Medical Science

To Cite:
Kowalska A, Kędzierska M, Biernacki M, Gręda J, Wójcicki M. The impact of chronic oxidative stress on the development of Alzheimer's disease. *Medical Science* 2026; 30: e1ms3756
doi:

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Peer-Review History
Received: 03 August 2025
Reviewed & Revised: 18/August/2025 to 11/December/2025
Accepted: 18 December 2025
Published: 03 January 2026

Peer-review Method
External peer-review was done through double-blind method.

Medical Science
pISSN 2321–7359; eISSN 2321–7367

The impact of chronic oxidative stress on the development of Alzheimer's disease

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ABSTRACT

Imbalanced redox homeostasis, known as oxidative stress (OS), underpins the pathology of Alzheimer's disease (AD). Because of their high oxygen consumption and postmitotic state, neurons are exceptionally prone to injury by reactive oxygen species (ROS). This review aims to analyze the evidence for the role of oxidative stress in the development of AD, from its molecular basis to clinical implications. A review of the literature (including PubMed over the last 13 years) indicates that mitochondrial dysfunction, the principal source of ROS in the cell, is a central and early phenomenon that may even precede classic Aβ pathology. The stress generated enters a vicious cycle with the Aβ peptide; Aβ itself (especially in complexes with metals) can generate ROS, which in turn intensifies its aggregation. OS also promotes tau hyperphosphorylation, leading to the formation of NFTs. The effects of this cascade are multi-level: from lipid peroxidation and membrane damage (e.g., by HNE), through protein oxidation resulting in an energy crisis, to nucleic acid damage, with particular sensitivity of mtDNA. The result is a breakdown of calcium homeostasis and excitotoxicity, leading to neuronal death. Despite such a strong theoretical basis, translating this knowledge into effective clinical interventions has proven problematic. Clinical trials using antioxidants (vitamins E and C, CoQ10) have failed to show any effect on cerebrospinal fluid biomarkers or on slowing disease progression. The potential of polyphenols (e.g., curcumin, EGCG), although strong in vitro, is drastically limited by their negligible bioavailability.

Keywords: oxidative stress, Alzheimer's disease, neurodegeneration, reactive oxygen species (ROS), mitochondrial dysfunction

1. INTRODUCTION

Alzheimer's disease(AD) is a neurodegenerative disorder affecting a significant proportion of the aging population. Its pathology is characterized by two distinct hallmarks: extracellular senile plaques, composed of amyloid beta (Aβ), and intracellular neurofibrillary tangles (NFTs),formed by hyperphosphorylated tau protein (Misrani et al., 2021; Ionescu-Tucker & Cotman, 2021). Although we do not know the exact mechanism that triggers the disease, these brains also show high levels of reactive oxygen species (ROS) (Ionescu-Tucker & Cotman, 2021). This concept describes a condition in which redox homeostasis is disrupted, allowing oxidants to prevail over antioxidant defenses. Such an imbalance consequently

disrupts precise redox signaling pathways and can culminate in molecular damage (Sies, 2020).

Reactive oxygen species (ROS)themselves, however, are not intrinsically pathological entities. At low, controlled concentrations, called "oxidative eustress," they are essential for the cell and play a key role in signaling (Sies, 2020; Averill-Bates, 2024). The problem arises when ROS production becomes uncontrolled or when defense mechanisms fail. It leads to non-selective damage to key biomolecules: proteins, lipids, and nucleic acids (Averill-Bates, 2024; Misrani et al., 2021). This entire process is inextricably linked to aging. Free radicals are believed to be one of the primary drivers of aging, and oxidative stressors accumulate in our bodies throughout our lives (Ionescu-Tucker & Cotman, 2021). This is partly because mitochondria - the leading ROS factories in the cell - begin to work less efficiently with age and become damaged themselves (Misrani et al., 2021; Ionescu-Tucker & Cotman, 2021). The brain is particularly susceptible to these changes. First, it consumes up to 20% of the body's oxygen, making it a breeding ground for ROS. Second, it is rich in polyunsaturated fatty acids, which are prone to oxidation. Third, its natural antioxidant systems are relatively poor (Misrani et al., 2021). There is one more factor. Neurons are postmitotic cells – they do not divide or regenerate. This means that any damage they suffer accumulates over decades and makes them highly vulnerable to long-term exposure to oxidative stress. Therefore, today, the accumulation of oxidative damage is considered a key mechanism underlying both normal cognitive aging and AD (Ionescu-Tucker & Cotman, 2021). The aim of this paper is to review research on the role of oxidative stress in the development and progression of Alzheimer's disease.

