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Obesity and Alzheimer's Disease: a Systematic Review

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Abstract: Background: Obesity has emerged as a major global public health concern and has been increasingly implicated as a modifiable risk factor for neurodegenerative diseases, including Alzheimer's disease (AD). While numerous epidemiological and experimental studies have examined the association between excess adiposity and cognitive decline, findings remain heterogeneous, particularly with respect to the timing of obesity exposure across the lifespan.

Objective: This systematic review aimed to synthesize current evidence on the relationship between obesity and Alzheimer's disease, focusing on epidemiological associations, underlying biological mechanisms, and the influence of life-course timing on disease risk.

Methods: A structured literature search was conducted in major electronic databases, including Google Scholar, PubMed, and Scopus, to identify peer-reviewed articles examining the association between obesity and Alzheimer's disease. Observational cohort studies, case-control studies, neuroimaging investigations, and mechanistic experimental studies published in English were considered. Eligible studies were screened according to predefined inclusion and exclusion criteria. Data were extracted and qualitatively synthesized to evaluate patterns of association, proposed mechanisms, and methodological considerations.

Results: The evidence consistently demonstrates that midlife obesity is associated with an increased risk of later-life Alzheimer's disease and cognitive decline. Central adiposity and metabolic syndrome components showed stronger associations with AD risk than body mass index (BMI) alone. Mechanistic studies suggest that obesity contributes to AD pathogenesis through chronic systemic inflammation, insulin resistance, oxidative stress, cerebrovascular dysfunction, and impaired brain glucose metabolism. Neuroimaging findings indicate structural and functional alterations in obesity that overlap with AD-vulnerable brain regions. In contrast, late-life obesity demonstrated inconsistent associations, likely influenced by reverse causation and disease-related weight loss.

Conclusion: The findings support the hypothesis that obesity – particularly during midlife – acts as a significant and potentially modifiable risk factor for Alzheimer's disease. The relationship appears to be mediated by complex metabolic and inflammatory pathways. Preventive strategies targeting obesity and metabolic dysfunction across the lifespan may contribute to reducing the future burden of Alzheimer's disease. Further longitudinal and biomarker-integrated studies are required to clarify causal mechanisms and inform targeted interventions.

Keywords: obesity, Alzheimer's disease, neurodegenerative, quality of life, metabolism, inflammation

Kurzfassung: Adipositas und Alzheimer – ein systematisches Review. Hintergrund: Adipositas hat sich zu einem bedeutenden globalen Problem der öffentlichen Gesundheit entwickelt und wird zunehmend als beeinflussbarer Risikofaktor für neurodegenerative Erkrankungen, einschließlich der Alzheimer-Krankheit (AD), diskutiert. Zwar haben zahlreiche epidemiologische und experimentelle Studien den Zusammenhang zwischen starkem Übergewicht und kognitivem Abbau untersucht, doch sind die Ergebnisse uneinheitlich – insbesondere im Hinblick auf den Zeitpunkt des Auftretens von Adipositas im Laufe des Lebens.

Zielsetzung: Diese systematische Übersichtsarbeit zielte darauf ab, die aktuelle Evidenz zum Zusammenhang zwischen Adipositas und der Alzheimer-Krankheit zusammenzuführen, wobei der Schwerpunkt auf epidemiologischen Assoziationen, zugrunde liegenden biologischen Mechanismen und dem Einfluss des Zeitpunkts im Lebensverlauf auf das Erkrankungsrisiko lag.

Methodik: Es wurde eine strukturierte Literaturrecherche in wichtigen elektronischen Datenbanken – darunter Google Scholar, PubMed und Scopus – durchgeführt, um peer-reviewte Artikel zu identifizieren, die den Zusammenhang zwischen Adipositas und der Alzheimer-Krankheit untersuchen. Berücksichtigt wurden englischsprachige Beobachtungsstudien (Kohorten- und Fall-Kontroll-Studien), Neuroimaging-Studien sowie mechanistische experimentelle Studien. Die in Frage kommenden Studien wurden anhand vorab festgelegter Ein- und Ausschlusskriterien gesichtet. Die Daten wurden extrahiert und qualitativ zusammengeführt, um Muster des Zusammenhangs, postulierte Mechanismen sowie methodische Aspekte zu bewerten.

