Review Article **AJAP (2020) 3:12**

American Journal of Anatomy and Physiology
(ISSN:2637-4714)

Metabolism: Pancreas and glycemic regulating hormones

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ABSTRACT

Understanding the physiology of the metabolic regulation of hormones responsible for glycemic control is of fundamental importance for a thorough understanding of Diabetes Mellitus. In this scenario, this chapter is intended to explain this regulation, as well as the main hormones that participate in this metabolic process.

Keywords: Insulin, Glucagon, Hormone Regulation

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How to cite this article:
Francisco Henrique da Silva, Matheus Alves Siqueira de Assunção, Alamisne Gomes da Silva, Evandro Valentim da Silva, Aline Fernanda Carneiro Cardoso, Fálba BernadeteRamos dos Anjos . Metabolism: Pancreas and glycemic regulating hormones .American Journal of Anatomy and Physiology, 2020, 3:12.

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Introduction

The pancreas (figure 1) is formed by two distinct components: the exocrine pancreas, a reservoir of digestive enzymes and the endocrine islets, the source of the vital metabolic hormone insulin[1]. This organ secretes two hormones, through the islets of Langerhans, which are important in the normal regulation of glucose metabolism, such as insulin and glucagon[2].

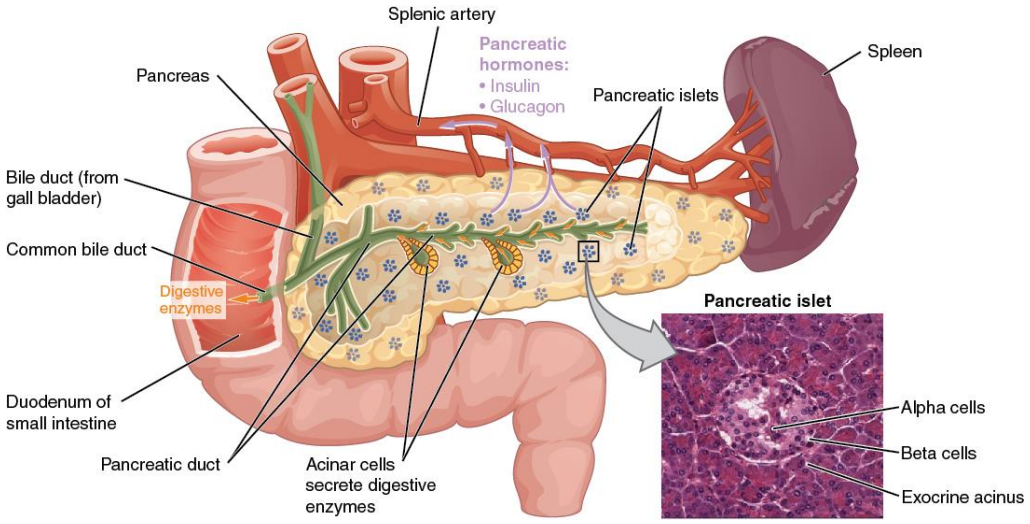

Figure 1. Physiological anatomy of acini and Langerhans image.

Source: OPENSTAX COLLEGE. Illustration from Anatomy & Physiology. 2013[26].

The islets contain three main cell types, the cells alpha (α), beta (β) and delta (δ). A alpha cells represent about 20% of the total islet cells and secrete glucagon; The beta constitute about 75% and secrete insulin and amylin and delta cells represent approximately 6% of the total and secrete somatostatin[3], according to table 1. In addition to the main secretory cells, there is a presence of Epsilon cells and as PP cells.

Table 1. Composition and cell production of pancreatic islets.

Cell type	Composition (%)	Hormonal production
Alphas Cells	18-20	Glucagon
Beta Cells	73-75	Insulin and Amylin
Delta Cells	4-6	Somatostatin
PP Cells	1	Pancreatic Peptide
Epsilon Cells	>1	Ghrelin

Addapted from Mansano [3]

In addition, the pancreas secretes digestive juice in the duodenum, aiding in the metabolism of foods where the hormones secreted are insulin, glucagon, somatostatin, pancreatic polypeptide and amylin [4].

Insulin

Insulin (Figure 2) is a protein monomer with two linked polypeptide chains, containing 21 amino acids in chain A and 30 amino acids in chain B, linked by disulfide bridge[5]. Insulin is a hormone

whose function is to maintain blood glucose within normal limits. The production of this hormone is regulated in response to the quality and quantity of the food eaten, after a meal rich in carbohydrates[6].

