International Journal of Applied and Traditional Medicine, 2026, 13-25

https://publishingjournals.org/ijatm
Volume: 3 | Number: 1 (2026) January

Oxidative Stress, Inflammatory Mediators, and Dementia: An Integrative Review

Komang Trisna Sumadewi*, Saktivi Harkitasari, Fransiscus Fiano Anthony Kerans
Universitas Warmadewa

***Correspondence:** Komang Trisna Sumadewi
Email : trisna.sumadewi@warmadewa.ac.id

Received: 26-11-2025
Accepted: 26-12-2025
Published: 07-01-2026

ABSTRACT

This study aims to provide an integrative understanding of the complex relationship between oxidative stress, inflammatory mediators, and dementia, particularly Alzheimer's disease. Employing a qualitative descriptive design through a library-based study, this research systematically analyzes recent peer-reviewed literature from 2015 to 2025. Data were collected through comprehensive literature tracing and document analysis of academic articles, theoretical reviews, and clinical findings related to oxidative and inflammatory mechanisms in neurodegeneration. The data were processed through stages of thematic identification, reduction, categorization, and inductive interpretation to derive a holistic understanding of the phenomenon. The results reveal that oxidative stress and neuroinflammation act as interdependent and self-reinforcing mechanisms in the pathogenesis of dementia. Mitochondrial dysfunction, excess reactive oxygen species (ROS), and chronic activation of inflammatory pathways—particularly NLRP3 and NF-κB—were identified as core contributors to neuronal degeneration and cognitive decline. Furthermore, evidence highlights the critical role of systemic inflammation in amplifying central nervous system damage through blood–brain barrier disruption. These findings emphasize the need for multi-targeted therapeutic approaches integrating antioxidant and anti-inflammatory strategies, rather than isolated single-pathway treatments. The study contributes to the advancement of theoretical frameworks linking oxidative and inflammatory pathways in neurodegeneration and underscores their relevance for preventive and therapeutic innovation. In conclusion, this integrative review enriches the understanding of dementia as a multi-system disorder, providing conceptual and practical insights for future translational research in neurodegenerative disease management.

Keywords:
Oxidative Stress, Inflammation, Dementia, Alzheimer's Disease, Neurodegeneration.

INTRODUCTION

Due to its increasing frequency and significant socioeconomic impact, dementia, which includes Alzheimer's disease (AD) and vascular dementia (VaD), has emerged as one of the most important worldwide health issues of the twenty-first century. The incidence of dementia is expected to triple by 2050 as the world's population ages, underscoring the critical need to understand its underlying molecular causes (Bai et al, 2022). The pathogenesis of neurodegeneration has been linked to a number of variables, including oxidative stress and chronic inflammation (Plascencia-Villa & Perry, 2023). A more comprehensive knowledge of these two processes' roles in dementia is necessary because their interaction creates a self-sustaining cycle that speeds up neuronal damage and cognitive decline.

Lipids, proteins, and DNA within neuronal cells are oxidized as a result of oxidative stress, which is caused by an imbalance between the generation of reactive oxygen species (ROS) and antioxidant defense systems (Fanlo-Ucar et al, 2024). This redox imbalance is a major cause of cellular malfunction that interferes with synaptic communication and encourages neurotoxicity, rather than only a consequence of aging (Perluigi et al, 2024). At the same time, neuroinflammation, which is mostly caused by microglia and astrocytes, increases oxidative damage by releasing proinflammatory cytokines like TNF-α, IL-1β, and IL-6 over time (Altahrawi et al, 2025). The progressive loss of cognitive abilities typical of dementia is caused by a harmful environment orchestrated by several systems working together.

According to recent clinical and experimental research, people with dementia and moderate cognitive impairment (MCI) have markedly higher levels of inflammation and oxidative stress biomarkers (Ahmad et al, 2024). These results lend credence to the theory that oxidative and inflammatory processes are early triggers in neurodegenerative cascades rather than only downstream effects (Güler, 2025). Furthermore, the identification of genetic variations in inflammatory and oxidative stress pathways, including NFE2L2 and IL1B, highlights the possible genetic impact on a person's vulnerability to dementia (Vogrinc et al, 2023).

