Open Access Journal of
Gerontology & Geriatric Medicine
ISSN: 2575-8543

Juniper Publishers
key to the Researchers

Mini Review
Volume 8 Issue 1 - April 2024
DOI: 10.19080/OAJGGM.2024.08.555730

OAJ Gerontol & Geriatric Med

A Comprehensive Review on Insulin Resistance

Luqman Hakeem[1], Hussnain Ahmad[2], Arooj Aslam[3], Kashif Nawa[3], Umer Ali[4], Hafiza Rabia Shaifq[3], Nabila Iqbal[4] and Muhammad Kaleem Ullah[3]

[1]*Department of chemistry Government college University Faisalabad Pakistan*

[2]*Department of Botany university of Agriculture Fasialabad Pakistan*

[3]*Department of Zoology university of Okara Pakistan*

[4]*Department of Biological sciences Tennessee State University of Texas USA*

Submission: March 4, 2024; **Published:** April 25, 2024

***Corresponding author**: Luqman Hakeem, Department of chemistry Government college University Faisalabad Pakistan

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, the it provides a comprehensive overview of insulin resistance, its consequences, and approaches to its management.

Keywords: Insulin Resistance; Obesity and Diabetes; Hyperinsulinemia; Metabolic Syndrome; Lipoprotein-derived fatty acid

Abbreviations: PACAP: Pituitary Adenylate Cyclase-Activating Polypeptide; GIP: Glucose-Dependent Insulin Tropic Polypeptide; GLP-1: Glucagon-Like Peptide-1

Introduction

Insulin resistance is a convoluted condition or impaired insulin sensitivity is a type of mechanism happens in which the cells that are present in your body organs like liver cell, fat cell and muscle cell don't retaliate as they should to insulin. Insulin resistance can be incurable or temporary. Development of insulin resistance also increases insulin production that's called hyperinsulinemia. The high level of insulin can lead towards obesity (weight gain) which in return harmful for health and make insulin resistance inadequate. Insulin resistance progresses to type 2 diabetes. Insulin resistance spoil glucose disposal. Seong J, Kang JY, Sun JS, & Kim KW [1]. In the civilized countries, diabetes and obesity reach pandemic proportions, the role of insulin resistance and its outcomes are attain distinction. In the forefront of medical, research the role of insulin and insulin resistance gain great importance Wilcox G [2]. Insulin and insulin like peptide have been recognize in all type of animals. Insulin is an endocrine peptide hormone that binds plasma membrane bound receptors in the target cells. On target tissues insulin also utilize indirect effects. Petersen MC, Shulman GI [3]. Insulin resistance is defined clinically as the inability of known quantity of endogenous or exogenous insulin to boost glucose absorption. Lebovitz HE [4]. Insulin resistance play a very important role in its development. It is a rudimentary characteristic of the etiology of type 2 diabetes. Insulin promote adipocyte triglyceride store by a number of implements. Insulin also promote the intake of fatty acids acquire from circulating lipoprotein. In adipocyte insulin exertion also require modifications in gene transcription Kahn BB, Flier JS [5]. Insulin resistance is the critical malignant constituent of many

metabolic diseases. In insulin targeting tissues insulin resistance is a physiological state of decreased responsiveness. For fat induced insulin resistance potential therapeutic approaches that intent abnormal fat assembling in the liver and [6] it's also enhance energy consumption in the skeletal muscle. Zimmet P, Alberti KGMM, Shaw J [7].

Discussions

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]. Biochemist Collip, who had joined the team, further established that this extract also restored hepatic glycogen mobilization and the ability to remove ketones when this study was 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 disorder where the body's cells lose their receptivity to the hormone insulin which is secreted by the pancreas and controls 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 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 is a peptide hormone that is secreted by the β cells of the pancreatic islets 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].

When peripheral insulin resistance exists in muscle and adipose tissue and pancreatic β cell secretion rises to maintain normal blood glucose levels, compensatory hyperinsulinemia results. The collection of anomalies and associated physical consequences that affect insulin-resistant people 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 used to identify those who are at a high risk of developing the cardiovascular morbidity linked to insulin resistance [20].

Changes in gene transcription, translation, and post-translational modification in the Golgi, as well as variables affecting insulin release from secretory granules, can all have an impact on 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]. However, these activities are also influenced by other elements such as fatty acids, amino acids, acetylcholine, pituitary adenylate cyclase-activating polypeptide (PACAP), glucose-dependent insulin tropic polypeptide (GIP), glucagon-like peptide-1 (GLP-1), and a number of other agonists [23].

