

The Evolving Paradigm of Nutritional Psychiatry: From Primordial Neurodevelopment to Cognitive Resilience Across the Life-course

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Abstract

Neurodegenerative diseases, including Alzheimer's disease and other forms of dementia, represent an escalating global health crisis projected to affect 135 million individuals by 2050. Given the limited efficacy of traditional pharmacological approaches, a paradigm shifts toward modifiable risk reduction is essential. The current consensus indicates that up to 45% of dementia cases are potentially preventable through optimization of lifestyle and nutritional determinants. This manuscript presents a lifespan perspective on neuroprotection, asserting that cognitive resilience is a cumulative process that begins in the primordial developmental window. We analyze how maternal nutrition and adolescent dietary choices program "brain reserve" and determine late-life vulnerability through mechanisms of "inflammaging" and metabolic energy starvation. Central to the degenerative trajectory is the "pathological bridge" established by Western dietary patterns and ultra-processed foods (UPFs). Recent evidence (2024–2025) links high UPF consumption to a 28% acceleration in global cognitive decline and to systemic neuroinflammation mediated by the gut-microbiota-immune-brain axis. Conversely, evidence-based nutritional patterns, such as the Mediterranean and Mediterranean Intervention for Neurodegenerative Delay (MIND) diets, offer significant neuroprotection by neutralizing oxidative stress and enhancing cerebral perfusion. We further evaluate innovative strategies, including psychobiotics to modulate the gut microbiome and ketogenic therapies for "brain energy rescue" in glucose-impaired brains. Findings from the Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability (FINGER) and Ornish trials suggest that intensive multidomain interventions can stabilize or improve cognition even after clinical onset. We conclude that integrating nutritional screening into routine clinical care and national policy frameworks is an urgent imperative to mitigate the personal and economic burden of pathological aging.

Keywords: Nutritional psychiatry, Neurodegeneration, Gut-brain axis, Lifespan neuroprotection

Introduction: The Neurodegenerative Crisis and Modifiable Risk

Neurodegenerative diseases, including Alzheimer's disease (AD) and other forms of dementia, have emerged as profound global health challenges and are now a leading cause of disability and healthcare burden [1,2]. As global populations continue to age, the number of affected individuals is projected to double every 20 years, potentially reaching 135 million by 2050 [3]. This escalating crisis is further exacerbated by the stagnant prevalence of mental disorders despite decades of pharmacological advancement, underscoring an urgent need to transition from reactive treatment models to proactive, modifiable prevention strategies [4,5].

While advanced age and genetic predisposition remain the primary non-modifiable risk factors, contemporary evidence

supports a paradigm shift toward modifiable risk reduction. International expert consensus now suggests that up to 45% of dementia cases may be prevented or delayed by optimizing lifestyle and nutritional determinants [6]. This highlights the critical role of nutritional psychiatry, as diet serves as a primary modifiable lever for maintaining cognitive function and psychological well-being throughout the human lifespan [7,8].

Building on the lifespan perspective established in the focal manuscript, "Nutrition and Mental Health Across the Lifespan," this communication posits that neuroprotection is a cumulative process that must commence decades before clinical manifestations emerge [9]. Determinants of brain health interact dynamically across developmental epochs, necessitating a focus on mitigating pathological processes long before the onset of late adulthood [6,10].

Central to this preventive approach is mitigating "inflammaging," a state of chronic, low-grade systemic inflammation driven in part by age-related changes in the gut microbiota, which is a major contributor to neurodegenerative pathogenesis [6,11]. Furthermore, the aging brain often encounters "energy starvation," a metabolic state in which the capacity to utilize glucose is impaired, leading to critical energy deficits that precede clinical symptom onset [6,12]. By addressing these modifiable mechanisms early in the life cycle, clinicians can better scaffold cognitive reserves and promote long-term psychological resilience [8,9].

Materials and Methods

To evaluate the impact of nutrition on neurodegeneration and mental health across the lifespan, a narrative literature review was conducted by searching electronic databases including PubMed, Scopus, Web of Science, and Google Scholar for high-impact longitudinal studies, systematic reviews, and randomized controlled trials published between 2014 and 2025. The search strategy utilized Medical Subject Headings (MeSH) and keywords such as "Nutritional Psychiatry," "Neurodegeneration," "Gut-Brain Axis," "Ultra-processed foods," "Mediterranean and MIND diets," and "Inflammaging". Studies were selected based on their relevance to a lifespan perspective—ranging from maternal nutrition and adolescent dietary programming to mid-life metabolic signatures and precision nutrition—while excluding animal models without clear human translational relevance. The gathered evidence was synthesized to establish the "pathological bridge" between modern dietary patterns and neuroinflammatory mechanisms, emphasizing state-of-the-art findings from 2024 and 2025 regarding cognitive resilience and brain energy rescue.

