nutrients such as glucose, amino acids, and fatty acids. Elevated levels of insulin and glucagon. Vagal (parasympathetic) stimulation can also promote somatostatin release.

v) Pancreatic Polypeptide (PP):

Source:

Pancreatic polypeptide is secreted by the F cells (also called PP cells) located in the islets of Langerhans of the pancreas. Smaller amounts may also be found in the small intestine, but the pancreas is the major site of secretion.

Synthesis:

Pancreatic polypeptide is synthesized in the F cells from a precursor molecule called prepropancreatic polypeptide. This precursor undergoes enzymatic cleavage to form the active hormone, pancreatic polypeptide (PP). It is stored in secretory granules and released into the bloodstream when stimulated.

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Functions:

Pancreatic polypeptide plays an important regulatory role in digestion and nutrient absorption. Its key functions include:

Inhibiting the secretion of pancreatic enzymes and bicarbonate ions from the exocrine pancreas. Suppressing gallbladder contraction, thereby reducing the flow of bile into the duodenum. Slowing gastric emptying, which delays the passage of food from the stomach to the small intestine. Modulating appetite and food intake through its action on the hypothalamus. By slowing digestion, it enhances nutrient absorption and allows more efficient processing of ingested food.

Regulation:

Pancreatic polypeptide secretion is influenced by various physiological and dietary factors:

Low blood glucose levels (hypoglycemia) stimulate PP secretion. High protein intake (especially after a protein-rich meal) increases its secretion. Fasting, exercise, and vagal (parasympathetic) stimulation also promote PP release. High blood glucose levels (hyperglycemia) and somatostatin inhibit PP secretion. Gastrointestinal hormones, like cholecystokinin (CCK) and secretin, may also affect its release indirectly.

vi) Diabetes Mellitus:

Introduction

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycaemia due to defects in insulin secretion, insulin action, or both. It affects the body's ability to regulate blood glucose levels and is associated with abnormalities in carbohydrate, fat, and protein metabolism.

It is one of the most common endocrine disorders, with a rapidly increasing global prevalence. The condition can lead to serious long-term complications involving the eyes, kidneys, nerves, and cardiovascular system. Understanding diabetes mellitus is essential for medical professionals, as early diagnosis and effective management can prevent or delay these complications and improve patient outcomes.

Types of Diabetes Mellitus

Diabetes mellitus is classified into several types based on its pathophysiology, onset, and cause. The major types include:

1. Type 1 Diabetes Mellitus (T1DM)

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Type 1 diabetes is an autoimmune disorder in which the immune system destroys the insulin-producing β-cells of the pancreas. It usually develops in childhood or adolescence and requires lifelong insulin therapy. This type accounts for about 5-10% of all diabetes cases.

2. Type 2 Diabetes Mellitus (T2DM)

This is the most common form of diabetes, accounting for nearly 90-95% of cases. It results from insulin resistance, often combined with relative insulin deficiency. It typically occurs in adults over the age of 40 but is increasingly being diagnosed in younger individuals due to sedentary lifestyles and obesity.

3. Gestational Diabetes Mellitus (GDM)

GDM is glucose intolerance first recognized during pregnancy. It is usually temporary but can increase the risk of developing type 2 diabetes later in life for both the mother and child.

Causes of Diabetes Mellitus

The causes of diabetes mellitus differ according to the type but are generally due to a combination of genetic, environmental, and lifestyle-related factors. In type 1 diabetes mellitus, the primary cause is an autoimmune destruction of the insulin-producing β-cells in the islets of Langerhans. This is often associated with genetic susceptibility, especially in individuals with specific HLA genes such as HLA-DR3 and HLA-DR4. Environmental triggers like viral infections, including rubella and coxsackievirus B, may also initiate or accelerate the autoimmune process.

In type 2 diabetes mellitus, the most common cause is insulin resistance in peripheral tissues, particularly muscle and adipose tissue, accompanied by a progressive decline in insulin secretion from β-cells. Obesity, particularly abdominal or visceral obesity, plays a central role in causing insulin resistance. Other contributing factors include a sedentary lifestyle, high-calorie diet rich in refined carbohydrates, increasing age, and a strong family history of diabetes.

Gestational diabetes mellitus is caused by hormonal changes during pregnancy, which result in increased insulin resistance. Placental hormones such as human placental lactogen reduce insulin sensitivity. The risk is higher in women who are overweight, of advanced maternal age, or have a history of gestational diabetes or large babies in previous pregnancies.