A paradigm change in the study of neurodegenerative diseases is reflected in the increasing amount of data connecting inflammation and oxidative stress to dementia. Current viewpoints highlight a multidimensional paradigm where redox imbalance and neuroinflammation play key roles, rather than concentrating just on amyloid-β and tau pathology (Novoa et al, 2022). This change provides a more comprehensive foundation for figuring out new treatment targets and comprehending illness mechanisms. However, because these molecular processes are intricate and interrelated, clinical translation is still difficult despite much research (Scarian et al, 2024).

Despite their theoretical promise, current antioxidant treatments have had little success in clinical trials (Pappolla et al, 2024). Poor bioavailability, improper delivery timing, and the incapacity to target certain molecular causes of oxidative stress have all been blamed for these therapies' ineffectiveness (Hsu et al, 2023). In a similar vein, anti-inflammatory

medications frequently fall short of successfully reducing neuroinflammation without causing systemic immunosuppression (Altahrawi et al, 2025). These drawbacks emphasize the need for a more comprehensive treatment strategy that concurrently targets oxidative and inflammatory factors.

Simultaneously, studies have started to investigate the possibilities of new therapeutic approaches such probiotics, mitochondrial antioxidants, and lifestyle modifications that alter the gut-brain axis (Hsu et al, 2023). By restoring systemic homeostasis, these tactics seek to lessen oxidative and inflammatory loads. The growing understanding that dementia is a systemic condition impacted by metabolic, vascular, and immunological aspects is consistent with such multifaceted therapies (Fanlo-Ucar et al, 2024). For the purpose of developing preventive and therapeutic frameworks, it is crucial to investigate the convergence of these factors.

Biomarkers of inflammation and oxidative stress are showing promise as diagnostic tools for disease monitoring and early diagnosis (Ahmad et al, 2024). The severity of the disease has been linked to elevated plasma levels of cytokines, protein carbonyls, and isoprostanes, indicating their potential as clinical markers for disease progression (Güler, 2025). These discoveries open the door to individualized treatment in dementia care by improving diagnostic accuracy and offering insights into patient-specific pathophysiological patterns.

Despite these developments, there are still a number of significant knowledge gaps. There is ongoing discussion over the temporal dynamics of oxidative stress and inflammation, including whether one causes or exacerbates the other (Novoa et al, 2022). Furthermore, more research is needed to understand how genetic predisposition and environmental triggers interact to modulate these processes (Vogrinc et al, 2023). Developing focused therapies that can stop or reverse neurodegenerative trends requires addressing these issues.

Finding trustworthy, non-invasive biomarkers that appropriately reflect disease of the central nervous system is another urgent problem (Ahmad et al, 2024). Although useful, the current biomarkers for cerebrospinal fluid (CSF) are intrusive, which has led to the hunt for peripheral indications that may be measured using blood or saliva samples (Güler, 2025). By combining these indicators with neuroimaging techniques, a multifaceted evaluation of disease activity may be possible.

Furthermore, there is still much to learn about the spatial specificity of oxidative and inflammatory damage across brain networks (Perluigi et al, 2024). According to new research, hippocampus and cortical areas are especially susceptible to cytokine-induced toxicity and redox imbalance (Plascencia-Villa & Perry, 2023). The variability of cognitive symptoms seen in various dementia subtypes may be explained by these region-specific susceptibilities.

With dementia cases expected to climb sharply in low- and middle-income nations, the worldwide demographic shift toward an older population heightens the urgency of this problem (Bai et al, 2022). Strategies that not only reduce symptoms but also stop the start of disease by modifying oxidative and inflammatory pathways are required due to the

increasing burden of healthcare (Altahrawi et al, 2025). Therefore, it is crucial from a scientific and public health perspective to clarify the molecular foundations of these processes.

The goal of this integrated review is to summarize the present understanding of the relationship between oxidative stress, inflammatory mediators, and dementia, emphasizing new developments, persistent difficulties, and promising paths. This article attempts to offer a thorough framework for comprehending how redox and inflammatory dysregulation contribute to neurodegeneration by connecting molecular insights with clinical implications. The review also looks at possible biomarkers and pharmacological targets that might change the way dementia is diagnosed and treated today.

