

NEUROPROTECTIVE TECHNIQUES FOR ALZHEIMER'S DISEASE: AN EXTENSIVE NARRATIVE ANALYSIS OF MECHANISMS AND FUTURE TREATMENT DIRECTIONS

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ABSTRACT

Background: Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by amyloid- β (A β) plaques, hyperphosphorylated tau, oxidative stress, mitochondrial dysfunction, synaptic degeneration, neurovascular dysregulation, and chronic neuroinflammation. Neuroprotection aims to preserve neuronal structure and function by intervening in convergent pathological pathways.

Aims: To summarize mechanistic evidence for neuroprotective strategies relevant to AD, including antioxidant, mitochondrial, cholinergic, neuropeptide, glial, cannabinoid, hormonal, metabolic, and A β -directed approaches.

Methodology: A systematic narrative review of literature published from January 1995 to March 2024 was described in the source manuscript. Web of Science, PubMed, and Scopus were searched, and studies were considered according to model validity, mechanistic clarity, translational relevance, and reproducibility.

Results: Thirty-four core mechanistic reviews and studies were compiled. Reported mechanisms included attenuation of reactive oxygen species, SIRT1 activation, TREM2 modulation, nitric oxide regulation, mitochondrial stabilization, A β clearance, reduction of tau phosphorylation, angiogenic support, and synaptic preservation.

Conclusions: The reviewed literature supports a multimodal neuroprotection framework targeting oxidative stress, neuroinflammation, mitochondrial dysfunction, and metabolic abnormalities. Early, biomarker-guided and combination approaches are identified as areas for future investigation.

KEYWORDS: Alzheimer's disease; neuroprotection; oxidative stress; microglia; SIRT1

INTRODUCTION

Alzheimer's disease (AD) is a major cause of disability in aging populations and is responsible for 60–70% of dementia cases globally [1]. Amyloid precursor protein-derived extracellular A β plaques, intracellular tau neurofibrillary tangles, synaptic degeneration, neuroinflammation, oxidative stress, and mitochondrial impairment are pathological features of AD [2]. Memantine, galantamine, rivastigmine, and donepezil are examples of traditional treatments that help with symptoms but do not substantially stop neuronal loss [3]. The goal of neuroprotection is to prevent neuronal death by reducing oxidative damage, restoring mitochondrial bioenergetics, regulating inflammatory cascades, and maintaining cellular viability [4]. Alzheimer's disease is increasingly considered a complex breakdown of brain homeostatic and defense systems rather than solely a disorder of protein misfolding. Interrupting pathological cascades before irreversible neuronal loss is the central objective of neuroprotection.

Several features make AD difficult to treat. The pathology develops silently for years before symptoms appear, so intervention often begins after substantial synaptic and neuronal loss has occurred [17]. Reviews of neuroprotective candidates also emphasize that no single molecular target accounts for neuronal death, and that oxidative, inflammatory, mitochondrial, vascular, and metabolic processes reinforce one another [10,13]. For this reason, the present review organizes neuroprotective strategies by the biological mechanism they address rather than by drug class, and highlights where these mechanisms converge.

The specific objectives of this review are (i) to describe the principal pathophysiological processes that neuroprotective approaches aim to interrupt; (ii) to summarize preclinical and early translational evidence for each category of intervention; (iii) to identify the limitations that have slowed clinical translation; and (iv) to outline directions for early, biomarker-guided, and combination approaches.

METHODS

Review Design: The source manuscript describes a structured narrative review with conceptual alignment to PRISMA reporting principles.

Registration: The review was described as a non-registered narrative review following PRISMA 2020 concepts.

Data Sources: Web of Science, PubMed, and Scopus were searched for literature published from 1995 to 2024.

Eligibility Criteria: Included material comprised in vitro and in vivo AD research models, mechanistic molecular studies, early clinical and translational research, and studies presenting neuroprotective effects. The source manuscript excluded

non-AD neurodegeneration, experiments limited to symptomatic cognitive enhancement, and observational case reports lacking mechanistic content.

Quality Assessment: The source manuscript states that quality was considered according to transgenic/model validity, mechanistic depth, consistency between models, and biomarker association/risk of prejudice.