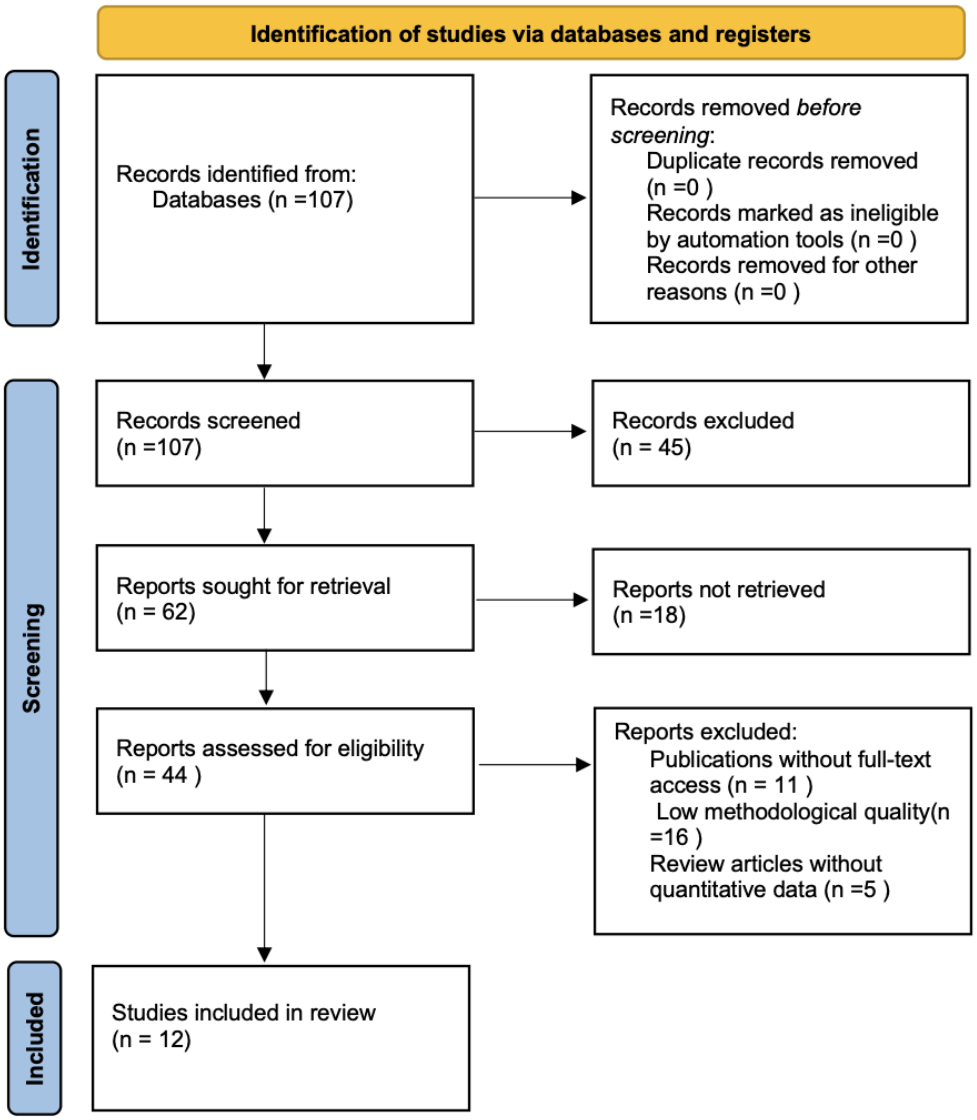

Figure 1: PRISMA flowchart

2. REVIEW METHODS

We prepared this review based on a systematic literature search in major scientific databases, including PubMed. The search process employed keywords: oxidative stress, Alzheimer's disease, neurodegeneration, reactive oxygen species, and mitochondrial dysfunction. Publications from the last 13 years, published in English or Polish, were included.