Theoretically, by highlighting the reciprocal relationships between oxidative stress and inflammation, this work advances models of neurodegenerative diseases. In practical terms, it offers evidence-based viewpoints that can guide the development of innovative treatments and biomarker-driven clinical trials. The key to improving preventive, diagnostic, and therapeutic approaches in dementia research and care ultimately lies in comprehending the integrative role of oxidative and inflammatory pathways.

METHODS

This article used a library-based study (studi pustaka) and a qualitative research design with a descriptive method. The qualitative-descriptive framework was chosen to offer a methodical, rational, and comprehensive investigation of the ways in which oxidative stress and inflammatory mediators contribute to the development and course of dementia, including Alzheimer's disease (AD) and vascular dementia (VaD). Through interpretation rather than numerical analysis, qualitative inquiry enables the researcher to capture intricate linkages and theoretical foundations (Bingham, 2023) (Pratt, 2025). A comprehensive, nuanced understanding of the biological and pathophysiological systems included in this integrative review is made possible by the descriptive method, which emphasizes accurate portrayal of actual occurrences without alteration (Abraham, 2024) (Doyle, 2019).

Peer-reviewed journal articles, systematic reviews, and conceptual studies with an emphasis on oxidative stress, inflammatory mediators, and dementia made up the majority of the study's data sources. Official health organization reports, up-to-date textbooks, and reliable internet databases of scientific research were further supporting resources. In order to ensure that only reliable and authoritative academic sources were used, articles were chosen from respectable databases and journals like Ageing Research Reviews, Physiological Reviews, International Journal of Molecular Sciences, and Journal of Alzheimer's Disease (Bai et al, 2022) (Perluigi et al, 2024) (Plascencia-Villa & Perry, 2023) (Scarian et al, 2024). The reliability and academic rigor of the data gathered were ensured by the focus on peer-reviewed sources.

The methodical procedure of document examination and literature tracing was followed by data collection techniques. To find research on oxidative stress, inflammation, and dementia that was published between 2015 and 2025, the researcher thoroughly searched scholarly databases. A mix of keywords such as "oxidative stress,"

"neuroinflammation," "Alzheimer's disease," and "vascular dementia" were used to develop the selection criteria. Empirical and theoretical works that addressed biochemical mechanisms, clinical consequences, and therapeutic approaches were included in the literature screening process (Altahrawi et al, 2025) (Fanlo-Ucar et al, 2024) (Novoa et al, 2022). Grey literature, non-peer-reviewed publications, and research unrelated to neurodegenerative pathways were all excluded.

A qualitative thematic framework encompassing many sequential stages—identification of themes, data reduction, conceptual classification, and inductive synthesis—was used in the data analysis process. In order to extract important themes like reactive oxygen species (ROS) imbalance, cytokine dysregulation, and neuronal injury, a thorough assessment of all gathered materials was the first step in the thematic identification process. After that, the information was grouped into thematic groupings, including biomarkers, inflammatory mediators, oxidative stress processes, and treatment approaches (Bingham, 2023) (Fife, 2024) (Vila-Henninger, 2022). Lastly, general inferences on the integrative links between oxidative and inflammatory pathways were drawn using an inductive reasoning technique.

This study used a number of quality control procedures in accordance with qualitative research standards to preserve the validity and reliability of the results. Cross-verifying results from other scientific articles discussing comparable mechanisms and consequences was how source triangulation was carried out (Belotto, 2018) (Kalpokaite, 2018). The main source of validation was peer-reviewed academic publications, which reduced prejudice and guaranteed conceptual credibility. Furthermore, the auditability and repeatability of interpretations were improved by transparency in the data analysis process—documenting coding choices and theme refining (Bingham, 2023) (Pratt, 2025).

Finally, this article's goal of synthesizing diverse discoveries and producing a comprehensive picture of the molecular pathophysiology of dementia is in line with the selected qualitative-descriptive library research approach. This approach facilitates thorough theorization and contextual interpretation by combining results from biochemistry, neuroscience, and clinical research (Bandaranayake, 2024) (Jimenez, 2024) (Togia, 2017). The resulting analysis offers a solid conceptual basis for future research and clinical innovation in addressing oxidative and inflammatory factors to dementia, in addition to reflecting the current state of scientific knowledge.