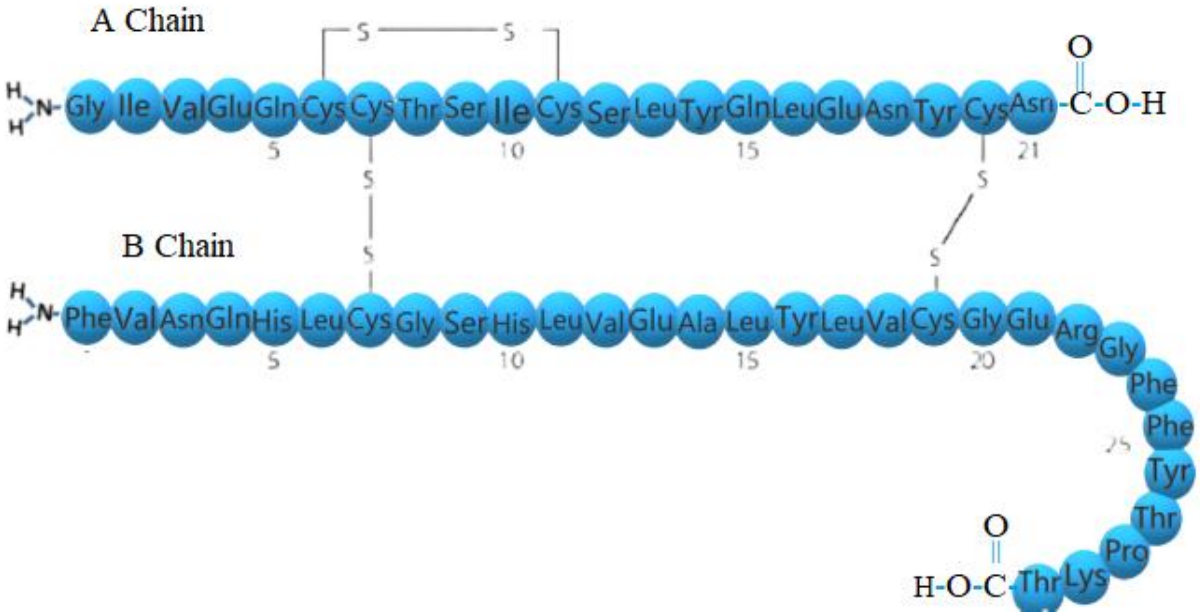

Figure 2. Structure of insulin **Source**: Author

Glucagon

Glucagon (Figure 3) has 29 amino acids in its chain and causes an opposite effect to that of insulin[7]. The main effects on glucose metabolism are the breakdown of hepatic glycogen (glycogenolysis) and the increase in gluconeogenesis in the liver. These effects increase the availability of glucose to other organs of the body, that is, it has a hyperglycemic action[8].

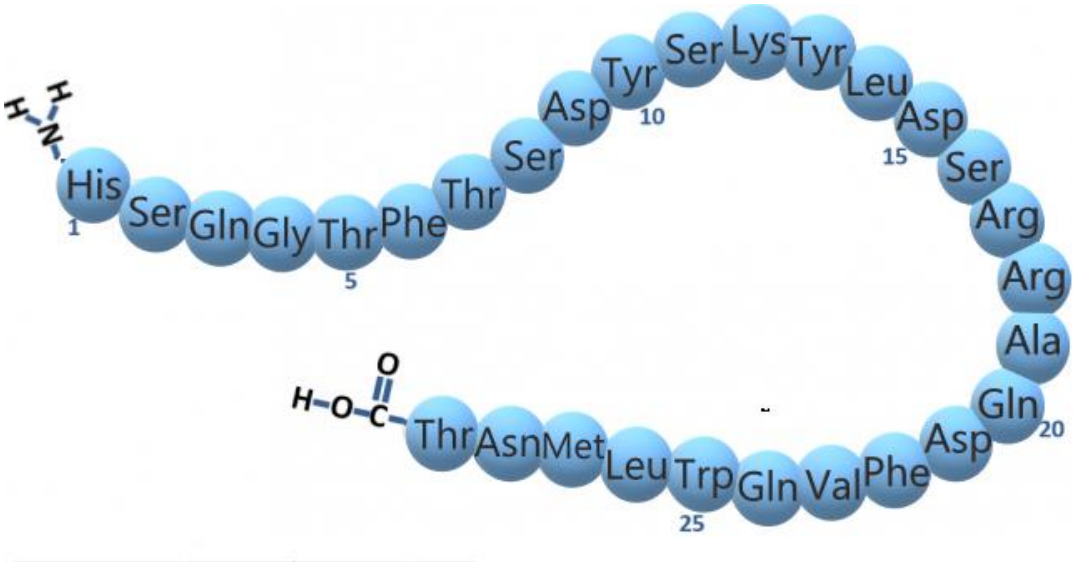

Figura 3. Structure of Glucagon. **Source**: Author

The hormones insulin and glucagon function as important feedback systems to regulate glycemia9 (Figure 4). When the glucose concentration rises, insulin is secreted, this causes the glu-

cose concentration to drop until it returns to normal (fasting between 70 to 99 mg / dL) [10] inversely, the decrease in blood glucose stimulates the secretion of glucagon that works in the opposite way the action of insulin, raising glucose to a normal level [11].