Inclusion & Exclusion

The review included systematic literature reviews that analyzed the relationship between chronic oxidative stress and the development or progression of Alzheimer's disease. Experimental and interventional studies on the effects of antioxidant therapies, lifestyle modifications that affect redox balance, and the cellular mechanisms underlying oxidative damage in the context of neurodegeneration were also included. The studies included in the review covered adult and elderly populations, as well as well-designed studies in animal models (e.g., transgenic AD mice) analyzing the molecular and cellular mechanisms of oxidative stress in the pathogenesis of AD.

The following were excluded from the analysis: case reports, editorial comments, and review articles without quantitative data, publications without full-text access, and articles of low methodological quality (e.g., no control group, small sample size <10 participants).

Study Selection & Data Extraction

The selection process was carried out by three independent authors, who evaluated titles and abstracts against pre-established criteria. In case of discrepancies, decisions were made by consensus. All data presented in this paper come from peer-reviewed scientific studies that met the above criteria.

A preliminary database search identified 107 records. 44 articles were selected for full-text evaluation. Based on inclusion and exclusion criteria, 12 publications were included in the final synthesis. The entire selection process is illustrated in the PRISMA diagram (Figure 1).

3. RESULTS & DISCUSSION

The role of reactive oxygen species (ROS) and nitrogen species (RNS)

Oxidative stress (OS) is an imbalance between oxidants and antioxidants, leading to increased production of reactive oxygen species. Increased ROS levels lead to damage to lipids, proteins, and nucleic acids, with pathological consequences (Song et al., 2021). A key source of ROS in nerve cells is the mitochondria. They produce, among other things, hydrogen peroxide (H_2O_2), which is formed by the dismutation of the superoxide anion radical ($O_2\cdot^-$) (Yin et al., 2016). Regulated levels of H_2O_2 participate in the redox regulation of cytosolic signaling pathways and nuclear transcription. Hydrogen peroxide exerts a biphasic effect on the insulin/IGF1 (IIS) signaling axis: it stimulates the pathway at physiological levels but suppresses it at excessive concentrations. Reactive nitrogen species also contribute to oxidative stress. An example of RNS is peroxynitrite, which, like $O_2\cdot^-$, can activate the NLRP3 inflammasome, a key element of the inflammatory response (Yin et al., 2016).

Mitochondrial dysfunction in neurons

Mitochondrial dysfunction and the associated cellular energy deficits are recognized as key elements in aging processes and in the pathophysiology of Alzheimer's disease. Numerous mitochondrial abnormalities are observed in the brains of AD patients, such as altered mitochondrial structure, decreased expression and activity of mitochondrial enzymes crucial for energy metabolism, reduced membrane potential and increased mitochondrial membrane permeability, and excessive ROS production (Song et al., 2021).

Postmortem studies of tissues from Alzheimer's disease patients confirm that the primary source of oxidative stress is mitochondrial dysfunction, including defects in the tricarboxylic acid (TCA) cycle and abnormalities in the electron transport chain and oxidative phosphorylation (OXPHOS) (Yin et al., 2016). Furthermore, it is associated with the direct effect of beta-amyloid (Aβ) on mitochondrial oxidative phosphorylation. Aβ has been shown to inhibit the activity of mitochondrial complexes I and IV (Yin et al., 2016).

Defense mechanisms: enzymatic and non-enzymatic

Nerve cells possess complex defense systems protecting them from oxidative stress. The key non-enzymatic mechanism is glutathione (GSH), which is described as the primary free radical scavenger in the brain, is synthesized in the cytosol, and then transported into the mitochondria (Yin et al., 2016; Song et al., 2021). In the brain, ROS are eliminated by GSH in a chemical reaction that converts reduced GSH into its oxidized form (GSSG) (Song et al., 2021). The maintenance of the mitochondrial redox state depends on the balance between the production of hydrogen peroxide and its enzymatic reduction to water. This process is mainly controlled by two enzymatic systems. The first one, the glutathione (GSH) dependent system, involves enzymes from the glutathione peroxidase (GPx) family, which use GSH as a cofactor to reduce H_2O_2 (Yin et al., 2016).