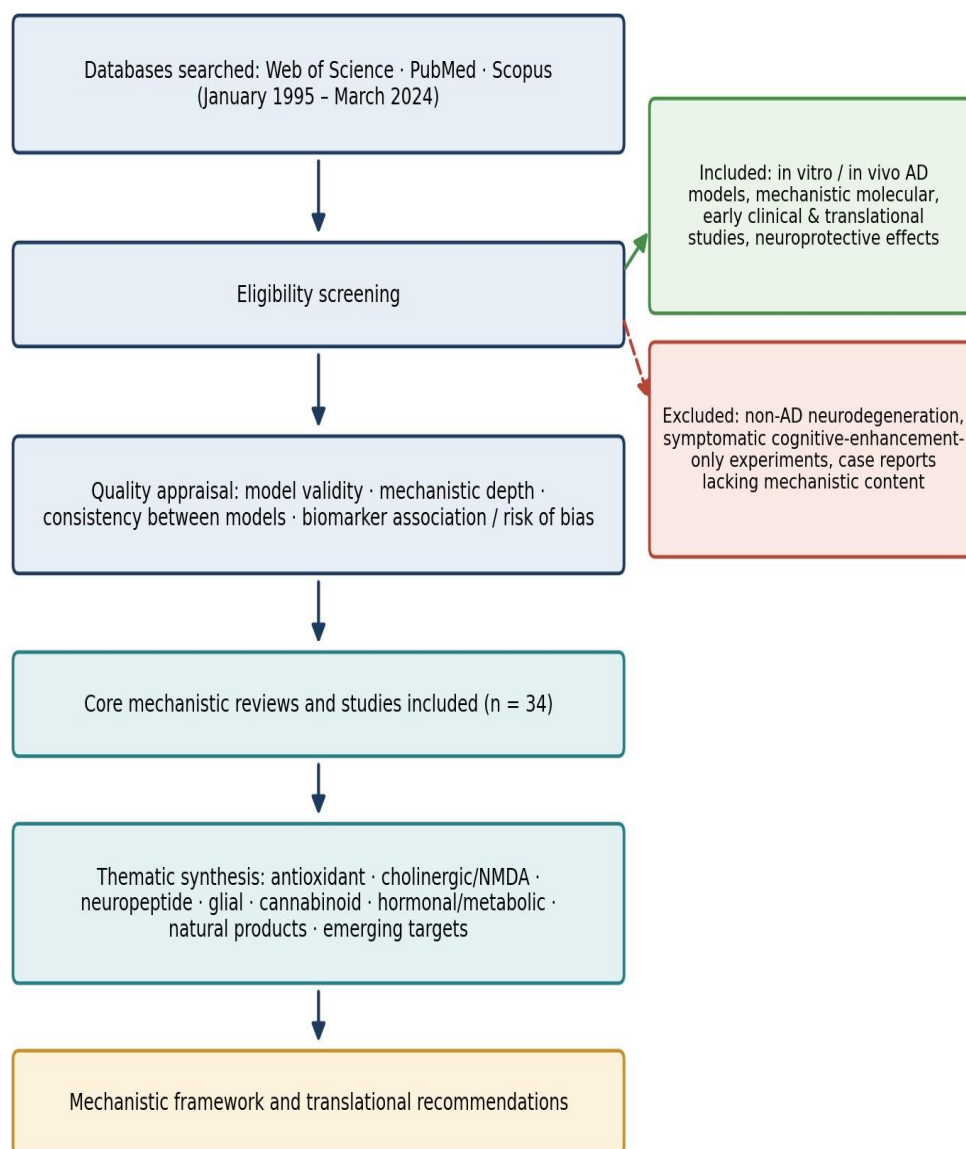


Figure 1. Flow diagram of the review process. Databases, date range, eligibility criteria, quality considerations, and the number of core studies (n = 34) are as described in the source manuscript.

PATHOPHYSIOLOGICAL BASIS OF NEUROPROTECTION

Oxidative stress and reactive oxygen species (ROS) contribute to lipid peroxidation, DNA damage, mitochondrial dysfunction, and apoptosis and may interact with neuroinflammation and tau pathology [11]. Nitric oxide has dual biological roles: physiological signaling and synaptic modulation, while excessive production may contribute to peroxynitrite-mediated neurotoxicity [15,16]. Microglia can display inflammatory or repair-oriented phenotypes, and TREM2 signaling has been associated with A β clearance and plaque compaction [30].

Mitochondrial dysfunction is regarded as an early and central event in AD. Impaired mitochondria generate more ROS and produce less energy, which in turn makes neurons, with their high metabolic demand, more vulnerable to A β - and tau-related toxicity [10,14]. Oxidative stress is therefore both a consequence and an amplifier of the amyloid and tau pathologies, and antioxidant strategies have been proposed as one route for interrupting this cycle [10].