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Secondary diabetes may result from underlying conditions such as pancreatitis, pancreatic surgery, or hormonal disorders like Cushing's syndrome and acromegaly. Certain medications, including corticosteroids, thiazide diuretics, and antipsychotic drugs, can also impair insulin function and lead to diabetes mellitus.

Symptoms of Diabetes Mellitus

The symptoms of diabetes mellitus can vary depending on the severity of hyperglycaemia and the type of diabetes. In many cases, especially in early type 2 diabetes, the symptoms may be mild or go unnoticed for a long time. However, the classical symptoms of diabetes mellitus are polyuria (increased urination), polydipsia (excessive thirst), and polyphagia (increased hunger), which occur due to the high blood glucose level and osmotic diuresis.

Weight loss is commonly observed in type 1 diabetes due to the breakdown of fat and muscle tissues for energy, as insulin is either absent or insufficient. Fatigue is another frequent symptom, resulting from the inability of cells to utilize glucose efficiently. In addition to these, patients may complain of blurred vision, dry mouth, delayed wound healing, recurrent infections (especially fungal and urinary tract infections), and numbness or tingling in the extremities.

In severe or uncontrolled diabetes, especially in type 1 cases, symptoms may progress rapidly and lead to diabetic ketoacidosis, presenting with nausea, vomiting, abdominal pain, and even altered consciousness. In gestational diabetes, symptoms are usually mild or absent but may be discovered during routine screening tests in pregnancy.

Diagnosis of Diabetes Mellitus

The diagnosis of diabetes mellitus is based on laboratory investigations that assess the level of glucose in the blood. One of the most commonly used tests is fasting blood glucose, where a level equal to or above 126 mg/dL after at least eight hours of fasting is considered diagnostic of diabetes. Another standard test is the oral glucose tolerance test (OGTT), in which 75 grams of glucose is given orally and blood glucose is measured two hours later. A value of 200 mg/dL or more at the two-hour mark confirms diabetes.

Random blood glucose testing is also used, especially in symptomatic patients, where a level of 200 mg/dL or more, regardless of fasting, is considered diagnostic if accompanied by classic symptoms of diabetes. Glycated

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haemoglobin (HbA1c) is another reliable marker that reflects the average blood glucose level over the past two to three months. An HbA1c value of 6.5% or more is diagnostic of diabetes mellitus.

Other supportive investigations may include urine sugar testing, although it is less reliable due to renal threshold variations. Ketone testing in blood or urine may be done in suspected diabetic ketoacidosis. C-peptide levels can help differentiate between type 1 and type 2 diabetes, as C-peptide is absent or low in type 1 and relatively preserved in type 2. These diagnostic methods help in the early identification and classification of diabetes for prompt treatment and complication prevention.

Treatment of Diabetes Mellitus

The treatment of diabetes mellitus aims to maintain normal blood glucose levels, prevent acute complications, and reduce the risk of long-term organ damage. The approach to treatment differs based on the type of diabetes and the patient's condition but generally includes lifestyle modification, pharmacological therapy, and regular monitoring.

In type 1 diabetes mellitus, insulin therapy is the cornerstone of treatment. Patients require lifelong insulin replacement because their bodies are unable to produce insulin. Various forms of insulin such as rapid-acting, intermediate-acting, and long-acting preparations are used either alone or in combination depending on blood sugar patterns. Insulin is administered subcutaneously and doses are adjusted based on blood glucose levels and dietary intake.

In type 2 diabetes mellitus, treatment typically begins with lifestyle modifications, including a balanced diet with controlled carbohydrate intake, regular physical activity, and weight reduction. If these measures are not sufficient, oral hypoglycaemic agents such as metformin, sulfonylureas, DPP-4 inhibitors, SGLT2 inhibitors, and thiazolidinediones are prescribed. In some cases, insulin may also be required when oral medications fail to achieve adequate glycaemic control.

In gestational diabetes, dietary management and exercise are first advised. If blood glucose remains uncontrolled, insulin is the preferred medication as oral hypoglycaemic drugs may cross the placenta and affect the fetus.

Monitoring of blood glucose using glucometers or continuous glucose monitors is essential for adjusting therapy. HbA1c testing is done periodically to assess long-