

Immobilization Stress and the Functional State of the Pancreas

Yusupova I.A., Azimova.S.B., Akhmedova D.B.

Tashkent State Medical University

Abstract: This article examines the effect of immobilization stress on the functional state of the pancreas. The neurohumoral mechanisms activated under stress and their influence on the exocrine and endocrine functions of the gland are analyzed. It has been established that stress causes pronounced morphofunctional changes, including reduced enzyme secretion, hyperglycemia, and dystrophic processes in pancreatic tissues. Identifying these changes allows for a deeper understanding of the pathogenesis of stress-associated disorders of the digestive system.

Keywords: immobilization stress, pancreas, exocrine function, endocrine function, hyperglycemia, morphology.

Introduction. Stress reactions of the body—whether triggered by a sudden loud sound requiring an immediate response or by chronic psychological strain—are accompanied by complex, multi-stage neurohumoral changes [1]. This means that the nervous and hormonal systems work closely together to mobilize the body's resources for adaptation to changing conditions. The sympathetic nervous system is activated first, leading to the release of adrenaline and noradrenaline, which increase heart rate, blood pressure, and blood glucose levels—ensuring the body is supplied with energy and prepared for action. In parallel, the hypothalamic–pituitary–adrenal (HPA) axis is activated, resulting in the release of cortisol—a hormone that regulates metabolism, suppresses the immune system, and helps the body cope with prolonged stress [2].

One form of stress exposure that attracts significant scientific interest is immobilization stress. This type of stress occurs during prolonged forced immobility, when the organism is unable to react actively to a threat [3]. For example, an animal caught by a predator or a person trapped in a confined space experiences immobilization stress. Such conditions are often used in experimental models, particularly in studies on laboratory animals, to investigate the effects of stress factors on various organs and systems. Subjecting animals to controlled immobilization stress allows researchers to observe physiological and behavioral changes and better understand the mechanisms and consequences of stress reactions.

Particular interest in stress research is directed toward the pancreas—an organ with a unique dual role. It functions both as an exocrine organ producing digestive enzymes (such as amylase, lipase, and proteases) [4] and as an endocrine organ releasing hormones that regulate blood glucose levels, such as insulin and glucagon. Insulin lowers blood glucose by enabling cellular uptake, while glucagon raises glucose levels by mobilizing hepatic reserves. The pancreas is deeply involved in metabolic regulation and digestion, ensuring stable glucose levels and the breakdown of carbohydrates, fats, and proteins. Stress, especially chronic stress, can significantly affect pancreatic function, leading to metabolic disturbances and increasing the risk of diseases such as diabetes.

Aim of the study. To investigate the impact of immobilization stress on the functional state of the pancreas and to determine the physiological and morphological changes that develop under this type of stress exposure.

Methods. The research was conducted through a literature review using major scientific and medical resources, including eLIBRARY, PubMed.NCBI, CyberLeninka, and the official portal of the International Diabetes Federation (IDF). Publications from 2000 to 2025 were included.

Results of our research.

Effect of stress on pancreatic physiology.

During immobilization stress—prolonged restriction of mobility accompanied by a sense of helplessness—the body initiates a complex cascade of reactions, the central element of which is activation of the hypothalamic–pituitary–adrenal axis (HPA). This axis includes the hypothalamus, pituitary gland, and adrenal glands, which work in tight coordination. In response to stress, the hypothalamus releases releasing hormones that stimulate the pituitary to secrete adrenocorticotrophic hormone (ACTH). ACTH then stimulates the adrenal cortex to produce corticosteroids, primarily cortisol. Simultaneously, sympathetic nervous system fibers are activated, causing the release of catecholamines—adrenaline and noradrenaline—from adrenal chromaffin cells [5].

These hormones enter the bloodstream and exert systemic effects, mobilizing energy reserves and preparing the body to overcome stress. Their influence extends to the pancreas, which plays a key role in glucose regulation.

Stress affects the pancreas in several ways. First, the balance of the autonomic nervous system is disturbed, with sympathetic activity predominating. This can cause vasoconstriction in pancreatic tissues, impairing microcirculation and delivering less oxygen and nutrients to alpha- and beta-cells. Second, regulation of digestive enzyme secretion is disrupted, leading to altered levels of amylase, lipase, and proteases, which may cause digestive disorders. Third, secretion of pancreatic hormones such as insulin and glucagon becomes altered. Increased glucagon and decreased insulin secretion contribute to stress-induced hyperglycemia—an adaptive response that, when prolonged, may lead to insulin resistance or type 2 diabetes. Additionally, microcirculatory disturbances caused by vasoconstriction and increased blood viscosity can result in ischemia and cellular damage in the pancreas [6,7].

The functional state of the pancreas—a vital organ performing both exocrine and endocrine functions—is highly vulnerable to stress. Its deterioration occurs through impaired enzyme production and through dysfunction of endocrine cells regulating blood glucose [8]. Under prolonged stress, complex biochemical processes develop that negatively affect pancreatic function.

Particularly, production of digestive enzymes—amylase, lipase, and proteases—decreases. This leads to impaired digestion, heaviness after meals, bloating, and other dyspeptic symptoms. Outflow of pancreatic juice into the duodenum also becomes impaired. Reduced outflow causes congestion within the gland, predisposing it to inflammation—pancreatitis. Chronic stress may also cause spasm in the smooth muscles surrounding pancreatic ducts, worsening obstruction and contributing to the development of scarring [9].

The endocrine portion of the pancreas is similarly affected. Chronic immobilization and accompanying stress increase glucocorticoid levels, which suppress insulin secretion and decrease tissue sensitivity to insulin, contributing to hyperglycemia [10]. Persistent metabolic disruptions may progress to insulin resistance, a major risk factor for type 2 diabetes, especially in genetically predisposed individuals [11].

Morphological changes under immobilization stress reflect profound structural disturbances. Microscopy reveals edema, vacuolization of acinar cells, inflammatory infiltrates, and in severe cases, areas of necrosis. These changes significantly impair pancreatic functionality and predispose to chronic disease development.

Conclusion. Immobilization stress exerts a complex adverse impact on the pancreas, disrupting both digestive and hormonal functions. These changes may trigger acute or chronic diseases, especially in the presence of additional risk factors. Understanding stress-induced pancreatic dysfunction is important for the prevention and treatment of disorders associated with pancreatic impairment.

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