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Reprogramming of GLP-1 Response at Prediabetes for the Prevention of Type 2 Diabetes: The Role of Albumin and GLP-1 Receptor Agonists

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Abbreviations: IGT: Impaired Glucose Tolerance; GIP: Glucose-Dependent Insulinotropic Polypeptide.

Letter to Editor

Prediabetes, a condition characterized by elevated blood sugar levels, poses a significant health risk as it increases the likelihood of developing type 2 diabetes, heart disease, and stroke. Despite its prevalence, early detection and intervention can prevent its progression [1]. Global Prevalence of Impaired Glucose Tolerance (IGT); In 2017, the global prevalence of IGT was estimated at 7.3%, affecting 352.1 million individuals [2]. Projections for 2045 indicate an alarming increase to 8.3%, equivalent to 587 million individuals. Prediabetes affects various age groups, with no significant gender difference, and nearly half of those with IGT are under 50 years old. According to the CDC, approximately 38.0% of the adult US population, totaling 97.6 million individuals, has prediabetes. Among those aged 65 years or older, 48.8% (27.2 million) are affected [2].

Early diagnosis is crucial for effective prevention strategies. Prediabetes as a Reversible Metabolic Disorder; Contrary to common belief, prediabetes is not exclusive to type 1 diabetes but is a chronic and reversible metabolic disorder [3]. It is characterized by elevated blood glucose levels, surpassing the normal threshold but not reaching the diagnostic criteria for diabetes. Lower albumin levels, irrespective of fat percentage, are associated with inflammatory markers in adipose tissue [4]. This suggests a link between albumin, an immune environment, and the risk of type 2 diabetes. Reduced albumin levels predict incident type 2 diabetes and prediabetes, indicating a potential role in their pathophysiology. The GLP-1 response to oral glucose is impaired by up to 25% in individuals with prediabetes and type 2 diabetes, particularly in women [5]. Factors such as greater insulin sensitivity, β-cell function, older age, and lower adiposity are linked to higher GLP-1 responses. This impairment may have implications for early diabetes prevention. Incretin-based therapies, including GLP-1 receptor agonists and dipeptidyl peptidase-4 inhibitors, show promise in improving β-cell function [6,7].

These therapies may contribute to stabilizing or reversing β-cell loss, providing a potential avenue for preventing the development of type 2 diabetes [8]. The incretin effect, characterized by a significant increase in plasma insulin response following glucose ingestion, is attributed to GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) [9]. GLP-1 plays a crucial role in glucose homeostasis by reducing glucagon secretion, slowing stomach emptying, and controlling body weight [10]. Preclinical studies demonstrate improvements in β-cell mass and function with GLP-1 receptor agonists. Long-term studies are needed to determine if these agonists can halt or delay the transition to type 2 diabetes [11,12] Ongoing research explores the optimal duration of GLP-1 receptor agonist therapy for sustained β-cell improvements [13].

According ADA 2024 guidelines diagnosis more people with the 2 hours plasma glucose compared with Fasting

blood glucose and A1C cut points so the postprandial is very important as target therapy to prevent prediabetes so giving Glp1 RA agonists can target postprandial plasma glucose so can prevent any elevation [14].

Conclusion

Understanding the intricate interplay between GLP-1, albumin, and prediabetes provides valuable insights for developing targeted interventions. Reprogramming GLP-1 response at the prediabetes stage, coupled with the exploration of albumin's role, holds promise in preventing the onset of type 2 diabetes.

Statements and Declarations

The authors declare that there are no conflicts of interest.

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