Journal of Nutrition and Health Sciences
Volume 13 | Issue 1
2393-9060

Review Article Open Access

Obesity and the Risk of Cognitive Decline and Dementia: Implications and Prevention Strategies

Auda Fares*

Senior consultant in Geriatric Medicine, Marin Hospital, Wesel, Germany

***Corresponding Author**: Auda Fares, Senior consultant in Geriatric Medicine, Marin Hospital, Wesel, Pastor-Janßen-Straße 2-38, 46483 Wesel, Germany, Tel: +4917625529330, Email: audafares@yahoo.com

Citation: Auda Fares (2026) Obesity and the Risk of Cognitive Decline and Dementia: Implications and Prevention Strategies. J Nutr Health Sci 13(1): 104

Received Date: February 04, 2026 **Accepted Date:** February 23, 2026 **Published Date:** February 26, 2026

Abstract

The global rise in obesity presents a significant public health challenge, with implications extending beyond metabolic disorders to include an increased risk of cognitive decline and dementia. Understanding the complex relationship between obesity and dementia is crucial for developing effective strategies to mitigate these risks. This literature review delves into the existing evidence base, exploring potential mechanisms linking obesity to dementia, examining possible interventions, and discussing their implications for preventing or delaying dementia onset in at-risk individuals.

Keyword: Dementia; Obesity; Overweight; Alzheimer's Disease; Cognition; Prevention

Introduction

The global rise in obesity, driven by factors such as lifestyle, stress, nutrition, genetics, and physical inactivity, is a growing public health concern with far-reaching consequences. The modern environment, characterized by readily available calorie--dense foods and increasingly sedentary lifestyles, contributes significantly to this escalating epidemic. Beyond its well-established links to metabolic disorders such as type 2 diabetes, cardiovascular disease, and certain cancers, obesity has been increasingly associated with cognitive decline, impaired synaptic plasticity, and reduced brain volume, significantly elevating the risk of developing Alzheimer's disease (AD) and other dementias. This association is particularly concerning given the progressive aging of the global population, making dementia a major public health challenge. Given the concerning statistic that an estimated 39% of adults were overweight in 2016 [1], and recognizing obesity as a modifiable risk factor, targeted interventions become crucial for dementia prevention. This literature review aims to explore the evidence base, potential mechanisms, possible interventions, and implications for preventing dementia in the context of the ongoing obesity epidemic. Specifically, we will examine the epidemiological evidence linking obesity to dementia risk, delve into the complex biological pathways that may mediate this relationship, and explore promising strategies for prevention and intervention, ultimately aiming to inform public health policy and clinical practice.