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 results from insulin resistance. Insulin resistance impairs glucose utilization. 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 is a peptide hormone released by the beta cells of the pancreatic islets of Langerhans. Mutagenicity promotes cell reproduction and growth, glucose uptake, carbohydrate, lipid, and protein metabolism, maintaining

How to cite this article: Luqman H, Hussnain A, Arooj A, Kashif N, Umer A, et al. A Comprehensive Review on Insulin Resistance. OAJ Gerontol & Geriatric Med. 2024; 8(1): 555730. DOI: 10.19080/OAJGGM.2024.08.555730

appropriate blood glucose levels. Insulin resistance is a reduced biological response to normal or high insulin levels. These actions are additionally impacted by several factors including fatty acids, amino acids, acetylcholine, pituitary adenylate cyclase-activating polypeptide (PACAP), glucose-dependent insulin tropic polypeptide (GIP), glucagon-like peptide-1 (GLP-1), and several other stimulating substances. This review article shows the overall resistance of insulin on diabetes and obesity peoples.

References

1. Seong J, Kang JY, Sun JS, Kim KW (2019) Hypothalamic inflammation and obesity: a mechanistic review. Arch Pharma Res 42(5): 383-392.
2. Wilcox G (2005) Insulin and insulin resistance. Clin Biochem Rev 26(2): 19-39.
3. Petersen MC, Shulman GI (2018) Mechanisms of insulin action and insulin resistance. Physiol Rev 98(4): 2133-2223.
4. Lebovitz HE (2001) Insulin resistance: definition and consequences. Exp Clin Endocrinol & Diabetes 109(Suppl 2): S135-S148.
5. Kahn BB, Flier JS (2000) Obesity and insulin resistance. J Clin Invest 106(4): 473-481.
6. Rostène W, De Meyts P (2021) Insulin: a 100-year-old discovery with a fascinating history. Endocr Rev 42(5): 503-527.
7. Zimmet P, Alberti K, Shaw J (2001)Global and societal implications of the diabetes epidemic. Nature 414(6865): 782-787.
8. Christesen HB, Jacobsen BB, Odili S, Buettger C, Cuesta-Munoz A, et al. (2002) The second activating glucokinase mutation (A456V) implications for glucose homeostasis and diabetes therapy. Diabetes 51(4): 1240-1246.
9. Mäkinen KK (2016) Gastrointestinal disturbances associated with the consumption of sugar alcohols with special consideration of xylitol: scientific review and instructions for dentists and other health-care professionals. Int J Dent 2016: 5967907.
10. Jacobsen SH, Olesen S, Dirksen C, Jørgensen N, Bojsen-Møller K, et al. (2012) Changes in gastrointestinal hormone responses, insulin sensitivity, and beta-cell function within 2 weeks after gastric bypass in non-diabetic subjects. Obes Surg 22(7): 1084-1096.
11. Verberne AJ, Sabetghadam A, Korim, WS (2014) Neural pathways that control the glucose counterregulatory response. Front Neurosci 8: 38.
12. Barber TM, Franks S (2021) Obesity and polycystic ovary syndrome. Clin Endocrinol 95(4): 531-541.
13. Sedighi S, Akbari SAA, Afrakhteh M, Esteki T, Majd HA, et al. (2015) Comparison of lifestyle in women with polycystic ovary syndrome and healthy women. Glob J Health Sci 7(1): 228.
14. Nasri H, Rafieian-Kopaei M (2014) Metformin: current knowledge. J Res Med Sci 19(7): 658.
15. Rojas LBA, Gomes MB (2013) Metformin: an old but still the best treatment for type 2 diabetes. Diabetol Metab Syndr 5(1): 1-15.
16. Leturque A, Brot-Laroche E, Le Gall M (2009) GLUT2 mutations, translocation, and receptor function in diet sugar managing. Am J Physiol Endocrinol Metab 296(5): E985-E992.
17. Von Ah Morano AE, Dorneles GP, Peres A, Lira FS (2020) The role of glucose homeostasis on immune function in response to exercise: The impact of low or higher energetic conditions. J Cell Physiol 235(4): 3169-3188.
18. Angelidi AM, Filippaios A, Mantzoros CS (2021) Severe insulin resistance syndromes. J Clin Invest 131(4): e142245.
19. Lebovitz HE (2001) Insulin resistance: definition and consequences. Exp Endocrinol Diabetes 109(Suppl 2): S135-S148.
20. Ormazabal V, Nair S, Elfeky O, Aguayo C, Salomon C, et al. (2018) Association between insulin resistance and the development of cardiovascular disease. Cardiovasc Diabetol 17(1): 122.
21. Ginsberg HN (2000) Insulin resistance and cardiovascular disease. J Clin Invest 106(4): 453-458.
22. Prentki M, Matschinsky FM, Madiraju SR (2013) Metabolic signaling in fuel-induced insulin secretion. Cell Metab 18(2): 162-185.
23. Lewis GF, Carpentier AC, Pereira S, Hahn M, Giacca A (2021) Direct and indirect control of hepatic glucose production by insulin. Cell Metab 33(4): 709-720.

DOI: 10.19080/OAJGGM.2024.08.555730

How to cite this article: Luqman H, Hussnain A, Arooj A, Kashif N, Umer A, et al. A Comprehensive Review on Insulin Resistance. OAJ Gerontol & Geriatric Med. 2024; 8(1): 555730. DOI: 10.19080/OAJGGM.2024.08.555730