Neuroinflammation adds a further layer. Glial cells can protect neurons by clearing debris and providing antioxidant and metabolic support, but sustained activation may lead to release of cytokines and nitric oxide that damage synapses [12,21]. The balance between these protective and harmful states is a recurring theme in the literature and forms the rationale for approaches that modulate, rather than simply suppress, glial activity [6,30].

Vascular and metabolic factors also contribute. Reduced cerebral perfusion, altered cholesterol handling, and hormonal changes with aging have each been linked to increased vulnerability to AD pathology, and some neuroprotective candidates act on these systems, including angiogenic support by transthyretin, cholesterol modulation by cyclodextrins, and estrogen signaling [25,26,31].

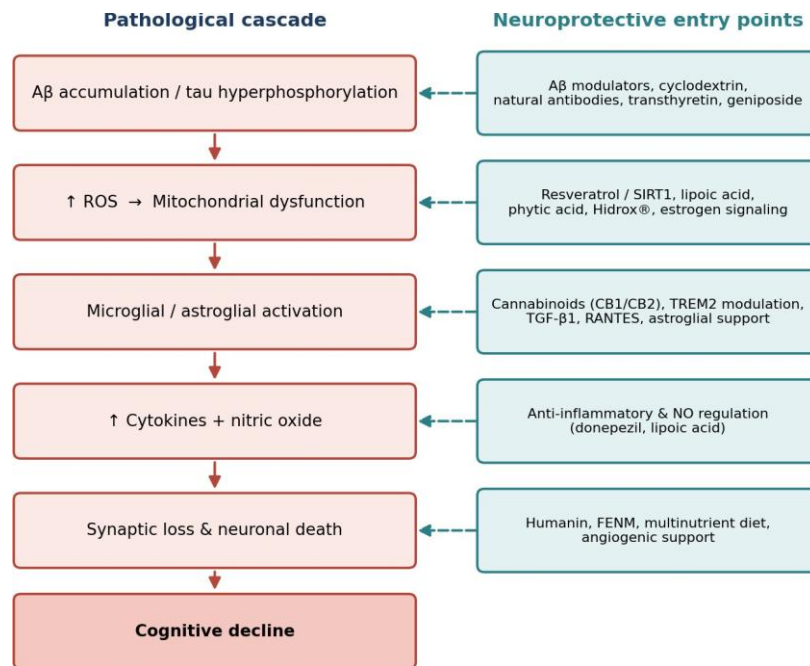


Figure 2. Pathological cascade in Alzheimer's disease (left) and the neuroprotective approaches discussed in this review mapped to the step they are proposed to target (right). Mapping is conceptual and summarizes the mechanisms described in the text.

ANTIOXIDANT NEUROPROTECTION

SIRT1 and resveratrol: Resveratrol has been reported to activate SIRT1, increase autophagy, decrease NF-κB signaling, and promote mitochondrial biogenesis [9,12,14]. Preclinical studies have reported reduced plaque burden and improved cognitive outcomes.

Lipoic acid: Lipoic acid has been described as reducing inflammatory cytokines and increasing glutathione levels [18].

Phytic acid: Phytic acid has been reported to reduce oxidative damage and chelate iron in AD models [34].

Together, these agents share a common logic: reducing oxidative burden and inflammatory signaling while supporting mitochondrial function. Much of the supporting evidence for resveratrol, however, comes from cell and animal models, and the reviews cited here point to limited bioavailability and the need for well-designed clinical studies [8,11,14].