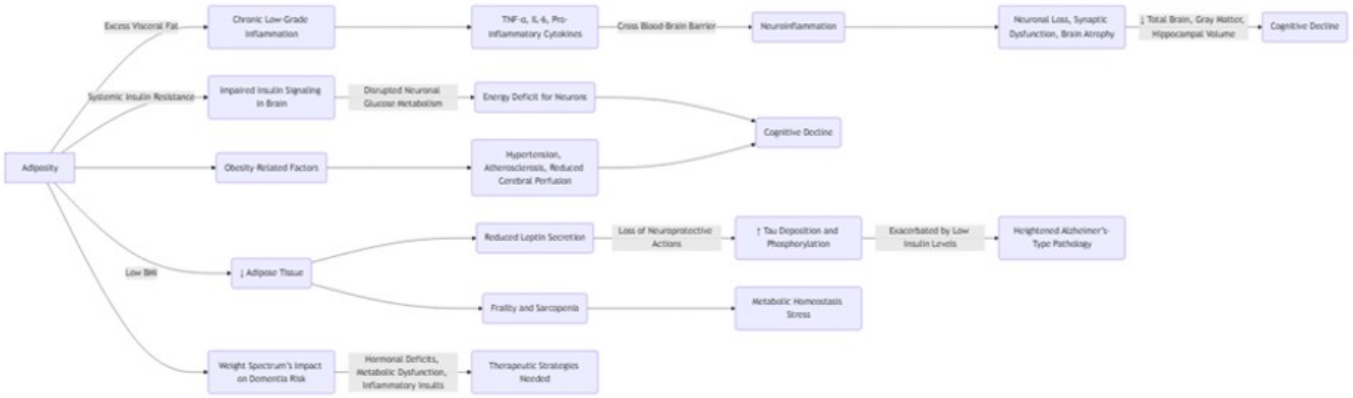

Figure 1: Potential Mechanisms

Material and Methods

This narrative review conducted a thorough literature search to identify practical strategies for reducing cognitive decline and dementia risk in individuals with obesity. English-language studies were sourced from major databases, including Medline, the Cochrane Library, and Google Scholar, without publication date restrictions to maximize inclusivity. The search strategy combined Medical Subject Headings (MeSH) terms such as "Dementia," "Obesity," "Alzheimer's disease," "Cognition," and "Prevention" with relevant free-text keywords to capture a broad spectrum of literature. Articles were selected if they specifically addressed mechanisms of cognitive impairment, associated risk factors, or preventive interventions within the obese population. The extracted evidence was then synthesized thematically to outline clinically relevant approaches and potential interventions aimed at mitigating cognitive deterioration in this high-risk group.

Epidemiological Evidence

There is increasing evidence to suggest that underweight BMI, overweight BMI and obese BMI predisposes individuals to developing cognitive impairment and dementia. Risks appeared to be highest for underweight and obese BMI. In recently umbrella review and meta-analysis found a significant positive association between risk of AD dementia and body weight (BMI ≥30 and BMI 25–29 at mid-life; BMI <18.5 [2]. These consist with results of A meta-analysis that included 19 longitudinal studies and 589 649 participants aged 35 to 65 years , followed up for up to 42 years found that the obesity (BMI ≥30) but not being over-

weight (BMI 25–30) was associated with late-life dementia [3]. Another meta-analysis that included 15 prospective studies and 15 435 participants, Follow-ups ranged from 3.2 to 36.0 years found that underweight BMI, overweight BMI and obese BMI were all associated with increased risk of dementia compared with normal BMI [4]. In one more published meta-analysis showed that dementia risk increased with obesity and underweight [5]. Additionally, Results from the Whitehall II study of more than 10,000 men and women with repeat BMI assessments for more than 28 years show that obesity (BMI ≥30 kg/m2) at age 50 years is a risk factor for dementia [6]. In contrast, meta-analysis that included 20 studies revealed that the Weight loss was associated with a significant improvement in improvements in performance across various cognitive domains [7].

Potential Mechanisms

The complex relationship between adiposity and dementia risk involves several potential mechanisms, with research highlighting both the detrimental effects of excess weight and the surprising vulnerabilities associated with lower body mass. While comorbidities like hypertension, cardiovascular disease, and diabetes are often implicated as mediating factors in the link between obesity and dementia, a more nuanced picture is emerging [8]. The metabolic dysfunction associated with obesity often leads to insulin resistance, which not only contributes to type 2 diabetes but also impairs insulin signaling in the brain. This can disrupt glucose metabolism, a critical energy source for neurons, ultimately impacting cognitive function.

Intriguingly, lower BMI has been associated with increased brain tau deposition, a key hallmark of Alzheimer's disease [9-11]. This may be driven by decreased levels of leptin, a hormone primarily secreted by fat tissue. Laboratory studies suggest leptin has a protective role, reducing the accumulation of amyloid beta and tau proteins. Moreover, lower BMI often corresponds with decreased insulin levels, which can potentially exacerbate tau phosphorylation in the brain [12,13]. This counterintuitive finding is supported by studies showing higher tau levels and phosphorylated tau in individuals with lower BMI compared to those with obesity [14]. Leptin's role in regulating appetite and energy expenditure also suggests a potential link to overall metabolic health and brain function. Conversely, obesity itself is strongly linked to brain atrophy, demonstrating smaller total brain volume, gray matter, and hippocampal volume [15]. This structural damage is thought to be mediated by the chronic low-grade inflammation associated with obesity. Adipose tissue, particularly visceral fat, is a source of pro-inflammatory cytokines such as TNF-α and IL-6, which can cross the blood-brain barrier and contribute to neuroinflammation. This inflammatory milieu can damage neurons, impair synaptic plasticity, and ultimately contribute to cognitive decline. Furthermore, obesity-related vascular dysfunction can reduce cerebral blood flow, further exacerbating brain atrophy. In essence, the connection between adiposity and dementia is not simply about having too much or too little weight; rather, it seems to be a delicately balanced network of hormonal, metabolic, and inflammatory processes that can significantly impact brain health [16,17]. Future research is needed to further elucidate the relative contributions of these various pathways and to identify potential therapeutic targets.