Ergebnisse: Die vorliegenden Erkenntnisse belegen eindeutig, dass Adipositas im mittleren Lebensalter mit einem erhöhten Risiko für Alzheimer und kognitiven Abbau im späteren Leben einhergeht. Zentrale Adipositas und Komponenten des metabolischen Syndroms zeigten stärkere Zusammenhänge mit dem Alzheimer-Risiko als der Body-Mass-Index (BMI) allein. Mechanistische Studien deuten darauf hin, dass Adipositas durch chronische systemische Entzündungen, Insulinresistenz, oxidativen Stress, zerebrovaskuläre Dysfunktion und einen beeinträchtigten Glukosestoffwechsel im Gehirn zur Pathogenese von Alzheimer beiträgt. Neuroimaging-Befunde weisen auf strukturelle und funktionelle Veränderungen bei Adipositas hin, die sich mit Alzheimer-anfälligen Hirnregionen überschneiden. Im Gegensatz dazu zeigte Adipositas im höheren Lebensalter uneinheitliche Zusammenhänge, die wahrscheinlich durch umgekehrte Kausalität und krankheitsbedingten Gewichtsverlust beeinflusst sind.

Schlussfolgerung: Die Ergebnisse stützen die Hypothese, dass Adipositas – insbesondere im mittleren Lebensalter – einen signifikanten und potenziell beeinflussbaren Risikofaktor für die Alzheimer-Krankheit darstellt. Dieser Zusammenhang scheint durch komplexe Stoffwechsel- und Entzündungsprozesse vermittelt zu werden. Präventionsstrategien, die über die gesamte Lebensspanne hinweg auf Adipositas und metabolische Dysfunktionen abzielen, könnten dazu beitragen, die künftige Krankheitslast durch Alzheimer zu verringern. Weitere Längsschnittstudien unter Einbeziehung von Biomarkern sind erforderlich, um die zugrunde liegenden Kausalmechanismen zu klären und gezielte Interventionen zu ermöglichen. **J Neurol Neurochir Psychiatr 2026; 27 (4): 81–8.**

Schlüsselwörter: Adipositas, Alzheimer-Erkrankung, neurodegenerativ, Lebensqualität, Stoffwechsel, Entzündung

Introduction

Alzheimer's disease (AD) is a progressive neurodegenerative disorder and the leading cause of dementia worldwide, characterized by progressive memory deterioration, impaired executive functioning, and eventual loss of independence [1]. The global prevalence of AD has risen dramatically with aging populations, presenting profound social, economic, and healthcare challenges. Despite decades of research, disease-modifying therapies remain limited, and current pharmacological interventions predominantly offer symptomatic relief rather than halting underlying pathology. Consequently, there is an urgent

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need to identify modifiable risk factors that contribute to the onset and progression of AD to inform preventive strategies.

Obesity, defined by excessive adipose tissue accumulation often quantified through body mass index (BMI), has reached epidemic proportions globally and is a well-established risk factor for metabolic disorders including type-2 diabetes mellitus (T2DM), hypertension, and cardiovascular disease (World Health Organization, 2025). A growing body of epidemiological and mechanistic research indicates that obesity may also influence brain health and cognitive decline, implicating it in the aetiology of AD [2, 3]. Midlife obesity, in particular, has been consistently associated with an elevated risk of developing dementia later in life. Longitudinal studies demonstrate that individuals who are overweight or obese between the ages of 45 and 65 have significantly higher odds of developing Alzheimer's disease decades later compared to those with normal weight [4]. This association persists after adjustment for demographic and lifestyle factors, suggesting that obesity may contribute independently to neurodegenerative processes.

Although the epidemiological link between obesity and AD risk is increasingly accepted, the underlying biological mechanisms remain incompletely understood. Obesity is a state of chronic low-grade systemic inflammation driven by adipose tissue dysfunction, which can lead to activation of pro-inflammatory pathways and immune cell infiltration [5–7]. These inflammatory processes are thought to extend to the central nervous system, exacerbating neuroinflammation – a recognized hallmark of AD pathology [8]. Obesity-induced inflammation is associated with increased levels of cytokines such as interleukin-6 and tumor necrosis factor-alpha, which can disrupt blood-brain barrier integrity and promote microglial activation, thereby potentially accelerating amyloid-β plaque formation and tau hyperphosphorylation [9, 10]. Additionally, obesity frequently co-exists with insulin resistance and T2DM, both of which have been independently linked to AD. Insulin resistance disrupts neuronal insulin signalling pathways that are vital for synaptic plasticity and glucose metabolism in the brain, contributing to metabolic dysfunction, impaired amyloid clearance, and neuronal loss [11–13].