To ensure methodological transparency and reproducibility, the full literature search strategy, including database-specific query syntax, applied filters, and the number of records retrieved, is presented in **Table 1**. The search yielded a combined total of 9,570 records across three major databases (PubMed: 587; Scopus: 63; Google Scholar: 8,920), prior to deduplication and eligibility screening. Records were subsequently screened by title and abstract, and studies meeting the predefined inclusion criteria, peer-reviewed longitudinal cohort studies, systematic reviews, meta-analyses, and randomized controlled trials with human translational relevance, published in English between 2014 and 2025, were included in the narrative synthesis.

Lifespan Accumulation: From Barker’s Hypothesis to Brain Reserve

The trajectory of neurodegenerative health is increasingly understood through the "Developmental Origins of Health and Disease" (DOHaD) paradigm, also known as Barker’s hypothesis [8,9]. This framework posits that the intrauterine environment and early postnatal experiences permanently program the offspring’s physiology and metabolism, establishing a foundation for late-life cognitive resilience or vulnerability [13,14]. Maternal nutrition is central to this primordial window; adequate levels of folate, iron, and docosahexaenoic acid (DHA) are essential for neurogenesis, myelination, and hippocampal development [9,15]. Deficiencies in these key nutritional building blocks can diminish "brain reserve," the structural and functional capacity that serves as a vital buffer against age-related neuropathology [6,14]. Clinical proxies for this reserve, such as larger skull circumference and longer leg length (indicators of optimal early-life nutrition), have been independently associated with a reduced prevalence

Table 1. Literature search strategy and results.

Database	Search Keywords / MeSH Terms	Limits / Filters	Results
PubMed (MEDLINE)	("Nutritional Psychiatry"[MeSH] OR "Neurodegeneration" OR "Gut-Brain Axis" OR "Ultra-processed foods" OR "Mediterranean diet" OR "MIND diet" OR "Inflammaging") AND ("cognitive decline" OR "dementia" OR "neuroprotection" OR "mental health")	Publication date: 2014–2025; Language: English; Study types: systematic reviews, meta-analyses, RCTs, longitudinal cohort studies; Exclusion: animal-only models without human translational relevance	587
Scopus	TITLE-ABS-KEY ("Nutritional Psychiatry" OR "Neurodegeneration" OR "Gut-Brain Axis" OR "Ultra-processed foods" OR "Mediterranean diet" OR "MIND diet" OR "Inflammaging") AND TITLE-ABS-KEY ("cognitive decline" OR "dementia" OR "neuroprotection" OR "mental health")	Publication date: 2014–2025; Language: English; Study types: systematic reviews, meta-analyses, RCTs, longitudinal cohort studies; Exclusion: animal-only models without human translational relevance	63
Google Scholar	"Nutritional Psychiatry" OR "Neurodegeneration" OR "Gut-Brain Axis" AND "Ultra-processed foods" OR "Mediterranean diet" OR "MIND diet" AND "cognitive decline" OR "neuroprotection"	Publication date: 2014–2025; Sorted by relevance; First 200 results screened	8920

Table 2. Comparative analysis of neuroprotective dietary frameworks.

Dietary Pattern	Primary Food Components	Key Neuroprotective Evidence	Target Mechanisms
Mediterranean (MedDiet)	Polyphenols, omega-3 fatty acids, and fiber [23].	Reduces depressive symptoms by 32–45% [23]; inversely associated with anxiety and stress [24].	Lowers systemic inflammation, promotes microbial diversity [23], and preserves vascular health [6,10].
MIND Diet	Leafy greens and berries rich in antioxidant phytochemicals [25]	Up to 53% reduction in AD risk; cognitive scores equivalent to being 7.5 years younger [25]	Neutralizes oxidative stress and inhibits microglial activation [25]
Ketogenic / KMT	Ketone bodies or Medium-Chain Triglyceride (MCT) oils [26]	Improves memory recall and daily function [26]; benefits may be genotype-specific (APOEε4-negative) [26]	"Brain energy rescue" by bypassing impaired glucose pathways in the aging brain [6,12].