Implications and Prevention Strategies

1. Integrated Metabolic and Cognitive Assessment: At the initial geriatric assessment or annual wellness visit for an elderly patient with obesity (BMI ≥30 kg/m^2), clinicians should proactively screen for cognitive impairment alongside metabolic dysfunction. This should not be a siloed process [18].

- **Cognitive Screening:** Utilize brief, validated tools such as the Montreal Cognitive Assessment (MoCA) or the Mini-Cog. A MoCA score below 26 (adjusted for education) warrants further neuropsychological evaluation. Screening should be repeated annually, or sooner if patient or caregiver reports changes [18].

- **Metabolic and Vascular Risk Stratification:** Beyond BMI, measure waist circumference (risk threshold: >102 cm for men, >88 cm for women) as a marker of visceral adiposity. Mandatory laboratory assessments should include fasting

glucose/HbA1c, lipid panel (focus on HDL and triglycerides), and blood pressure. The presence of metabolic syndrome (any three of: abdominal obesity, hypertriglyceridemia, low HDL, hypertension, hyperglycaemia) significantly elevates dementia risk [19].

- **Sarcopenic Obesity Screening:** This is a critical, high-risk phenotype in the elderly. Clinicians must screen for low muscle mass and function using simple measures: gait speed (<0.8 m/s suggests slow speed) and grip strength (using a dynamometer; thresholds vary by sex and BMI). Low muscle mass combined with high adiposity dramatically increases risks for disability, frailty, and cognitive decline. Bioelectrical impedance analysis (BIA) or, where available, DXA scans can provide more precise body composition data [20].

2. Dietary Pattern and Lifestyle History: Employ a structured dietary recall or screen (e.g., a modified Mediterranean Diet Adherence Screener) to evaluate adherence to protective patterns (Mediterranean, MIND, DASH) [18]. Gauge physical activity levels against the guideline of 150 min/week moderate exercise. Inquire about social determinants of health impacting food security and safe mobility [18].

Practical Preventive Approaches for Clinical Implementation

1. Timing of Intervention: Intervention should begin immediately upon the identification of obesity plus any one additional risk factor (metabolic syndrome, prediabetes, hypertension, or subjective cognitive complaint). The diagnosis of obesity in an elderly patient is itself a catalyst for a comprehensive risk-reduction discussion. There is no "too late" to initiate neuroprotective lifestyle changes; benefits are observed even in late-life adopters, particularly regarding functional preservation and slowing decline

2. Nutritional Prescription:

- **Diet Pattern:** Strongly recommend a **Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) diet**. This hybrid diet specifically emphasizes brain-healthy foods: weekly servings of berries, leafy greens, nuts, beans, and whole grains, and fish (particularly fatty fish for omega-3s), while limiting red meats, butter/margarine, and pastries/sweets [21].

3. Exercise Prescription:

- **Aerobics:** 150-300 minutes/week of moderate-intensity (e.g., brisk walking, water aerobics) or 75-150 minutes of vigorous-intensity exercise. Emphasize consistency over intensity [22].

- **Resistance Training:** Non-negotiable. 2-3 days/week, targeting all major muscle groups. Use body weight, resistance bands, or light weights. The goal is to maintain or increase strength to support daily function.

- **Balance Flexibility:** Daily or at least 2-3 times/week. Tai Chi is highly recommended for its dual benefits for balance and cognitive engagement [23].