Despite accumulating evidence linking metabolic dysfunction to AD, important gaps remain in our understanding. The relationship between late-life obesity and AD risk is complex, with some studies suggesting that obesity in older age may confer a paradoxical protective effect, potentially due to reverse causation (i.e., preclinical AD-related weight loss) [14, 15]. Further, the relative contributions of overall adiposity versus central fat distribution, as well as the roles of genetic predispositions such as APOE-ε4 status, warrant deeper investigation. These uncertainties underscore the need for integrated research frameworks that consider life course trajectories of body composition and metabolic health in relation to neurodegeneration.

The present study aims to synthesize current evidence on the association between obesity and Alzheimer's disease and to clarify the biological mechanisms that may mediate this link. Specifically, this research will (1) evaluate the strength and consistency of epidemiological associations between obesity at different life stages and AD risk, (2) examine mechanistic pathways linking adiposity with AD pathology, and (3) identify gaps and future directions for research and intervention. By elucidating the complex interplay between metabolic health and neurodegeneration, this work seeks to inform preventative strategies targeting modifiable risk factors for AD.

Methodology

Study Design

This study was conducted as a systematic review to evaluate the association between obesity and Alzheimer's disease (AD). The methodology followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure transparency, reproducibility, and methodological rigor.

Search Strategy

A comprehensive literature search was performed across the following electronic databases:

- PubMed/MEDLINE
- Scopus
- Web of Science
- Cochrane Library

The search strategy combined Medical Subject Headings (MeSH) and free-text terms related to obesity and Alzheimer's disease. Key search terms included:

- "obesity" OR "adiposity" OR "body mass index" OR "BMI" OR "central obesity" OR "visceral fat"
- "Alzheimer's disease" OR "Alzheimer disease" OR "dementia" OR "cognitive decline"
- "insulin resistance" OR "metabolic syndrome" OR "neuroinflammation"

Boolean operators (AND/OR) were used to refine the search. The search was limited to peer-reviewed articles published in English. Reference lists of included studies and relevant reviews were manually screened to identify additional eligible studies.

Inclusion Criteria

Studies were included if they:

- Investigated the association between obesity (measured by BMI, waist circumference, waist-to-hip ratio, or imaging-based adiposity measures) and Alzheimer's disease or AD-related cognitive decline.
- Included adult human participants (≥ 18 years).
- Used observational (cohort, case-control, cross-sectional) or interventional study designs.
- Reported clinically diagnosed AD, biomarker-confirmed AD, or validated cognitive outcomes.
- Were published in peer-reviewed journals.

Exclusion Criteria

Studies were excluded if they:

- Focused exclusively on non-Alzheimer dementias without specific AD analysis.
- Did not clearly define or measure obesity.
- Were case reports, editorials, conference abstracts, or non-peer-reviewed publications.
- Involved pediatric populations.
- Were not available in English.

Study Selection Process

All retrieved records were imported into reference management software, and duplicates were removed. Two independent reviewers screened titles and abstracts for relevance. Full texts of potentially eligible studies were then assessed independently by both reviewers against the inclusion and exclusion criteria.

Disagreements were resolved through discussion or consultation with a third reviewer. A PRISMA flow diagram was used to document the selection process, including the number of records identified, screened, excluded, and included.

Data Extraction

A standardized data extraction form was developed to collect relevant information from included studies. The following variables were extracted:

- Author(s) and year of publication
- Country and study design
- Sample size and participant characteristics
- Age at obesity assessment (midlife vs. late-life)
- Measures of obesity (BMI, waist circumference, etc.)
- Outcome measures (clinical AD diagnosis, cognitive scores, biomarkers)
- Duration of follow-up (for longitudinal studies)
- Adjusted confounders
- Main findings and effect estimates

Data extraction was conducted independently by two reviewers to minimize bias.

Quality Assessment

The methodological quality and risk of bias of included studies were assessed using validated tools appropriate to study design:

- Newcastle-Ottawa Scale (NOS) for cohort and case-control studies
- Joanna Briggs Institute (JBI) checklist for cross-sectional studies
- Cochrane Risk of Bias Tool for randomized controlled trials

Studies were rated as low, moderate, or high risk of bias. Quality assessment was performed independently by two reviewers, with discrepancies resolved by consensus.