The main mitochondrial isoforms are GPx1 (located in the matrix) and GPx4 (in the intermembrane space). Reduced GSH is then regenerated from GSSG by the enzyme glutathione reductase (Yin et al., 2016). The second, however, is the thioredoxin (Trx)-supported peroxiredoxin system. In this system, electrons are transferred from NADPH to thioredoxin-2 (Trx2) with the involvement of thioredoxin reductase 2 (TrxR2). Trx2 then reduces peroxiredoxins (Prx), which directly neutralize H_2O_2 (Yin et al., 2016). The two key mitochondrial peroxiredoxins are Prx3 and Prx5, which reduce H_2O_2, organic hydroperoxides, and peroxynitrite. Prx3 itself is responsible for catalyzing the removal of up to 90% of mitochondrial H_2O_2 (Yin et al., 2016).

The role of oxidative stress in the pathogenesis of Alzheimer's disease

There is growing evidence that this mechanism plays a key role in the development of neurodegenerative diseases, including Alzheimer's disease (AD). When excessive ROS production is accompanied by disturbances in metal homeostasis and antioxidant system failure, there is a direct impairment of neuronal function. Synaptic dysfunction, followed by cognitive deficits, is a direct consequence of the accumulation of these damages at the molecular level. Reactive oxygen species play a key role in promoting the main pathological features of AD. It has been shown that oligomeric Aβ itself, especially in complexes with transition metals such as copper (Cu) and iron (Fe), is capable of generating ROS, leading to a vicious cycle in which Aβ accumulation exacerbates oxidative stress, which in turn may accelerate peptide aggregation (Ionescu-Tucker & Cotman, 2021; Tönnies & Trushina, 2017).

Oxidative stress is also a factor promoting tau protein hyperphosphorylation. As a result, it detaches from microtubules, which disrupts their stability and initiates the formation of intracellular neurofibrillary tangles (NFTs). It is generally agreed that only the coaction between Aβ and tau pathologies triggers the complete neurodegenerative process (Tönnies & Trushina, 2017).

ROS have harmful effects on many key biomolecules in neurons. Cell membranes appear to be particularly vulnerable to attack due to the ability of the hydrophobic Aβ peptide to deposit in their structure. The presence of Aβ initiates a process of lipid peroxidation, which in turn generates secondary, highly reactive aldehydes, such as 4-hydroxy-2-nonenal (HNE). These compounds are capable of covalently modifying proteins, which can be done profoundly.

Damage to the cell and mitochondria membranes impair ion transport, cell signaling, and organelle integrity (Butterfield & Halliwell, 2019; Tönnies & Trushina, 2017). ROS also leads to protein oxidation, as indicated by an increase in protein carbonyl levels. This damage affects key enzymes involved in energy metabolism, including glycolysis and tricarboxylic acid cycle enzymes, resulting in reduced ATP production and an energy crisis in neurons (Butterfield & Halliwell, 2019). At the same time, damage to nucleic acids occurs, both nuclear and mitochondrial DNA (mtDNA). mtDNA is ten times more susceptible to oxidative damage due to its proximity to the source of ROS production and the lack of protective histones (Ionescu-Tucker & Cotman, 2021). This damage, in the form of single- and double-strand breaks (SSBs and DSBs), disrupts gene transcription and genome integrity, which are early events in the progression of AD.

A profound and ultimate consequence of oxidative stress is also the disruption of calcium homeostasis. When reduced ATP levels prevent neurons from maintaining normal ion gradients, and Aβ simultaneously induces Ca^{2+} influx through NMDA receptors, the cell enters a state of excitotoxicity. This toxic calcium overload itself becomes another blow to the mitochondria, escalating ROS production. This closes a self-perpetuating pathological cycle, the result of which is synaptic dysfunction, neuronal death, and progressive dementia characteristic of Alzheimer's disease (Tönnies & Trushina, 2017; Butterfield & Halliwell, 2019).