- **Practical Integration:** Partner with physical therapists for initial assessment and program design, especially for frail patients. Promote "exercise snacks" (multiple 5–10-minute walks) for those with low endurance. Strongly encourage group-based programs for social engagement, which itself is neuroprotective.

4. Cognitive and Social Engagement:

- Screen for and address social isolation and depression, which are potent risk factors for both obesity and dementia [18].
- Recommend structured cognitive training programs or lifelong learning activities (e.g., digital literacy classes, book clubs) as part of the dementia prevention plan.

5. Sleep Hygiene and Stress Management:

- Screen for sleep apnea (high prevalence in obese elderly) and treating sleep apnea improves metabolic health and cognition. Effective CPAP therapy improves metabolic and cognitive outcomes [24].
- Cognitive Engagement: Prescribe non-digital cognitive activities: social engagement (senior centers, volunteering), complex card games, learning a new skill, or music engagement. Link these to existing community hubs.

6. Pharmacologic and Surgical Considerations:

- **Pharmacotherapy:** GLP-1 receptor agonists (e.g., semaglutide, liraglutide) show promise for weight loss and may have direct neuroprotective effects. Their use in the elderly requires extreme caution, starting at low doses, with vigilant monitoring for gastrointestinal side effects (risk of dehydration, malnutrition) and cost/access barriers [25,26].
- **Polypharmacy Review:** Conduct a comprehensive medication review. Identify and deprescribe medications with anticholinergic burden, sedative effects, or those that promote weight gain where alternatives exist. This is a direct intervention to reduce cognitive fog and metabolic harm.
- **Bariatric Surgery:** Consideration in elderly patients (>65-70) is exceptionally rare and requires a rigorous, multidisciplinary geriatric-specific evaluation. The perioperative risks (sarcopenia, cognitive fluctuation, nutritional deficiencies) often outweigh potential long-term benefits for prevention of dementia. It is generally contraindicated in frail patients [18].

7. Interprofessional Care Model:

- Establish a referral network: Registered Dietitian with geriatric expertise, Physical Therapist/Occupational Therapist, Clinical Psychologist (for behavioral change/cognitive health), and Social Worker (to address food insecurity, transportation).
- Utilize allied health professionals (medical assistants, nurses) to conduct initial screening, provide education, and support between physician visits.

Addressing Implementation Challenges at the Clinical Level

To overcome clinician-level barriers (time, training):

- **Integrate Screening:** Combine cognitive (MoCA), frailty, and nutritional (MNA) screens into the annual wellness visit workflow. Use nursing staff for initial data collection.
- **Brief Intervention Training:** Train clinicians in the **"5 A's" model** (Ask, Advise, Assess, Assist, and Arrange) specifically tailored for obesity and brain health. A 1–2-minute focused conversation on the obesity-dementia link and

one specific, achievable goal (e.g., "This week, can you add one 10-minute walk after dinner?") is more effective than no intervention.

- **Standardized Protocols:** Develop clinic-specific, evidence-based care pathways or algorithms that dictate when to screen, refer, and follow up, reducing decision fatigue.

- **Leverage Technology:** Recommend validated health apps for tracking diet, steps, and cognitive games. Utilize telehealth for dietitian consultations and supervised exercise sessions to improve access.

By embracing a geriatric-sensitive framework, clinicians can transform the office visit from a narrow, weight-centric encounter into a proactive, “brain-healthy” prescription that directly targets the metabolic pathways linking excess adiposity to neurodegeneration in older adults. In this reframed model, every conversation becomes a strategic opportunity to lower dementia risk through personalized blends of nutrition counseling, cognitively stimulating physical activity, sleep hygiene, and stress-management rather than merely recording a number on the scale. Turning this evidence-based vision into routine care requires a solid methodological scaffold: systematic clinician education that demystifies neuro-metabolic links, standardized pathways that embed brain-health metrics into electronic health records, and policy incentives that reimburse multidisciplinary, preventive interventions. Crucially, care teams must extend beyond physicians to include dietitians, physical therapists, social workers, and community services liaisons who can address non-medical barriers such as food insecurity, transportation gaps, and social isolation. When these structural supports converge, the clinical encounter evolves into a high-impact platform that simultaneously preserves cognitive vitality and manages weight, bridging the gap between scientific insight and tangible health outcomes for the elderly.

