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Review IJIN

A Comprehensive Review on Insulin Resistance

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Abstract

Insulin resistance is a complex condition where cells in the body, such as those in the liver, fat, and muscle, do not respond properly to insulin. Insulin resistance can be temporary or incurable, leading to increased insulin production known as hyperinsulinemia, which may contribute to obesity and eventually progress to type 2 diabetes. This mini-review elaborates the historical context of insulin discovery and its therapeutic use. It highlights the importance of insulin and insulin resistance in the context of metabolic diseases and discusses factors contributing to insulin resistance, such as poor diet, obesity, heredity, physical inactivity, and certain medical conditions. Lifestyle changes and pharmacological therapies are suggested for managing insulin resistance. The role of insulin in maintaining blood glucose levels and its resistance in various tissues, leading to compensatory hyperinsulinemia, are also discussed. It emphasizes the significance of understanding risk factors, lifestyle changes, and pharmacological interventions in preventing and treating insulin resistance. Additionally, it touches upon insulin's effects on glucose metabolism, the insulin resistance syndrome, and the clinical diagnostic entity known as metabolic syndrome. The impact of various factors on insulin secretion and production is also mentioned. Overall, it provides a comprehensive overview of insulin resistance, its consequences, and approaches to its management.

Keywords: Insulin resistance, type 2 diabetes, hyperinsulinemia, metabolic syndrome, Lifestyle changes

INTRODUCTION

Insulin resistance, or impaired insulin sensitivity, is a type of mechanism that happens in which the cells that are present in your body's organs, like the liver, fat cells, and muscle cells, don't retaliate as they should to insulin. Insulin resistance can be incurable or temporary. The development of insulin resistance also increases insulin production; that's called hyperinsulinemia. The high level of insulin can lead to obesity (weight gain), which in turn is harmful for health and makes insulin resistance inadequate. Insulin resistance progresses to type 2 diabetes. Insulin resistance spoils glucose disposal [1]. In civilised countries, diabetes and obesity reach pandemic proportions, and the role of insulin resistance and its outcomes are gaining distinction. In the forefront of medical research, the roles of insulin and insulin resistance gain great importance [2]. Insulin and insulin-like peptides have been recognised in all types of animals. Insulin, a peptide hormone produced by the endocrine system, attaches to receptors on the surface of target cells. It exerts both direct and indirect effects on target organs.

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According to Petersen MC and Shulman GI (2018) [3], insulin resistance occurs when a specified amount of insulin, whether produced within the body or administered externally, fails to adequately facilitate glucose uptake. Lebovitz HE (2001) [4] highlights that insulin resistance is crucial in its progression, serving as a fundamental aspect in the pathogenesis of type 2 diabetes. Insulin promotes adipocyte triglyceride storage through several mechanisms. Insulin also promotes the intake of fatty acids acquired from circulating lipoproteins. In adipocytes, insulin exertion also requires modifications in gene transcription [5].

Insulin resistance is a critical malignant constituent of many metabolic diseases. In tissues targeted by insulin, insulin resistance describes a condition where there is a diminished response to the hormone's action. For fat-induced insulin resistance, there are potential therapeutic approaches that target abnormal fat assembling in the liver [6]. They also enhance energy consumption in the skeletal muscle (2001) [7].

REVIEW OF LITERATURE

A decade later, in 1921, insulin was at last isolated, refined, and made available in a form that could be used therapeutically. Canine tests were initiated in May 1921 by Toronto surgeon Banting, who was supervised by McLeod, Professor of Carbohydrate Metabolism, and with the help of medical student Best. They noticed a reduction in blood glucose in dogs with pancreatectomy-induced diabetes by giving them cold pancreatic extracts intravenously [6]. Joining the team, biochemist Collip advanced the research by demonstrating that the extract not only replenished glycogen in the liver but also improved the removal of ketones, a finding reported to the American Physiological Association in December 1921.