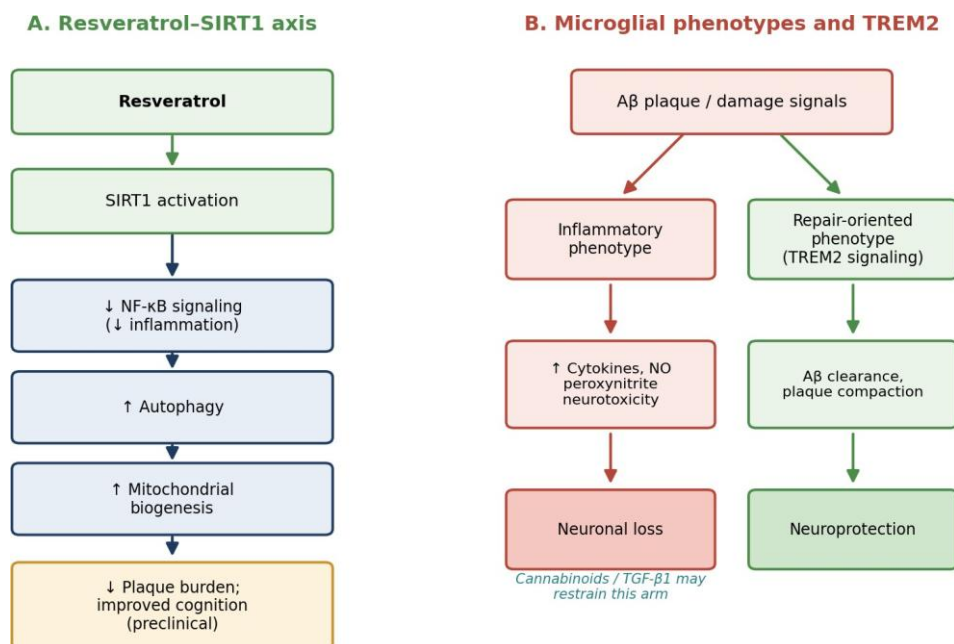


Figure 3. Proposed mechanisms of neuroprotection. (A) Resveratrol–SIRT1 signaling and its reported downstream effects. (B) Microglial phenotypes in the presence of Aβ, with TREM2 signaling associated with repair-oriented function.

CHOLINERGIC AND NMDA-BASED NEUROPROTECTION

Donepezil: In addition to acetylcholinesterase inhibition, donepezil has been reported to have anti-inflammatory and anti-apoptotic properties [2].

Fluoroethylnormemantine (FENM): Chronic administration of FENM has been reported to reduce excitotoxicity and preserve synaptic density in mouse models [23].

NEUROPEPTIDE-BASED STUDIES

Humanin: Humanin has been reported to prevent A β -induced apoptosis by interfering with caspase activation and Bax translocation [20].

Intranasal fatty neuropeptides: Intranasal administration has been described as decreasing tau pathology and increasing central nervous system bioavailability [10].

Natural antibodies: Natural antibodies targeting amyloid assemblies may diminish with aging and advancing AD [19].

MICROGLIA AND GLIAL MODULATION

Blocking excessive microglial activation may decrease neuronal loss [6]. Astroglia provide antioxidant support and metabolic coupling and may exhibit neuroprotective or neurotoxic properties depending on activation state [13,21]. RANTES upregulation has been proposed as a possible neuroprotective response [7], while TGF- β 1 signaling has been associated with repair and modulation of inflammatory cascades [8].

CANNABINOID-BASED NEUROPROTECTION

Cannabinoids acting through CB1 and CB2 receptors have been reported to reduce oxidative stress, excitotoxicity, and A β -associated neuroinflammation in experimental models [5].

HORMONAL AND METABOLIC REGULATION

Estrogen signaling: Activation of ER α and ER β has been associated with mitochondrial function and synaptic plasticity [25].

Transthyretin: Transthyretin-related neuroprotection has been associated with proteolytic activation, inhibition of fibrillation, angiogenic activity, and A β binding [31,32].

NATURAL PRODUCTS AND MULTINUTRIENT APPROACHES

Geniposide has been associated with decreased tau phosphorylation [22]. Alkaloids from *Dendrobium nobile* have reported antioxidant and anti-inflammatory properties [33]. Multinutrient diets have been reported to improve synaptic preservation and cerebral perfusion in experimental AD [29]. Hydroxytyrosol-containing Hidrox® has been investigated in relation to neuroprotection and biochemical links between traumatic brain injury and AD [24].

EMERGING TARGETS

Cyclodextrins have been investigated for modulation of A β aggregation and cholesterol metabolism [26]. The translational results of A β modulators have not been entirely consistent [27]. Exercise-induced neuroprotection may involve exerkines and related signaling pathways [28].

SUMMARY OF NEUROPROTECTIVE STRATEGIES

Table 1 consolidates the strategies discussed above, and Figure 4 summarizes them as a multimodal framework. The evidence base is predominantly mechanistic and preclinical, with a smaller body of early translational work.