Future Directions and Research Priorities

While continued research to fully understand the specific mechanisms by which obesity contributes to dementia is vital, equally important is investing in education and training to improve the implementation of obesity science into clinical practice. Research should focus on:

- **Longitudinal studies:** To determine the temporal relationship between obesity and dementia and to identify critical periods for intervention.

- **Mechanistic studies:** To elucidate the precise mechanisms by which obesity contributes to neurodegeneration.

- **Intervention studies:** To evaluate the effectiveness of different obesity prevention and treatment strategies in reducing dementia risk.

Conclusion

The evidence strongly suggests a complex and multifaceted relationship between obesity and dementia risk. While the exact mechanisms remain to be fully elucidated, the interplay of metabolic dysfunction, inflammation, hormonal imbalances, and structural brain changes appears to play a crucial role. Given the escalating global prevalence of obesity and the devastating impact of dementia, targeted prevention strategies are urgently needed. These strategies should focus on promoting healthy lifestyles from a young age, with an emphasis on balanced nutrition, regular physical activity, and weight management. Furthermore, it is essential to improve the implementation of existing evidence-based interventions in clinical practice and to continue investing in research to better understand the underlying mechanisms and to identify novel therapeutic targets. By addressing

the obesity epidemic, we can potentially mitigate the risk of dementia and improve the overall health and well-being of future generations.

Conflict of Interest

The author declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethical Consideration

Not applicable

References

1. NCD Risk Factor Collaboration (NCD-RisC) (2017) Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. Lancet, 390: 2627–42.

2. Jones A, Ali MU, Kenny M, Mayhew A, Mokashi V, He H, et al. (2024) Potentially Modifiable Risk Factors for Dementia and Mild Cognitive Impairment: An Umbrella Review and Meta-Analysis. Dement Geriatr Cogn Disord. 53: 91-106.

3. Albanese E, Launer LJ, Egger M, Prince MJ, Giannakopoulos P, Wolters FJ, et al. (2017) Body mass index in midlife and dementia: Systematic review and meta-regression analysis of 589,649 men and women followed in longitudinal studies. Alzheimers Dement (Amst). 8: 165-78.

4. Anstey KJ, Cherbuin N, Budge M, Young J (2011) Body mass index in midlife and late-life as a risk factor for dementia: a meta-analysis of prospective studies. Obes Rev. 12: e426-37.

5. Beydoun M.A., Beydoun H.A., Wang Y (2008) Obesity and central obesity as risk factors for incident dementia and its subtypes: a systematic review and meta-analysis. Obes Rev. 9: 204–18.

6. Singh-Manoux A, Dugravot A, Shipley M, Brunner EJ, Elbaz A, Sabia S, et al. (2018) Obesity trajectories and risk of dementia: 28 years of follow-up in the Whitehall II Study. Alzheimers Dement. 14: 178-86.

7. Veronese N, Facchini S, Stubbs B, Luchini C, Solmi M, Manzato E, et al. (2017) Weight loss is associated with improvements in cognitive function among overweight and obese people: A systematic review and meta-analysis. Neurosci Biobehav Rev. 72: 87-94.

8. Nordestgaard, L.T., Christoffersen, M., Frikke-Schmidt, R (2022) Shared Risk Factors between Dementia and Atherosclerotic Cardiovascular Disease. Int. J. Mol. Sci. 23: 9777.

9. Greco SJ, Sarkar S, Johnston JM, Zhu X, Su B, Casadesus G, et al. (2008) Leptin reduces Alzheimer's disease-related tau phosphorylation in neuronal cells. Biochem Biophys Res Commun. 376: 536–41.