Data Synthesis

A narrative synthesis approach was employed to summarize findings across studies due to expected heterogeneity in study design, population characteristics, and outcome measures.

Where sufficient homogeneity existed (e.g., similar exposure and outcome definitions), quantitative synthesis (meta-analysis) was considered. Effect sizes such as relative risks (RR), odds ratios (OR), or hazard ratios (HR) with 95% confidence intervals (CI) were extracted or calculated.

Heterogeneity was assessed using the I2 statistic. If meta-analysis was feasible:

- A random-effects model was applied due to anticipated variability between studies.
- Subgroup analyses were conducted based on life stage (midlife vs. late-life obesity), sex, and obesity measurement method.
- Sensitivity analyses were performed to evaluate the influence of study quality.

Publication bias was assessed using funnel plots and Egger's regression test where applicable.

Results

See Table 1.

Discussion

The present systematic review synthesizes epidemiological, biomarker, and mechanistic evidence examining the relationship between obesity and Alzheimer's disease (AD). Overall, the findings demonstrate a consistent association between midlife obesity and increased AD risk, while also revealing complex, age-dependent, and biologically nuanced relationships in later life. The following discussion analyzes each key result in detail, interprets its significance, and situates it within the broader context of existing evidence.

BMI trajectories and neurodegeneration (Lee et al., 2026) [2]

Lee and colleagues provide important longitudinal evidence linking BMI changes across adulthood to AD-related neurodegeneration. Their finding that a declining BMI trajectory from midlife to late-life is associated with reduced cerebral glucose metabolism in AD-signature regions suggests that weight loss in older adults may reflect ongoing neurodegenerative processes rather than confer protection [30, 31]. Notably, the absence of significant associations between BMI and amyloid-β or tau pathology implies that obesity may exert a stronger influence on metabolic and neurodegenerative pathways than on core amyloid deposition. This distinction is significant because it suggests that obesity may contribute to AD progression through brain hypometabolism rather than directly initiating amyloid accumulation. The findings reinforce the need to interpret late-life weight loss cautiously, as it may represent prodromal AD rather than improved metabolic health [32].

Life-stage-dependent risk patterns (Qu et al., 2020; Son et al., 2025) [16, 26]

The meta-analyses by Qu et al. and Son et al. consistently demonstrate that midlife obesity increases the risk of cognitive impairment and AD, while late-life overweight or obesity may show neutral or even inverse associations – often termed the "obesity paradox." The dose-response analysis identifying increased AD risk at BMI thresholds above approximately 29–30 kg/m² in midlife strengthens the argument for a causal contribution of excess adiposity during critical metabolic years [33]. However, the apparent protective associations observed in older adults likely reflect reverse causation, survival bias, or confounding due to preclinical weight loss [34, 35]. Son et al.'s finding that visceral adiposity measures show stronger associations than BMI alone further highlights the limitations of relying solely on BMI as an exposure metric. Together, these studies underscore that timing and adiposity distribution matter, suggesting that interventions targeting midlife obesity may offer the greatest preventive potential.

Table 1: Results

BMI changes from midlife to late-life and their relationship with Alzheimer's disease pathologies

Lee S et al, KBASE Research Group (2026) [2]

Lee and colleagues conducted a longitudinal cohort study investigating associations between BMI trajectories from midlife to late-life and in vivo Alzheimer's disease biomarkers measured via PET imaging. The results demonstrated that a decreasing BMI trajectory over time was significantly associated with reduced cerebral glucose metabolism in AD-signature regions, a marker of neurodegeneration, whereas higher midlife BMI correlated with lower cerebral metabolism. No significant associations were found between BMI patterns and amyloid-β or tau pathology. These findings suggest that weight loss across the adult lifespan mirrors AD-related neurodegeneration, underscoring the importance of long-term weight management in elderly populations.

Association of body mass index with risk of cognitive impairment and dementia: a systematic review and meta-analysis of prospective studies

Qu Y et al (2020) [16]

This large meta-analysis pooled data from numerous prospective studies (≈ 20,083 records) to examine the associations between BMI categories and dementia outcomes, including Alzheimer's disease. Results indicated that midlife obesity and underweight status were linked to elevated risk of cognitive impairment and dementia, whereas late-life overweight and obesity appeared associated with somewhat reduced risk. The dose-response analysis showed that AD risk increased with BMI values exceeding ~ 29–30 kg/m^2 in midlife. These findings underscore a life-stage-dependent relationship between body mass and AD risk.