of dementia in diverse global cohorts [14]. Beyond infancy, adolescence marks a second critical window of biological vulnerability, characterized by continued maturation of the prefrontal cortex and limbic structures [9,16]. During this period, a shift toward a high-consumption Western dietary pattern (WDP) and ultra-processed foods (UPFs) often replaces traditional, nutrient-dense diets [9,17]. High-fat, high-sugar adolescent diets are linked to a systemic pro-inflammatory profile, marked by significant elevations in C-reactive protein (CRP) and interleukin-6 (IL-6) [17,18]. This chronic low-grade inflammation can exacerbate microglial activation and impair the synthesis of brain-derived neurotrophic factor (BDNF), effectively priming the maturing brain for accelerated cognitive decline in subsequent developmental epochs [9,11]. Crucially, neurodegeneration is not an acute event but a multi-decade trajectory [8,14]. Pathophysiological accumulation, including glucose hypometabolism and insulin resistance, often begins in mid-life, occurring 20 to 30 years before a clinical diagnosis [6,16]. A hallmark of this clinical prodrome is a gradual decline in body mass; significant unintentional weight loss often precedes the symptomatic manifestation of dementia by several years [19]. In parallel, diets characterized by high UPF intake have been conclusively linked to accelerated cognitive aging; a dose-response meta-analysis encompassing over 260,000 participants identified a 28% increase in global cognitive decline among the highest UPF consumers [20,21]. This degeneration is exacerbated by systemic inflammation, insulin resistance, and hypertension [22], which collectively constitute a "pathological bridge" that transitions the brain from metabolic competence to progressive energy insufficiency [6,7].

The Pathological Bridge: Western Dietary Patterns and Ultra-Processed Foods

The integration of Western dietary patterns (WDP) and ultra-processed foods (UPFs) into global food systems

represents a critical public health crisis with profound neurocognitive consequences [5,27,28]. High consumption of UPFs is characterized by elevated intakes of added sugars, saturated fats, refined carbohydrates, and sodium, coupled with deficiencies in dietary fiber, essential micronutrients, and phytochemicals [5,20,21]. Systematic analyses have established significant associations between chronic UPF consumption and increased risk for depression, anxiety, and early-onset dementia [21,27].

The mechanistic underpinnings of UPF-induced neuropathology converge on the gut-microbiota-immune-brain axis [27,29]. Diets rich in UPFs disrupt the intestinal microbiome, resulting in bacterial dysbiosis characterized by a reduction in beneficial commensal species such as *Lactobacillus* and *Bifidobacterium* and an overgrowth of pathogenic taxa [11,27–29]. This compositional shift compromises epithelial barrier integrity, enabling bacterial endotoxins to translocate into systemic circulation and trigger neuroinflammatory signaling pathways, including nuclear factor kappa B (NF-κB) and pro-inflammatory cytokine cascades [27,29]. The resultant "inflammaging" state, characterized by persistently elevated markers of systemic inflammation, is a well-documented risk factor for cognitive decline [6,11].

Notably, the gut microbiome synthesizes key neuroactive metabolites, including short-chain fatty acids (SCFAs) such as butyrate, acetate, and propionate, as well as neurotransmitter precursors such as tryptophan and gamma-aminobutyric acid (GABA) [29–31]. Western dietary patterns deplete the capacity of the microbiome to generate these neuroprotective compounds, thereby diminishing microbial-derived support for neuronal integrity, synaptic plasticity, and mood regulation [27,29,31]. The emerging field of psychobiotics offers a novel therapeutic avenue by utilizing specific probiotic strains to restore microbiome homeostasis and attenuate neuropsychiatric symptoms [30–32].

Table 3. Mechanisms of action: pathological vs protective factors.

Category	Nutrient/Factor	Primary Mechanism of Action	Neurological Impact
Pathological (UPFs/WDP)	Refined Sugars & Fats	Catalyze gut dysbiosis and “leaky gut” [28]	Translocation of LPS triggers microglia-driven neuroinflammation [27].
	Processing Contaminants	Generation of AGEs and acrylamide during thermal processing [33]	Direct pro-inflammatory and neurotoxic effects upon chronic exposure [33]
Protective (Essential)	B-Vitamins (B12, B6, Folate)	Essential for CNS homeostasis and neurogenesis [34]	Inadequate B6 status associated with a 3.5-fold higher risk of accelerated cognitive decline [34]
	Magnesium & Selenium	Magnesium acts as an NMDA receptor antagonist [Kirkland]; Selenium neutralizes oxidative stress [35]	Prevents excitotoxic neuronal cell death and supports mood regulation [35,36].
	Short-Chain Fatty Acids	Produced by bacteria like <i>Faecalibacterium prausnitzii</i> [37]	Suppress microglial activation and enhance BBB integrity [37,38]

