Commentary

Open Access

Iranian Journal of Diabetes andObesity (IJDO)

Obesity Epidemic is an Introduction of Type 2 Diabetes Mellitus and Alzheimer's Epidemic

Roya Rafaiee[1], Seyedeh Maryam Mousavi Bourkheili[2], Raheleh Rafaiee[3]*

[1]MD, Department of Internal Medicine, School of Medicine, Zahedan University of Medical Sciences, Zahedan, Iran.

[2]MSc, School of Advanced Technologies in Medicine, Mazandaran University of Medical Sciences, Sari, Iran.

[3]MD, Ph.D., Psychiatry and Behavioral Sciences Research Center, Addiction Institute, Mazandaran University of Medical Sciences, Sari, Mazandaran, Iran.

Abstract

The global rise of obesity has increased the prevalence of type 2 diabetes mellitus (T2DM) and the risk of Alzheimer's disease (AD). The obesity, a key risk factor of T2DM, leads to insulin resistance, which contributes to cognitive decline and potentially AD through shared metabolic pathways. This Commentary highlights the connection between obesity, T2DM, and AD, suggesting that lifestyle changes like weight loss and discontinuation of sugar consumption may help to prevent these conditions. Comprehensive public health strategies focusing on diet, exercise, and education are essential to reduce the growing health crisis and improve metabolic and cognitive health globally.

Keywords: Obesity, Type 2 diabetes mellitus, Type 3 diabetes, Alzheimer's disease, Epidemic

QR Code:

Citation: Rafaiee R, Mousavi Bourkheili S M, Rafaiee R. Obesity Epidemic is an Introduction of Type 2 Diabetes Mellitus and Alzheimer's Epidemic. IJDO 2025; 17 (2) :76-78

URL: http://ijdo.ssu.ac.ir/article-1-949-en.html

10.18502/ijdo.v17i2.18845

Article info:

Received: 3 December 2024

Accepted: 28 April 2025

Published in May 2025

Corresponding Author:

Raheleh Rafaiee, Psychiatry and Behavioral Sciences Research Center, Addiction Institute, Mazandaran University of Medical Sciences, Sari, Mazandaran, Iran.

Tell: (98) 113 328 5659

Email: r.rafaie@mazums.ac.ir

Orcid ID: 0000-0003-2796-5868

Introduction

The global obesity epidemic is an urgent public health problem affecting populations in both developed and developing countries. The prevalence of obesity has risen dramatically, with major implications for health and economic systems worldwide. This situation is confirmed by key findings from recent research that highlight the current state of the obesity epidemic. In 2008, 34% of adults worldwide were classified as overweight or obese (1,2). Among children under 5 years old, more than 40 million are overweight, with significant regional differences. For example, the obesity rate in America and Europe is over 20%. The prevalence of obesity among adolescents has also risen sharply, with doubling rates in some regions (3). Obesity is associated with serious health risks, including type 2 diabetes mellitus (T2DM), cardiovascular disease, and certain cancers (4).

The T2DM epidemic is a major global health crisis. Currently, more than 150 million people worldwide are affected by T2DM, and estimates suggested which rises to more than 300 million by 2025. The epidemic is particularly pronounced in developing countries, where lifestyle changes. Key risk factors include obesity, physical inactivity, and an aging population, which are exacerbating the epidemic (5).

The relationship between obesity and T2DM is well documented and complex. The obesity is considered as an important modifiable risk factor of T2DM, with several studies emphasizing the significant correlation between body weight and insulin resistance. The obesity leads to increased insulin resistance, which requires higher insulin levels to maintain glucose tolerance. The duration of obesity and weight fluctuations are key predictors of T2DM risk, with long-term obesity significantly increase the likelihood of developing T2DM (6). A cohort study found that various measures of obesity, including waist circumference and body mass index (BMI), were strongly associated with the occurrence of T2DM, with hazard ratios indicating a six-fold increase in risk for individuals in the highest quartile of waist-to-height ratio (7). The prevalence of obesity has increased significantly worldwide and correlates with rising rates of T2DM, particularly in urban populations with sedentary lifestyle. Factors such as diet, physical activity, and genetic tendency, complicate the link between obesity and T2DM.

T2DM and Alzheimer's disease (AD), both diseases share common pathophysiological mechanisms, including insulin resistance and metabolic dysfunction, which may contribute to cognitive decline (8). Understanding this overlap is crucial for the development of therapeutic strategies that address diabetes and AD.