Adiposity, type-2 diabetes, and Alzheimer's disease

Luchsinger JA, Gustafson DR (2009) [17]

Luchsinger and Gustafson's review focused on epidemiological links among adiposity, type-2 diabetes (T2D), and Alzheimer's disease. The authors reported that higher adiposity in middle age is consistently related to increased AD risk, potentially mediated by hyperinsulinemia, cerebrovascular disease, and metabolic disturbances. However, evidence for late-life adiposity was conflicting, with some studies suggesting weaker or inverse relationships. The review emphasized the complex interplay between obesity-related metabolic factors and neurodegeneration.

Obesity, insulin resistance, and Alzheimer's disease

Hildreth et al (2012) [18]

Hildreth and colleagues reviewed evidence linking obesity-induced insulin resistance (IR) to Alzheimer's disease pathology. They highlighted that peripheral IR and hyperinsulinemia are consistently elevated in individuals with AD and may precede clinical symptoms. Higher IR was associated with reduced cerebral glucose metabolism and increased amyloid plaque burden in later life, suggesting that obesity-related metabolic dysfunction might drive early AD changes. However, the authors noted heterogeneity among studies and the need for further investigation.

A potential link between visceral obesity and risk of Alzheimer's disease

Al-Kuraishy HM et al (2023) [19]

Al-Kuraishy and colleagues reviewed the potential link between visceral obesity and Alzheimer's disease. They reported that visceral adipose tissue secretes pro-inflammatory cytokines that promote systemic inflammation and insulin resistance, which in turn may impair brain insulin signaling and contribute to AD pathology. The review emphasized oxidative stress, neuroinflammation, and dysregulated adipocytokines as key mechanisms by which visceral adiposity may elevate AD risk.

Adiposity and Alzheimer's disease

Luchsinger JA, Mayeux R (2007) [20]

In this earlier review, Luchsinger and Mayeux discussed the relationship between adiposity and Alzheimer's disease, noting that higher body fat, particularly when measured by BMI, was associated with increased risk of AD. They highlighted pathways including hyperinsulinemia and diabetes as mediators of this association, while also recognizing methodological challenges in longitudinal studies. This foundational work helped frame later research on metabolic risk factors and AD.

Does obesity increase the risk of dementia: a literature review

Anjum I, et al (2018) [21]

A systematic review exploring obesity and dementia, including Alzheimer's disease, revealed that midlife obesity is associated with accelerated cognitive decline, whereas late-life obesity displayed inconsistent or inverse associations with dementia risk. The authors noted the role of inflammatory adipokines and vascular contributions to neurodegeneration, emphasizing heterogeneous epidemiological findings and the need for improved measurement methods.

The link between metabolic syndrome and Alzheimer's disease: a mutual relationship and long rigorous investigation

Al-Kuraishy HM et al (2023) [22]

A 2023 review on metabolic syndrome highlighted that components including visceral obesity, insulin resistance, hypertension, and dyslipidemia synergistically promote AD neuropathology. MetS was linked to neuroinflammation, oxidative stress, impaired insulin signaling, and mitochondrial dysfunction, all processes implicated in AD progression. The integrated perspective underscores multifactorial metabolic contributions to AD risk beyond body weight alone.

Body mass index in midlife and late-life as a risk factor for dementia: a meta-analysis of prospective studies

Anstey KJ et al (2011) [23]

In this dose-response meta-analysis, elevated midlife BMI was linked to increased risk of Alzheimer's disease and other dementia subtypes. Specifically, dementia risk rose significantly when BMI exceeded midlife thresholds (~30 kg/m^2), whereas higher late-life BMI showed protective associations under certain cut-offs. These nuanced results reinforce age-dependent effects of adiposity on neurodegenerative risk.

Body mass index levels and changes before and after dementia diagnosis and risk of all-cause mortality: a nationwide cohort study

Huh Y et al (2026) [24]

Huh and colleagues conducted a large meta-analysis of 2.8 million adults examining anthropometry and dementia risk, including Alzheimer's disease outcomes. The study found that higher BMI and adverse weight change patterns were associated with increased dementia risk, reinforcing earlier findings on midlife adiposity as a significant predictor. The analysis emphasized the clinical relevance of long-term body weight monitoring in dementia prevention strategies.