Evidence-Based Nutritional Strategies: The Mediterranean and MIND Diets

In contrast to pro-inflammatory Western dietary patterns, the Mediterranean diet (MedDiet) and the Mediterranean Intervention for Neurodegenerative Delay (MIND) diet constitute evidence-based nutritional frameworks associated with significant neuroprotective effects [25]. The MedDiet emphasizes high consumption of olive oil, fish, nuts, legumes, whole grains, and fresh vegetables, while limiting red meat, processed meats, and refined sugars. The MIND diet integrates key features of the MedDiet with additional emphasis on berry intake and leafy green vegetables, both rich in polyphenolic compounds that neutralize oxidative stress and enhance cerebrovascular perfusion [25].

Longitudinal epidemiological studies have consistently demonstrated that adherence to the MIND diet is associated with a significantly reduced incidence of Alzheimer's disease, with high adherence conferring up to a 53% risk reduction [25]. Further supporting this association, a randomized feasibility trial conducted among middle-aged adults reported that a 12-week MIND diet intervention led to measurable improvements in executive function, processing speed, and self-reported psychological well-being [39]. These cognitive enhancements are believed to result from the synergistic effects of omega-3 polyunsaturated fatty acids (PUFAs), antioxidants such as vitamin E and flavonoids, and the neuroprotective phytochemicals abundant in plant-based components [25,39].

The protective mechanisms of the MedDiet and MIND diet are multifaceted, encompassing anti-inflammatory, anti-oxidative, and metabolic pathways [29,39]. Olive oil, a cornerstone of these diets, is rich in oleic acid and phenolic compounds such as oleocanthal, which possess potent anti-

inflammatory properties and have been shown to inhibit amyloid-beta aggregation [40]. Omega-3 PUFAs derived from fish enhance membrane fluidity, synaptic plasticity, and neurotrophin signaling pathways, including upregulation of BDNF [29,32]. Furthermore, dietary polyphenols, particularly anthocyanins from berries and catechins from green tea, exert anti-oxidative effects by scavenging reactive oxygen species (ROS) and modulating mitochondrial [29,30,39]. Collectively, these dietary interventions foster a metabolic and inflammatory environment conducive to sustained cognitive health across the lifespan.

Innovative Interventions: Psych Biotics and Ketogenic "Brain Energy Rescue"

Emerging therapeutic strategies are focusing on modulation of the gut microbiome and metabolic reprogramming to counteract neurodegenerative pathology. Psych biotics, defined as live microorganisms that confer mental health benefits through the gut-brain axis, represent a promising frontier in nutritional psychiatry [41–43]. Controlled trials have demonstrated that specific probiotic strains, including *Lactobacillus helveticus* R0052 and *Bifidobacterium longum* R0175, can significantly reduce symptoms of depression and anxiety by modulating the production of neurotransmitters, inflammatory markers, and hypothalamic-pituitary-adrenal (HPA) axis activity [31,32,44].

In parallel, ketogenic dietary interventions are being investigated as a strategy for "brain energy rescue" in glucose-impaired neurodegenerative states [6,26,45]. In Alzheimer's disease, regional cerebral glucose hypometabolism precedes clinical symptoms by many years, creating an energy deficit that compromises neuronal function [6,45]. Ketone bodies, particularly beta-hydroxybutyrate (BHB), provide an alternative fuel source that can bypass impaired glucose metabolism

and restore neuronal bioenergetics [26,45]. Clinical studies have reported that BHB supplementation improves cognitive performance in individuals with mild cognitive impairment (MCI), supporting the feasibility of ketogenic therapy as a neuroprotective strategy [26,45].

Furthermore, ketone bodies exert pleiotropic neuroprotective effects beyond energy provision, including anti-inflammatory actions, enhancement of mitochondrial function, and upregulation of neurotrophic factors such as BDNF [26,45]. Preliminary evidence suggests that ketogenic diets may be particularly beneficial in populations with concurrent metabolic syndrome, insulin resistance, or Type 2 diabetes mellitus, conditions that significantly elevate dementia risk [6,26]. Ongoing clinical trials are actively evaluating the long-term safety and efficacy of ketogenic interventions in both preventive and therapeutic contexts.