Approach	Reported / proposed mechanism	Evidence type	Ref.
Resveratrol	SIRT1 activation, autophagy, reduced NF- κ B signaling, mitochondrial biogenesis	Preclinical; review	[8,11,14]
Lipoic acid	Reduced inflammatory cytokines; increased glutathione	Review of experimental work	[18]
Phytic acid	Reduced oxidative damage; iron chelation	AD and PD models; review	[34]
Donepezil	Acetylcholinesterase inhibition plus anti-inflammatory and anti-apoptotic effects	Experimental	[2]
FENM	Reduced excitotoxicity; preserved synaptic density	Mouse models	[23]
Humanin	Blocks A β -induced apoptosis (caspase activation, Bax translocation)	Cell-based studies	[20]
Intranasal fatty neuropeptide	Reduced tau pathology; improved CNS delivery	Preclinical	[9]
Natural antibodies	Target amyloid assemblies; levels fall with aging and AD	Human biomarker study	[19]

Cannabinoids (CB1/CB2)	Reduced oxidative stress, excitotoxicity, and A β -associated neuroinflammation	Experimental models	[5]
Glial modulation (TREM2, RANTES, TGF- β 1)	A β clearance and plaque compaction; repair and control of inflammatory cascades	Imaging, brain tissue and preclinical	[6,7,30]
Estrogen signaling	ER α /ER β activation linked to mitochondrial function and synaptic plasticity	Review of experimental work	[25]
Transthyretin	Proteolytic activation, inhibition of fibrillation, angiogenic activity, A β binding	Preclinical	[31,32]
Geniposide	Decreased tau phosphorylation	Review of preclinical studies	[22]
Dendrobium nobile alkaloids	Antioxidant and anti-inflammatory activity	AD models; review	[33]
Multinutrient diet	Synaptic preservation; improved cerebral perfusion	Mouse model	[29]
Hidro $\text{X}^{\text{®}}$	Neuroprotection; biochemical links between TBI and AD	Experimental	[24]
Cyclodextrin	Modulation of A β aggregation and cholesterol metabolism	Cell and mouse models	[26]
Exerkines	Exercise-induced signaling pathways	Review	[28]

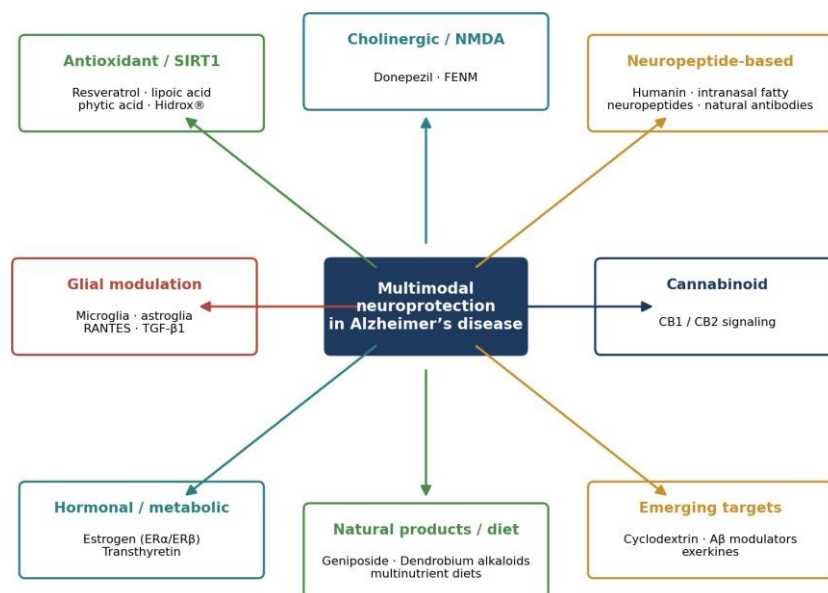


Figure 4. Multimodal framework of neuroprotective approaches in Alzheimer's disease, grouped by mechanism.

MECHANISTIC SCHEMATIC

A β accumulation
↓
ROS ↑ → Mitochondrial dysfunction
↓ Microglial activation
↓
Cytokines + NO ↑
↓
Synaptic loss → Cognitive decline

Neuroprotective interventions described in the source: antioxidants (resveratrol, lipoic acid); SIRT1 activation; TREM2 modulation; estrogen signaling; cannabinoid receptors; humanin; transthyretin–A β binding; and cyclodextrin lipid modulation.