Continued page 85

Table 1: Results – Continuation

Mechanisms underlying obesity-induced Aβ accumulation in Alzheimer's disease: a qualitative review

Wen W et al (2024) [25]

In a qualitative review investigating mechanistic links between obesity and Alzheimer's disease, Wen et al. synthesized evidence suggesting that obesity accelerates amyloid-β (Aβ) accumulation in the brain through multiple pathways, including chronic insulin resistance, adipocyte-driven inflammation, and alterations in lipid metabolism that impair Aβ clearance. The authors highlighted that adipose-derived cytokines and metabolic dysfunction facilitate blood-brain barrier disruption and microglial activation, thereby promoting amyloidogenic processes. The review underscored the need for more in-vivo studies to validate these proposed mechanisms and to clarify how obesity-related metabolic disturbances directly contribute to hallmark AD pathology.

Obesity paradox in Alzheimer's disease: a systematic review and meta-analysis of anthropometric measures and age-dependent effects

Son J et al (2025) [26]

Son et al. conducted a systematic review and meta-analysis examining how different anthropometric measures relate to Alzheimer's disease risk across age groups. They reported an "obesity paradox," wherein conventional markers of obesity (BMI, waist circumference) were associated with higher AD risk in midlife, but showed inconsistent or sometimes inverse associations in older adults. The analysis highlights that anthropometric indicators may differentially relate to AD depending on age stage, with visceral adiposity measures demonstrating stronger associations than overall BMI. These findings substantiate the notion of age-dependent risk modulation and emphasize careful interpretation of obesity measures in AD research.

Effects of obesity on plasma biomarker and amyloid PET trajectories in Alzheimer's disease

Mohammadi S et al, ADNI Collaborators (2025) [27]

Using data from the Alzheimer's Disease Neuroimaging Initiative (ADNI), Mohammadi and colleagues examined how baseline obesity status impacts Alzheimer's blood biomarkers (BBMs) and amyloid PET imaging trajectories. Their results demonstrated that higher body mass index at baseline correlated with lower baseline levels of plasma phosphorylated tau (p-tau217) and other neurodegeneration markers, but longitudinal analysis showed that higher BMI was associated with slower increases in certain AD biomarkers and amyloid burden over time. These complex findings suggest that obesity may influence both peripheral biomarker expression and central amyloid deposition differently across disease stages, highlighting the nuanced effects of adiposity on AD biomarker trajectories.

Obesity during preclinical Alzheimer's disease development exacerbates brain metabolic decline

Anderson T et al (2024) [28]

In an experimental and translational study, Anderson et al. showed that obesity accelerates metabolic decline in brain regions susceptible to Alzheimer's pathology even in the preclinical stage. Using animal models and human cohort data, they found that high-fat diet-induced obesity led to impaired glucose metabolism in hippocampal and cortical areas critical for memory and cognitive function. These metabolic changes mirrored early AD neuropathology and were associated with worsened cognitive performance, supporting the hypothesis that obesity drives early brain metabolic dysfunction that may precede clinical AD.

Obesity and Alzheimer's disease dementia: Examining inflammatory links to cognitive decline and neuropsychiatric symptoms

Koriath CA, Perneczky R (2025) [29]

Koriath and Perneczky critically examined the relationship between obesity, systemic inflammation, and Alzheimer's disease dementia. Their review highlighted that individuals with obesity exhibit elevated inflammatory biomarkers (C-reactive protein, complement C3) which correlated with increased cognitive decline and more severe neuropsychiatric symptoms in AD patients. The authors underscored that chronic inflammation might mediate obesity's detrimental effects on cognition, potentially through microglial activation and cytokine-driven neurodegenerative pathways. This work supports the significance of targeting inflammatory processes when assessing obesity-associated AD risk.

Obesity, type-2 diabetes, and metabolic dysfunction (Luchsinger & Gustafson, 2009; Luchsinger & Mayeux, 2007) [17, 20]

The foundational reviews by Luchsinger and colleagues established the conceptual framework linking obesity, type-2 diabetes (T2D), and AD. Their consistent observation that midlife adiposity increases AD risk, potentially mediated by hyperinsulinemia and cerebrovascular disease, remains highly relevant. These works emphasize that obesity does not act in isolation but operates within a broader metabolic network involving insulin resistance, vascular injury, and systemic inflammation [36]. Importantly, they also acknowledged inconsistencies in late-life findings, highlighting methodological challenges such as confounding and measurement variability. These early insights laid the groundwork for understanding AD as a metabolic-vascular neurodegenerative disorder, a perspective that continues to guide contemporary research.

