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The Insulin-heart Connection: Linking Physiology to Insulin Resistance

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Abstract

The relationship between insulin and heart function is a critical yet complex aspect of human physiology. The heart, a metabolically demanding organ, relies heavily on insulin for energy regulation. Insulin, a hormone produced by the pancreas, is essential for glucose uptake in tissues and plays a pivotal role in metabolic homeostasis. Insulin resistance, a condition characterized by diminished cellular response to insulin, leads to various metabolic disorders, including Type 2 Diabetes Mellitus (T2DM) and Cardiovascular Diseases (CVD). This mini review aims to bridge the physiological aspects of insulin function with the pathological consequences of insulin resistance, highlighting the intricate connection between insulin and heart health.

Keywords: Cardiovascular diseases • Diabetes mellitus • Epidemiological findings • Pathological consequences

Introduction

Insulin is synthesized in the β-cells of the pancreatic islets of Langerhans. Its secretion is primarily stimulated by increased blood glucose levels, although amino acids and gastrointestinal hormones also play a role. Upon stimulation, insulin is released into the bloodstream, where it facilitates glucose uptake by tissues, particularly muscle and adipose tissue, through its receptor-mediated action. The insulin signaling pathway is critical for maintaining glucose homeostasis. Insulin binds to the Insulin Receptor (IR) on the cell surface, initiating a cascade of events. Binding of insulin to IR leads to its autophosphorylation, activating its intrinsic tyrosine kinase activity. The phosphorylated IR phosphorylates Insulin Receptor Substrates (IRS), which then activate Phosphatidylinositol 3-kinase (PI3K). PI3K activates Akt (protein kinase B), a central node in insulin signaling that promotes glucose transporter type 4 (GLUT4) translocation to the cell membrane, facilitating glucose uptake. Akt also enhances glycogen synthesis by inhibiting Glycogen Synthase Kinase-3 (GSK-3) [1].

Literature Review

The heart's primary energy sources are fatty acids and glucose, with insulin playing a crucial role in substrate preference and utilization. Under normal conditions, insulin promotes glucose uptake in cardiomyocytes by stimulating GLUT4 translocation, ensuring adequate ATP production. Insulin also modulates fatty acid metabolism by inhibiting Hormone-Sensitive Lipase (HSL) and activating Acetyl-CoA Carboxylase (ACC), thus reducing fatty acid oxidation and promoting triglyceride storage. Insulin enhances nitric oxide (NO) production in endothelial cells, promoting vasodilation and improving blood flow. Insulin reduces inflammation by inhibiting nuclear factor kappa B (NF-κB) signaling and decreasing pro-inflammatory cytokine production [2].

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Insulin signaling via the PI3K/Akt pathway inhibits apoptotic pathways, thus protecting cardiomyocytes from programmed cell death. Insulin resistance is a state where normal levels of insulin fail to produce an adequate physiological response. This condition involves multiple factors. Genetic variations can affect insulin signaling components, predisposing individuals to insulin resistance. Excess adipose tissue, particularly visceral fat, secretes adipokines and inflammatory cytokines that interfere with insulin signaling. Lack of physical activity reduces insulin sensitivity by diminishing glucose uptake and altering muscle metabolism [3].

Discussion

Phosphorylation of IRS on serine/threonine residues (rather than tyrosine) impairs its ability to activate downstream signaling. Chronic inflammation activates stress kinases such as c-Jun N-terminal kinase (JNK) and IκB kinase (IKK), which inhibit insulin signaling. Excessive intracellular lipid intermediates (e.g., diacylglycerol) activate Protein Kinase C (PKC), which disrupts insulin signaling. Insulin resistance alters cardiac metabolism, leading to increased reliance on fatty acids for energy production. This shift results in greater oxygen consumption and reduced cardiac efficiency, predisposing the heart to ischemic injury. Additionally, impaired glucose uptake can cause glucotoxicity, further damaging cardiac cells. Increased workload and metabolic stress promote hypertrophic remodeling of the heart. Insulin resistance impairs myocardial relaxation, leading to diastolic dysfunction and Heart Failure with Preserved Ejection Fraction (HFpEF) [4].

Insulin resistance accelerates atherosclerotic plaque formation, increasing the risk of Coronary Artery Disease (CAD). A balanced diet rich in whole grains, lean proteins, healthy fats, and low in processed sugars can improve insulin sensitivity. Regular physical activity enhances glucose uptake in muscles and improves overall insulin sensitivity. Several pharmacological agents are used to manage insulin resistance and its cardiac complications. This first-line antidiabetic drug improves insulin sensitivity and has cardioprotective effects. TZDs enhance insulin sensitivity by activating peroxisome Proliferator-Activated Receptor-Gamma (PPAR-γ), although they may have adverse cardiovascular effects [5].

These agents improve insulin secretion and have beneficial effects on cardiovascular outcomes. These drugs promote glucose excretion via urine and have shown significant cardiovascular benefits in diabetic patients. Targeting inflammatory pathways may improve insulin sensitivity and reduce cardiovascular risk. Enhancing mitochondrial function can improve cardiac

energy metabolism in insulin-resistant states. Targeting genetic factors involved in insulin signaling holds potential for treating insulin resistance at its root cause [6].

Conclusion

The intricate connection between insulin and heart health underscores the importance of maintaining insulin sensitivity for cardiovascular well-being. Insulin resistance disrupts normal cardiac metabolism, leading to structural and functional impairments that predispose individuals to heart disease. Addressing insulin resistance through lifestyle modifications and pharmacological interventions can significantly reduce cardiovascular risk and improve overall health outcomes. Continued research into the molecular mechanisms and therapeutic targets of insulin resistance will pave the way for innovative treatments and improved management of cardiovascular diseases.

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Conflict of Interest

Authors declare no conflict of interest.

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