DISCUSSION

The source manuscript argues that amyloid-centered approaches alone may be insufficient for neuroprotection in AD and emphasizes convergent pathways involving oxidative stress, inflammation, mitochondrial impairment, and metabolic dysregulation [4,27]. It identifies late-stage intervention, APOE genotype variability, phenotypic heterogeneity, and blood–brain barrier penetration as translational challenges [17]. Biomarker-driven strategies involving TREM2 imaging,

SIRT1 activation status, oxidative stress markers, and inflammatory cytokines are discussed as potential approaches to improve trial precision [30].

Several themes emerge across the reviewed literature. First, the same downstream events, namely oxidative stress, mitochondrial dysfunction, and neuroinflammation, appear repeatedly regardless of the initiating trigger, which supports targeting convergent pathways [4,10]. Second, many interventions act on more than one pathway; resveratrol, for instance, is linked to inflammatory, autophagic, and mitochondrial effects [8,14]. Third, glial cells and nitric oxide have both protective and harmful roles, so the aim is often to shift their balance rather than to block them [12,16,30].

Table 2. Translational challenges and possible responses identified in the literature.

Challenge	Why it matters	Proposed response
Late-stage intervention	Substantial neuronal loss has occurred before treatment begins [17]	Earlier, biomarker-guided enrolment when compensatory mechanisms remain [31]
APOE genotype and phenotypic heterogeneity	Patients may respond differently to the same agent [17]	Stratified trial design
Blood–brain barrier penetration	Limits brain exposure of many candidates [17]	Alternative delivery, such as the intranasal route [9]
Inconsistent translation of A β modulators	Results have not been entirely consistent [27]	Combination and multi-target approaches [4,27]
Lack of precise outcome markers	Hard to show a neuroprotective effect	TREM2 imaging, SIRT1 activation status, oxidative stress markers, cytokines [30]

Proposed translational roadmap

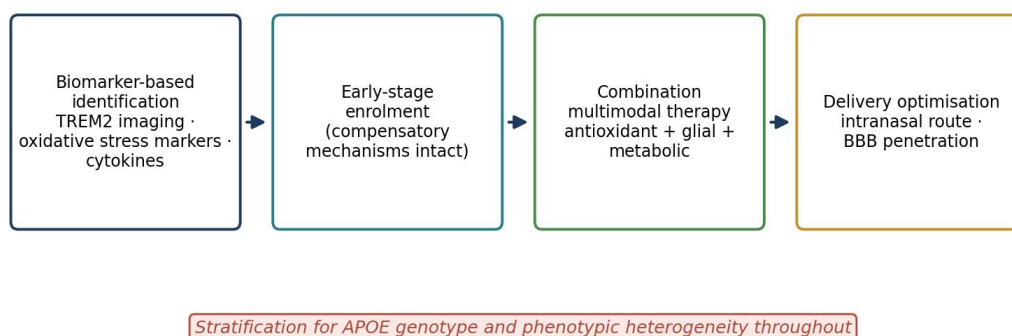


Figure 5. Proposed translational roadmap for neuroprotective therapy in Alzheimer’s disease, based on the challenges and directions discussed in this review.

LIMITATIONS

This review has several limitations. It is a narrative synthesis rather than a quantitative meta-analysis, and no formal registration was made. The included literature is dominated by in vitro and animal studies, which may not predict clinical benefit, and the quality of individual studies was appraised qualitatively. Because the review covers many mechanisms, the depth of discussion for each is limited. Findings should therefore be interpreted as hypothesis-generating.

FUTURE DIRECTIONS

Future work may prioritize (i) trials in early or prodromal AD, using biomarkers to select participants and track response; (ii) rational combinations of agents acting on complementary pathways, such as antioxidant, glial-modulating, and metabolic strategies; (iii) improved delivery routes, including intranasal administration, to overcome blood–brain barrier limits; and (iv) clarification of dose, bioavailability, and safety for natural products such as resveratrol before clinical use [4,11]. Lifestyle-related approaches, including exercise-induced signaling and multinutrient diets, may also complement pharmacological strategies [28,29].

CONCLUSION

The reviewed literature presents neuroprotection in AD as a multifactorial strategy involving oxidative, inflammatory, peptide, hormonal, and metabolic pathways [17]. Combination approaches and earlier intervention are emphasized. The source manuscript identifies intranasal neuropeptide delivery, polyphenol-mediated SIRT1 activation, and microglial immunomodulation as areas warranting further investigation, particularly during early disease stages when compensatory mechanisms may remain functional.

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