Insulin resistance and brain hypometabolism (Hildreth et al., 2012) [18]

Hildreth and colleagues extend the metabolic hypothesis by identifying insulin resistance (IR) as a key intermediary between obesity and AD pathology. Their findings that peripheral IR and hyperinsulinemia are elevated in AD and associated with reduced cerebral glucose metabolism and increased amyloid burden support the "type-3 diabetes" hypothesis of AD. The implication is that chronic metabolic stress may impair neuronal insulin signalling, reducing synaptic plasticity and glucose utilization in vulnerable brain regions [37]. Although heterogeneity among studies was noted, the consistent link between IR and hypometabolism aligns closely with Lee et al.'s imaging findings, suggesting a convergent pathway through which obesity contributes to neurodegeneration.

Visceral obesity and inflammatory mechanisms (Al-Kuraishy et al., 2023) [19]

The emphasis on visceral adiposity rather than generalized obesity provides important mechanistic clarification. Al-Kuraishy et al. demonstrate that visceral fat acts as an endocrine organ, releasing pro-inflammatory cytokines and adipokines that promote systemic inflammation and oxidative stress [38]. These processes impair blood-brain barrier integrity and exacerbate microglial activation, potentially accelerating AD pathology. This work complements epidemiological findings indicating stronger associations for central adiposity measures than BMI [39]. It also suggests that interventions targeting visceral fat reduction may yield greater neuroprotective benefits than weight reduction alone.

Metabolic syndrome as a multifactorial contributor (Al-Kuraishy et al., 2023) [22]

The review of metabolic syndrome (MetS) expands the scope beyond obesity to include hypertension, dyslipidemia, and im-

paired glucose tolerance. The evidence that MetS components synergistically promote neuroinflammation, mitochondrial dysfunction, and oxidative stress suggests that AD risk is amplified by cumulative metabolic burden rather than isolated obesity [40]. This integrated perspective helps to explain why some obese individuals do not develop AD while others with clustered metabolic abnormalities are at substantially higher risk. It reinforces the importance of comprehensive cardiometabolic risk management in AD prevention strategies.

Amyloid-β accumulation pathways (Wen et al., 2024) [25]

Wen et al. provide mechanistic insight into how obesity may influence amyloid pathology. Their synthesis of evidence indicating that obesity accelerates Aβ accumulation through impaired clearance, insulin resistance, and inflammatory signalling suggests a plausible biological link between metabolic dysfunction and hallmark AD pathology. However, the need for more in vivo validation highlights ongoing uncertainty [41–43]. Notably, Lee et al. did not observe direct associations between BMI and amyloid deposition, suggesting that amyloid accumulation may be indirectly mediated or influenced by disease stage. This discrepancy underscores the complexity of disentangling metabolic from amyloid pathways.

Biomarker trajectories and stage-specific effects (Mohammadi et al., 2025) [27]

Mohammadi and colleagues provide nuanced evidence that obesity may influence plasma biomarkers and amyloid PET trajectories differently across disease stages. Their finding that higher baseline BMI correlated with lower baseline plasma p-tau levels but slower longitudinal biomarker progression complicates traditional interpretations of obesity as uniformly detrimental [44, 45]. These results may reflect altered biomarker expression in obese individuals or stage-specific metabolic interactions. Importantly, this study highlights the need for longitudinal biomarker studies to clarify whether obesity modifies disease expression rather than simply increasing risk.

Preclinical brain metabolic decline (Anderson et al., 2024) [28]

The translational work by Anderson et al. strengthens causal inference by demonstrating that obesity exacerbates brain metabolic decline even before clinical AD onset. The observed impairments in hippocampal and cortical glucose metabolism parallel human imaging findings and provide experimental support for the metabolic hypothesis. These findings suggest that obesity may lower the threshold for neurodegeneration by compromising neuronal energy metabolism during preclinical stages [46, 47]. The alignment between experimental and human cohort evidence enhances confidence in the biological plausibility of the association.

Inflammatory burden and clinical expression (Koriath & Perneczky, 2025) [29]

The observation that obese individuals with AD exhibit elevated inflammatory markers correlated with worse cognitive decline and neuropsychiatric symptoms emphasizes the clinical consequences of obesity-associated inflammation. Chronic systemic inflammation may amplify microglial activation and accelerate neuronal damage, influencing symptom severity [48–50]. This finding integrates well with mechanistic reviews highlighting cytokine-mediated neurotoxicity and underscores inflammation as a therapeutic target.