The role of antioxidant interventions in Alzheimer's disease

Oxidative stress is now widely recognized as one of the key pathogenic factors in Alzheimer's disease (AD), which forms the basis for research into new treatment strategies (Jomova et al., 2023; Juszczyk et al., 2021). has led directly to interest in the therapeutic potential

of antioxidants, including polyphenols, coenzyme Q10, and vitamins E and C (Juszczyk et al., 2021; Stefaniak et al., 2022). Although the theoretical rationale for such interventions seems strong, their translation into clinical practice has proven to be very problematic.

Vitamins E and C

The study of these two molecules stems from their specific interaction. Vitamin E (αtocopherol) is an essential lipophilic compound that protects cell membranes from oxidation. Vitamin C, on the other hand, as a hydrophilic antioxidant, supports this process – it has been shown that, at least in vitro, it can regenerate tocopherol. As the main fat-soluble antioxidant, α-tocopherol (vitamin E), is responsible for protecting cell membranes from Peroxidation.

Vitamin C, a hydrophilic antioxidant, complements this mechanism, among other things, through its proven in vitro ability to regenerate tocopherol (Jomova et al., 2023; Juszczyk et al., 2021). Therefore, their combined action should alleviate oxidative stress driven by the β-amyloid (Aβ) peptide. Initial optimism, which drew strength from epidemiological data (such as the Rotterdam Study) (Stefaniak et al., 2022; Juszczyk et al., 2021) and from individual studies suggesting a slowdown in the disease, has been greatly dampened.

More recent analyses, using Mendelian randomization, for example, have failed to confirm a direct link between vitamin C levels and AD risk (Stefaniak et al., 2022). More problematically, key clinical trials have failed to show that supplementation affects central biomarkers of pathology- namely, Aβ and tau levels in cerebrospinal fluid (CSF) (Rauchová, 2021). These studies revealed a frustrating discrepancy: even when vitamin concentrations in CSF were successfully increased. There was no real improvement in cognitive function in patients (Juszczyk et al., 2021). Today, this gap between promising laboratory results and clinical failure is primarily attributable to barriers to the transport and effective use of these molecules within the brain (Jomova et al., 2023).

Table 1: Summary of key findings on the role of oxidative stress in Alzheimer's disease

Research Category	**Key Findings from Literature Review**
Primary Sources of ROS	Mitochondrial dysfunction is identified as the central and early source of ROS (Song et al., 2021).
Molecular Targets of Damage	Lipids: Aβ initiates lipid peroxidation, leading to membrane damage and production of toxic aldehydes like HNE (Butterfield & Halliwell, 2019). Proteins: Oxidation of enzymes involves in energy metabolism (glycolysis, TCA cycle) leads to cellular energy crisis (Butterfield & Halliwell, 2019). Nucleic Acids: Mitochondrial DNA (mtDNA) is exceptionally vulnerable to oxidative damage due to lack of histones and proximity to ROS source (Ionescu-Tucker & Cotman, 2021).
Pathological Consequences	Oxidative stress promotes tau hyperphosphorylation (formation of NFTs) and exacerbates Aβ aggregation (Tönnies & Trushina, 2017). The ultimate consequence is the disruption of calcium homeostasis, excitotoxicity, and neuronal death (Butterfield & Halliwell, 2019).
Clinical Interventions Antioxidants Vit E, C, CoQ10	Clinical trials failed to demonstrate significant effects on cognitive decline or CSF biomarkers, despite theoretical rationale (Stefaniak et al., 2022; Rauchová, 2021; Juszczyk et al., 2021).
Potencial of Polyphenols	Compounds like curcumin and EGCG show strong anti-amyloidogenic potential in vitro, but their therapeutic use is limited by negligible bioavailability (Jomova et al., 2023; Stefaniak et al., 2022).

Coenzyme Q10 (CoQ10)

Coenzyme Q10 (CoQ10), a key component of the respiratory chain and an antioxidant that protects membranes, has been studied equally intensively. As with vitamins, studies in animal models have been promising; CoQ10 supplementation (including new formulations such as Ubisol-Q10) inhibited Aβ pathologies and improved memory in mice (Rauchová, 2021; Juszczyk et al., 2021).