T2DM is associated with an increased risk of developing AD, possibly due to its negative effects on cerebral vasculature and metabolic processes. Cognitive deficits in diabetic patients, especially the elderly, manifest as learning and memory impairments that can exacerbate neurodegenerative processes such as AD. Recent studies suggest that diabetes drugs could be repurposed to alter the pathophysiology of AD (9). Direct administration of insulin to the brain has shown promise for improving cognitive function, suggesting a potential therapeutic role in the treatment of AD (10). Insulin resistance in neurons is a major contributor to the production of beta-amyloid protein, a key factor in the pathology of AD. This process involves several molecular mechanisms that interfere with the normal processing of the amyloid precursor protein (APP) and insulin resistance signaling (9). Insulin promotes the metabolism of APP via a phosphatidylinositol 3-kinase (PI3-K) signaling pathway, thereby enhancing the secretion of soluble APP. In neurons, insulin signaling is critical for regulating the balance between beta-secretase and alpha-secretase

pathways that determine the production of beta-amyloid variants. Beta-amyloid peptides can inhibit insulin receptor signaling, leading to impaired insulin action and further exacerbating insulin resistance. This mutual interference suggests that elevated beta-amyloid levels may interfere with the beneficial effects of insulin on APP processing, creating a vicious cycle that promotes neurodegeneration (9).

In conclusion, to prevent AD, lifestyle changes such as a sugar-free diet, lowering insulin levels, and losing weight are beneficial. Weight loss in overweight and obese individuals has been shown to improve cognitive function and reduce the risk of AD. Excessive sugar consumption has also been linked to obesity, cognitive decline, and AD, as it can trigger chronic inflammation and alter the gut microbiome. Therefore, this cycle must be stopped somewhere so that the progression of this epidemic does not become a pandemic. Public health emphasizing the reduction of obesity through dietary changes, increased physical activity, and education are crucial in curbing the epidemic. Collaborative efforts across health sectors, policymakers, and communities will be essential in effectively reducing the burden of T2DM and AD. Public education, schools, and media play a critical role in promoting healthier lifestyles by reducing sugar consumption and supporting weight loss.

Acknowledgments

The authors wish to thank Mazandaran University of Medical Sciences.

Funding

None.

Conflict of Interest

The authors declare no conflict of interest.

Authors' contributions

All authors had an equal role in the design, work, manuscript writing, editing and approved the final manuscript.

References

1. Finucane MM, Stevens GA, Cowan MJ, Danaei G, Lin JK, Paciorek CJ, et al. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9· 1 million participants. The lancet. 2011;377(9765):557-67.
2. Aaseth J, Ellefsen S, Alehagen U, Sundfør TM, Alexander J. Diets and drugs for weight loss and health in obesity–An update. Biomedicine & Pharmacotherapy. 2021;140:111789.
3. Mohajan D, Mohajan HK. Obesity and its related diseases: a new escalating alarming in global health. Journal of Innovations in Medical Research. 2023;2(3):12-23.
4. Scully T, Ettela A, LeRoith D, Gallagher EJ. Obesity, type 2 diabetes, and cancer risk. Frontiers in oncology. 2021;10:615375.
5. Chandrasekaran P, Weiskirchen R. The role of obesity in type 2 diabetes mellitus—An overview. International journal of molecular sciences. 2024;25(3):1882.
6. Parekh J, Shende H, Kumar A, Tuli HS, Kaur G. Assessing the Clinical Correlation between Alzheimer's disease and Type-2 Diabetes Mellitus: Current Strategies and Emerging Perspectives. Current Pharmacology Reports. 2024;10(2):145-58.
7. Da Silva M, Fritz J, Mboya IB, Sun M, Wahlström J, van Guelpen B, et al. Cohort profile: the Obesity and Disease Development Sweden (ODDS) study, a pooled cohort. BMJ open. 2024;14(7):e084836.
8. Bednarz K, Siuda J. Alzheimer's disease and type 2 diabetes mellitus: similarities in pathomechanisms lead to therapeutic opportunities. Neurologia i Neurochirurgia Polska. 2021;55(5):418-28.
9. Sędzikowska A, Szablewski L. Insulin and insulin resistance in Alzheimer's disease. International journal of molecular sciences. 2021;22(18):9987.
10. Tyagi A, Pugazhenthi S. Targeting insulin resistance to treat cognitive dysfunction. Molecular neurobiology. 2021;58(6):2672-91.