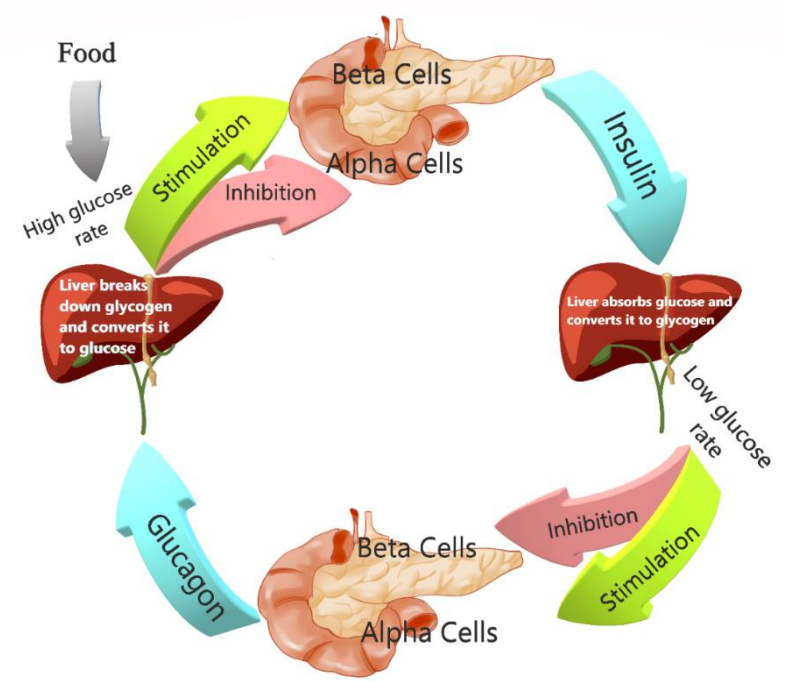

Figure 4. Regulation of insulin and glucagon Addapted from Castilho[27].

The interrelationships established between cell types in the islets of Langerhans enable intercellular communication[12], and in this way insulin inhibits the secretion of glucagon, amylin inhibits the secretion of insulin and somatostatin inhibits the secretion of both insulin and glucagon[13].The liver is responsible for producing about 90% of glucose (Figure 5) in the fasted state, 2/3 are used by non-insulin dependent tissues, mainly the central nervous system[14, 15]. Glucose is a monosaccharide, which when absorbed into the bloodstream causes insulin secretion. As a consequence, glucose uptake, storage and use begins especially for muscles, adipose tissue and liver, with a hypoglycemic action[16].

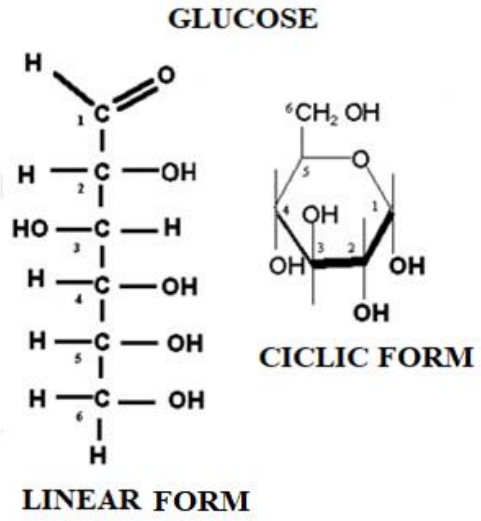

Figura 5. Structure of Glucose **Source**: Author

Metabolic glucose changes during pregnancy

In the first half of pregnancy, endocrine-metabolic changes are characterized by inhibition of alanine, an important glycogen precursor, and by greater sensitivity of insulin tissues, leading to a decrease in fasting glycemic levels[17]. A partir da segunda metade, o aumento da tolerância à glicose e do hiperinsulinismo, caracterizando a resistência à insulina, parece ser um evento pós-receptor e está qualificado com ação metabólica diabética, como progesterona, estrogênio, prolactina, prolactina, cortisol e somatotrofina coriônica[18, 19].

With the advancement of hormone production management, it is possible to activate peripheral insulin resistance[20], triggering GDM, that is, it occurs when the pancreas reaches maximum insulin production and this volume remains insufficient to reverse the effects of placental hormones. Another factor that can be considered hyperglycemic is the degradation of insulin by enzymes of the placental membrane, similar to liver insulins[21,22]Thus, the risk of diabetes increases or works harder to produce insulin, but insulin is not low in blood glucose levels. An extra glucose in the blood crosses the placenta, also stimulating the production of insulin in the concept[23], which will have more energy available during its development. This behavior can trigger complications, being an energy stored in the form of fat, favoring the occurrence of macrosomia (excess weight in the newborn) [24,25].

Conclusion

The regulatory physiology of the pancreas provides effective glycemic control, where alterations related to this regulation can cause pathologies and comorbidities, as observed in Diabetes, capable of promoting symptoms that lead the individual to clinical disease.

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