A month later, in January 1922, the first human trials were carried out on a 14-year-old boy with diabetes, whose biochemical abnormalities and clinical symptoms were virtually corrected when the pancreas isolate was given to him [8]. The active ingredient was dubbed insulin in May 1922, and the Association of American Physicians was given a presentation of the study's findings. After that, Eli Lilly started producing pig insulin and improved purification using isoelectric precipitation. By early 1923, commercial amounts were produced. The Nobel Prize was given to Banting and McLeod in 1923 [9]. Insulin resistance is a condition characterized by the diminished responsiveness of the body's cells to insulin, a hormone produced by the pancreas to regulate blood sugar levels [10]. Hyperglycemia, or elevated blood sugar results from cells' inability to properly absorb glucose from the bloodstream when they are resistant to insulin. If treatment is not received, this may eventually result in type 2 diabetes [11]. Insulin resistance is caused by a number of variables, including poor food, obesity, heredity, physical inactivity, and certain medical diseases, including PCOS [12]. Because it generates chemicals that prevent insulin from doing its job, excess fat, particularly belly fat, is a major contributor to insulin resistance.

Moreover, non-alcoholic fatty liver disease, elevated blood pressure, and aberrant cholesterol levels are associated with insulin resistance [13]. Changes in lifestyle, such as eating a balanced diet low in refined carbs and sugars, exercising frequently, and keeping a healthy weight, are necessary to manage insulin resistance. Doctors may prescribe drugs like metformin to assist lower blood sugar and increase insulin sensitivity [14]. To sum up, insulin resistance is a serious health issue that can result in type 2 diabetes and other life-threatening consequences. Preventing and treating insulin resistance requires an understanding of risk factors, addressing them with lifestyle changes, and, when required, pharmacological therapies [15].

Insulin, a peptide hormone, is produced and released by the pancreatic islet cells known as β cells of Langerhans. It helps to maintain normal blood glucose levels by encouraging cell reproduction and growth through its mutagenic effects, aiding cellular glucose uptake, and regulating the metabolism of carbohydrates, fats, and proteins [16]. Insulin resistance is characterized by an attenuated biological

response to a normal or increased insulin level. Traditionally, this has been understood to mean reduced sensitivity to insulin-mediated glucose elimination [17].

Compensatory hyperinsulinemia occurs when muscle and fat tissues show resistance to insulin, leading to increased insulin production by pancreatic β cells to sustain normal blood sugar levels. The collection of anomalies and associated physical consequences that affect insulin-resistant people is more frequently referred to as insulin resistance syndrome [18]. The composite consequences of excess insulin and varying resistance to its actions are expected to be reflected in signs of the insulin resistance syndrome, given tissue heterogeneity in insulin dependency and sensitivity [19]. The clinical diagnostic entity known as metabolic syndrome is used to identify those who are at a high risk of developing the cardiovascular morbidity linked to insulin resistance [20].

Alterations in gene transcription, translation, and post-translational modification within the Golgi apparatus, along with factors influencing the release of insulin from secretory granules, can collectively influence insulin secretion. Influences on the bulk and differentiation of β cells may lead to modifications that are longer-lasting [21]. It is not unexpected that glucose has a variety of effects on insulin production and secretion, considering insulin's essential function in glucose utilization and metabolism [22]. Nevertheless, these processes are also impacted by additional factors, including fatty acids, amino acids, acetylcholine, pituitary adenylate cyclase-activating polypeptide (PACAP), glucose-dependent insulinotropic polypeptide (GIP), glucagon-like peptide-1 (GLP-1), and various other agonists [23].

DISCUSSION AND CONCLUSION

Insulin resistance is a complicated disorder in which liver, fat, and muscle cells don't respond to insulin as they should. Insulin resistance increases insulin production, causing hyperinsulinemia. High insulin levels can cause obesity, which harms health and reduces insulin resistance. Type 2 diabetes arises due to insulin resistance, which hinders the effective use of glucose. Insulin promotes adipocyte triglyceride storage in several ways. Insulin promotes lipoprotein-derived fatty acid consumption. Adipose tissue insulin exertion also alters gene transcription. High blood pressure, cholesterol, and non-alcoholic fatty liver disease are linked to insulin resistance. Insulin, a peptide hormone secreted by the pancreatic beta cells within the islets of Langerhans, plays a crucial role in promoting cell growth and reproduction, facilitating glucose uptake, and regulating carbohydrate, lipid, and protein metabolism to maintain optimal blood glucose levels. Insulin resistance refers to a diminished biological response to normal or elevated levels of insulin. Additionally, various factors such as fatty acids, amino acids, acetylcholine, pituitary adenylate cyclase-activating polypeptide (PACAP), glucose-dependent insulinotropic polypeptide (GIP), glucagon-like peptide-1 (GLP-1), and other stimulatory agents can further influence these actions. This review article shows the overall resistance of insulin to diabetes and obesity in people.

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