Overall Patterns and Inconsistencies

Across studies, a consistent pattern emerges: midlife obesity is a robust risk factor for AD, whereas late-life findings are heterogeneous and influenced by reverse causation and survival bias. Visceral adiposity, insulin resistance, and metabolic syndrome appear more predictive than BMI alone. Mechanistically, metabolic dysfunction, neuroinflammation, and impaired glucose metabolism represent converging pathways linking obesity to neurodegeneration. Inconsistencies – particularly regarding amyloid associations and late-life obesity – likely reflect differences in study design, measurement methods, disease stage, and population characteristics.

Limitations of the Evidence

Several limitations should be acknowledged. Many studies rely on BMI as a proxy for adiposity, which does not differentiate fat distribution or lean mass. Reverse causation remains a significant concern in late-life analyses. Observational designs limit causal inference, and heterogeneity in diagnostic criteria and biomarker measurement complicates comparisons. Furthermore, genetic moderators such as APOE ε4 status are not uniformly accounted for across studies.

Implications and Future Directions

Collectively, the evidence suggests that obesity – particularly in midlife – is a modifiable risk factor for Alzheimer's disease, mediated largely through metabolic and inflammatory pathways. Preventive strategies should prioritize early metabolic health optimization, including weight management, glycemic control, and reduction of visceral adiposity. Future research should incorporate precise adiposity measures, longitudinal biomarker tracking, and stratification by genetic risk to clarify stage-specific and mechanistic relationships.

In summary, the findings support a model in which obesity contributes to AD primarily through chronic metabolic stress and neuroinflammation, influencing neurodegeneration and possibly modifying amyloid dynamics. Addressing obesity and metabolic syndrome may therefore represent a critical avenue for reducing the global burden of Alzheimer's disease.

Conclusion

This systematic review consolidates epidemiological, mechanistic, and biomarker-based evidence examining the relationship between obesity and Alzheimer's disease (AD). The collective findings strongly indicate that midlife obesity is associated with an increased risk of developing Alzheimer's disease and related cognitive decline, whereas associations in late-life appear heterogeneous and may be influenced by reverse causation, survival bias, and disease-related weight loss. Across studies, central or visceral adiposity, insulin resistance, and components of metabolic syndrome demonstrated stronger and more consistent associations with AD risk than body mass index (BMI) alone, highlighting the importance of metabolic health beyond simple weight classification.

Mechanistically, obesity appears to contribute to AD pathogenesis through interconnected pathways involving chronic systemic inflammation, impaired insulin signalling, oxidative stress, cerebrovascular dysfunction, and altered brain glucose metabolism. Neuroimaging and experimental studies further support the hypothesis that obesity accelerates brain hypometabolism in AD-vulnerable regions, potentially lowering the threshold for neurodegeneration during preclinical stages. While evidence linking obesity directly to amyloid-β and tau pathology remains mixed, the broader metabolic and inflammatory environment associated with obesity likely amplifies neurodegenerative processes and influences disease progression.

Importantly, the life-course perspective emerging from this review underscores that timing of exposure is critical. Obesity during midlife represents a particularly sensitive window during which excess adiposity may initiate long-term metabolic and vascular changes that predispose individuals to later cognitive impairment. Conversely, late-life weight changes may reflect underlying neuropathology rather than act as independent risk modifiers. These findings emphasize the need for cautious interpretation of anthropometric measures in older populations.

Despite growing evidence, limitations remain. Many studies rely on BMI as a surrogate for adiposity, lack longitudinal biomarker confirmation, or are observational in nature, limiting causal inference. Future research should prioritize precise measures of visceral fat, integrate multimodal biomarkers, account for genetic susceptibility (e.g., APOE status), and evaluate the impact of targeted metabolic interventions on long-term cognitive outcomes.

In conclusion, the evidence synthesized in this review supports the conceptualization of Alzheimer's disease, at least in part, as a metabolically influenced neurodegenerative disorder. Obesity – particularly when present in midlife – represents a potentially modifiable risk factor, and its prevention and management may play a meaningful role in reducing the global burden of AD. Strengthening metabolic health through early lifestyle interventions and comprehensive cardiometabolic risk control may therefore constitute an important component of future dementia prevention strategies.

Conflict of interest

None.

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