10. Marwarha G, Dasari B, Prasanthi JR, Schommer J, Ghribi O (2010) Leptin reduces the accumulation of Abeta and phosphorylated tau induced by 27-hydroxycholesterol in rabbit organotypic slices. J Alzheimers Dis. 19: 1007–19.

11. Lee S, Byun MS, Yi D, Kim MJ, Jung JH, Kong N, et al. (2023) Body mass index and two-year change of in vivo Alzheimer's disease pathologies in cognitively normal older adults. Alz Res Therapy, 15: 108

12. Craft S, Watson GS (2004) Insulin and neurodegenerative disease: shared and specific mechanisms. Lancet Neurol. 3: 169–78.

13. Hong M, Lee VMY (1997) Insulin and insulin-like growth factor-1 regulate tau phosphorylation in cultured human neurons. J Biol Chem. 272: 19547–53.

14. Zhang XX, Ma YH, Hu HY, Ma LZ, Tan L, Yu JT (2022) Late-Life Obesity Associated with Tau Pathology in Cognitively Normal Individuals: The CABLE Study. J Alzheimers Dis. 85: 877-87.

15. Han YP, Tang X, Han M, Yang J, Cardoso MA, Zhou J, Simó R (2021) Relationship between obesity and structural brain ab-

normality: Accumulated evidence from observational studies. Ageing Res Rev. 71: 101445.

16. Parimisetty A, Dorsemans AC, Awada R, Ravanan P, Diotel N, Lefebvre d'Hellencourt C (2016) Secret talk between adipose tissue and central nervous system via secreted factors-an emerging frontier in the neurodegenerative research. J Neuroinflammation. 13: 67.

17. Pou KM, Massaro JM, Hoffmann U, Vasan RS, Maurovich-Horvat P, Larson MG, et al. (2007) Visceral and subcutaneous adipose tissue volumes are cross-sectionally related to markers of inflammation and oxidative stress: the Framingham Heart Study. Circulation. 116: 1234-41.

18. Dewar S (2025) A geriatrician's approach to managing the complex older adult with obesity. Obesity (Silver Spring). 33: 41-56.

19. Ross R, Neeland IJ, Yamashita S, Shai I, Seidell J, Magni P, et al. (2020) Waist circumference as a vital sign in clinical practice: a Consensus Statement from the IAS and ICCR Working Group on Visceral Obesity. Nat Rev Endocrinol. 16: 177-89.

20. Voulgaridou G, Tyrovolas S, Detopoulou P, Tsoumana D, Drakaki M, Apostolou T, et al. (2024) Diagnostic Criteria and Measurement Techniques of Sarcopenia: A Critical Evaluation of the Up-to-Date Evidence. Nutrients. 16: 436.

21. Liu X, Morris MC, Dhana K, Ventrelle J, Johnson K, Bishop L, et al. (2021) Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) study: Rationale, design and baseline characteristics of a randomized control trial of the MIND diet on cognitive decline. Contemp Clin Trials. 102: 106270.

22. WHO Guidelines on Physical Activity and Sedentary Behaviour. Geneva: World Health Organization; 2020. RECOMMENDATIONS. Available from: https://www.ncbi.nlm.nih.gov/books/NBK566046/

23. Zhai W, Zhai Q, Wei L, Wen X () Can The Frequency and Intensity of Tai Chi Influence the Magnitude of Adaptations in Health Markers and Physical Fitness in Older Adults? A Randomized Controlled Study. J Sports Sci Med. 24: 871-80.

24. Benkirane O, Mairesse O, Peigneux P (2024) Impact of CPAP Therapy on Cognition and Fatigue in Patients with Moderate to Severe Sleep Apnea: A Longitudinal Observational Study. Clocks Sleep. 6: 789-816.

25. Athauda D, Greig NH, Meissner WG, Foltynie T, Gandhi S (2026) The promise of GLP-1 receptor agonists for neurodegenerative diseases. J Clin Invest. 136: e194745.

26. Wilbon SS, Kolonin MG (2023) GLP1 Receptor Agonists-Effects beyond Obesity and Diabetes. Cells. 13: 65.