Transferring these results to the clinical setting has proven problematic. There is not even a fundamental consensus on the actual status of CoQ10 in the brains of AD patients, elevated, decreased, and unchanged levels have all been reported (Rauchová, 2021; Jomova et al., 2023). This uncertainty is reflected in the results of intervention studies. Both trials using an analog (idebenone) and CoQ10 itself have failed to provide evidence of a slowdown in cognitive decline. Newer strategies, such as the mitochondria-targeted MitoQ, which is effective in animal models (Rauchová, 2021), may have some potential.

Polyphenols

There are also high hopes for plant polyphenols, which have the potential to delay cognitive decline. Their advantage is that they work in multiple ways: they can chelate (bind) metals, which inhibits the harmful Fenton reaction, and they also have anti-inflammatory and antioxidant effects. There is a lot of evidence from preclinical (laboratory) studies. Curcumin and resveratrol show anti-amyloidogenic potential, i.e., they can inhibit the formation of amyloid (Jomova et al., 2023; Juszczyk et al., 2021). EGCG, known from green tea, inhibits β-amyloid aggregation and may also affect tau protein pathology (Stefaniak et al., 2022; Juszczyk et al., 2021).

Unfortunately, all this promising in vitro potential faces a fundamental pharmacological barrier: these compounds have negligible bioavailability. This means that they are poor absorbed or rapidly metabolized. This problem is particularly well described in the case of curcumin. For this reason, despite strong evidence from basic research. The real therapeutic value of polyphenols for patients remains unproven (Jomova et al., 2023).

Further research is needed, focused on finding a way to deliver them more effectively to the body (Jomova et al., 2023; Stefaniak et al., 2022). Table 1 provides a comparative overview of the established molecular mechanisms of oxidative stress and the corresponding limitations of antioxidant therapies.

4. CONCLUSION

Oxidative stress in neurons occurs when the production of reactive oxygen and nitrogen species (ROS/RNS) exceeds the body's defense capabilities. Mitochondria are a key source of ROS, and their dysfunction is considered an early and central mechanism of neurodegeneration, driving further production of oxidants. Although ROS destroy biomolecules (lipids, proteins, DNA) in excess, they serve as signaling molecules at low concentrations. The cell defends itself using non-enzymatic (GSH) and enzymatic (GPx, Trx, Prx) systems, which are NADPH-dependent.

This mechanism is fundamental in Alzheimer's disease (AD). It is believed that mitochondrial dysfunction (the source of ROS) can initiate the disease even before the appearance of Aβ. Stress and the Aβ peptide enter a vicious cycle, mutually driving their production and promoting tau hyperphosphorylation (NFT). This leads to lipid peroxidation, an energy crisis (through protein oxidation), and DNA damage (especially mtDNA). The result is a breakdown in calcium homeostasis: ATP deficits and Ca^{2+} influx (induced by Aβ) lead to excitotoxicity, synapse and neuron death, which explains cognitive deficits.

Despite such a strong theoretical basis, antioxidant interventions have failed in clinical trials. Although epidemiological and preclinical data were promising, key trials with high doses of vitamins E and C and coenzyme Q10 have not shown efficacy in inhibiting cognitive decline or changing AD biomarker levels (Aβ, tau). On the other hand, although polyphenols show strong effects in laboratory studies, their practical use is limited due to low bioavailability. So far, antioxidant treatments for Alzheimer’s disease have not demonstrated apparent effectiveness.

Acknowledgments

The authors have no acknowledgments to disclose.

Author's contribution

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Writing-review and editing: Michał Biernacki, Michał Wójcicki, Justyna Gręda.
Visualization: Agnieszka Kowalska, Milena Kędzierska.

All authors have read and agreed with the published version of the manuscript.

Informed consent

Not applicable.

Ethical approval

Not applicable. This article does not contain any studies with human participants or animals performed by any of the authors.

Funding

This research did not receive any external funding like specific grant from funding agencies in the public, commercial, or nonprofit sectors.

Conflict of interest

The authors declare that they have no conflicts of interests, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data and materials availability

All data associated with this study will be available based on reasonable request to the Corresponding Author.

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