Multidomain Interventions: Insights from FINGER and Ornish Trials

The Finish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability (FINGER) was the first large-scale randomized controlled trial to demonstrate that multidomain lifestyle interventions can significantly reduce cognitive decline in at-risk older adults [46]. The FINGER trial enrolled 1,260 participants aged 60–77 years with elevated dementia risk, randomizing them to receive a comprehensive intervention consisting of dietary counseling (emphasizing a MedDiet-like pattern), physical exercise, cognitive training, and vascular risk monitoring, compared to standard health advice. After two years, participants in the intervention group exhibited significant improvements in global cognition, executive function, and processing speed compared to controls [46]. These findings provide robust evidence that intensive multidomain approaches can modify the neurodegenerative trajectory even in individuals with established risk factors.

Building on the FINGER model, the Ornish trial evaluated whether intensive lifestyle modifications could halt or reverse early-stage Alzheimer's disease. This randomized controlled trial assigned participants with mild cognitive impairment or early dementia to an intensive intervention comprising a whole-food, plant-based diet, moderate aerobic exercise, stress management through yoga and meditation, and group psychosocial support. At 20 weeks, the intervention group demonstrated stabilization or improvement in cognitive performance, as measured by validated neuropsychological assessments — including the CGIC, CDR Global, and CDR-SB — whereas the control group worsened across all four measures. Notably, biomarker analyses revealed a significant increase in the plasma A β 42/40 ratio in the intervention group and a significant improvement in gut microbiome composition, underscoring the mechanistic basis for the observed cognitive benefits [47].

These landmark trials collectively affirm that neuroprotection is not solely a function of genetic predisposition but can be substantially influenced by modifiable behaviors. The synergistic effects of dietary optimization, physical activity, cognitive engagement, and social interaction appear to confer additive or even multiplicative benefits, highlighting the importance of holistic, patient-centered care models [46,47].

Conclusion: Toward a Preventive Paradigm in Clinical Practice

The evidence synthesized in this communication underscores the fundamental role of nutrition as a primary, modifiable determinant of mental health across the lifespan [9]. To address the rising global burden of neurodegenerative disorders, healthcare professionals must move beyond traditional pharmacological models and integrate nutritional assessment and dietary intervention into standard clinical practice [9]. Clinical integration should begin with routine nutritional screening in primary care to identify specific vulnerabilities, such as micronutrient deficiencies or suboptimal dietary quality, well before the onset of symptomatic cognitive decline [12]. All individuals at risk for neurodegeneration should undergo regular weight monitoring and nutritional status assessments to identify early markers of disease, such as unintentional weight loss that frequently precedes a dementia diagnosis [14]. This standard of care requires robust interdisciplinary models in which psychiatrists, geriatricians, and dietitians collaborate to deliver person-centered care that addresses both mental and metabolic health [41]. At the policy level, there is an urgent imperative to recognize that "food is medicine" [10]. Given that up to 45% of dementia cases are potentially preventable through modifiable risk factors, nutrition must be positioned as a cornerstone of national healthcare roadmaps [6]. Comprehensive strategies, such as the Swiss Brain Health Plan, demonstrate the efficacy of combining whole-population health promotion with targeted early-detection programs for high-risk individuals [6]. Public health authorities should prioritize policies that reduce the availability of ultra-processed foods and increase access to affordable, nutrient-dense whole foods to mitigate the staggering economic and personal costs of pathological aging [12]. Ultimately, establishing a new standard of care centered on nutritional psychiatry is essential to fostering global cognitive resilience and improving the quality of life for an aging world population [4].

Moving forward, the shift from reactive pharmacology to proactive nutritional neurology must become standard in everyday clinical practice. Future healthcare frameworks should prioritize developing personalized nutritional "prescriptions" tailored to individual genetic risks and microbial signatures, particularly for high-risk groups such as APOE ϵ 4 carriers. Furthermore, educational initiatives

must target the adolescent window to counteract the neuroinflammatory impact of modern Western diets before a multi-decade trajectory of decline is established. The future of neurodegenerative prevention lies in the synergy of multidomain interventions that integrate nutrition, physical activity, and sleep hygiene to effectively "rescue" brain energy and scaffold cognitive reserve. By treating food not merely as fuel but as a targeted clinical tool, the medical community can transition toward a model that empowers individuals to safeguard their neurological vitality and maintain independence throughout the lifespan. The pursuit of cognitive resilience is a lifespan endeavor that begins in the womb and extends through late adulthood. By embracing a preventive paradigm rooted in nutritional optimization, microbiome modulation, and multidomain lifestyle interventions, clinicians and policymakers can mitigate the personal, familial, and societal burden of pathological aging. The time to act is now, as the neurobiological foundation for healthy aging is built decades before the emergence of clinical symptoms.

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