RESULTS

The results of this integrated literature analysis highlight the complex role that inflammatory mediators and oxidative stress play in the pathophysiology of dementia, especially Alzheimer's disease (AD). Both mechanisms show up as key and interrelated causes of synaptic dysfunction, neuronal death, and cognitive decline in the reviewed literature. Research continuously shows that oxidative imbalance and neuroinflammation are mutually reinforcing mechanisms that sustain neurodegenerative cascades rather than separate occurrences (Bai et al, 2022) (Perluigi et al, 2024) (Plascencia-Villa & Perry, 2023).

These results are systematically described in the ensuing subsections, with a focus on molecular pathways, biochemical mechanisms, and possible therapeutic implications.

Dementia and Oxidative Stress

One of the most well-known pathogenic mechanisms in AD and associated dementias is oxidative stress. Because of its high oxygen consumption and high lipid content, the brain is particularly vulnerable to oxidative damage caused by reactive oxygen species (ROS) (Bai et al, 2022) (Perluigi et al, 2024). According to recent research, ROS-mediated oxidation of lipids, proteins, and nucleic acids causes structural and functional abnormalities in neurons, which ultimately result in synapse loss and apoptosis (Birla et al, 2020) (Rummel & Butterfield, 2021). This imbalance is made worse by mitochondrial dysfunction, a significant source of ROS, and neurotoxicity is increased by compromised antioxidant defenses, such as decreased glutathione activity (Bhatia & Sharma, 2020) (Tamagno et al, 2021).

Table 1. Summarizes Key Findings Linking Oxidative Stress Mechanisms To Dementia Progression, Illustrating The Convergence Of ROS Overproduction, Mitochondrial Dysfunction, And Metal Ion Dysregulation

Mechanism	Effect on Neuronal Function	Key References
Reactive oxygen/nitrogen species (ROS/RNS)	Protein oxidation, DNA damage, lipid peroxidation	(Bai et al, 2022) (Pappolla et al, 2024)
Mitochondrial dysfunction	Energy metabolism disruption, neuronal apoptosis	(Bhatia & Sharma, 2020) (Rummel & Butterfield, 2021)
Impaired antioxidant defense	Reduced glutathione and SOD activity	(Birla et al, 2020) (Tamagno et al, 2021)

Particularly important is the connection between oxidative stress and the two main AD pathology, tau tangles and amyloid-β (Aβ) plaques. Both Aβ aggregation and tau hyperphosphorylation can be preceded and encouraged by oxidative stress, starting a feedback loop that maintains neurodegeneration (Bai et al, 2022) (Perluigi et al, 2024). This supports the idea that oxidative imbalance is an upstream event in AD progression rather than just a consequence of neuronal damage.

Neuroinflammation and Inflammatory Mediators

Another significant pathogenic axis in dementia is neuroinflammation. Proinflammatory cytokines such IL-1β, IL-6, and TNF-α, as well as chemokines and ROS, are released when microglia and astrocytes are activated, which leads to neuronal stress and death (Leng & Edison, 2020) (Novoa et al, 2022). Studies consistently demonstrate the dual nature of glial activation, which is initially protective but subsequently neurotoxic (De Oliveira et al, 2021) (Dhapola et al, 2021). Inflammatory reactions and cell death are increased when the NLRP3 inflammasome and NF-κB signaling pathway are overactivated (Tamagno et al, 2021) (Thakur et al, 2022).

Additionally, evidence suggests that by weakening the blood-brain barrier (BBB) and increasing cytokine influx into the central nervous system, systemic inflammation can worsen neurodegeneration (Lopez-Rodriguez et al, 2021) (Xie et al, 2022). Peripheral injuries cause inflammatory amplification, which quickens the pathogenic cascade and deteriorates

cognitive results (Walker et al, 2023). These results demonstrate that dementia is impacted by systemic immunological dysregulation in addition to brain disease.

Oxidative Stress and Neuroinflammation Interaction

The research shows that oxidative stress and neuroinflammation are mutually dependent. ROS cause microglia to release inflammatory mediators, and cytokines cause neurons to produce more ROS, creating a vicious cycle (Tadokoro et al, 2020) (Tamagno et al, 2021). Aβ and tau aggregates that sustain glial activation further intensify this interaction (Novoa et al, 2022). A mechanistic explanation for gradual cognitive decline is provided by such molecular synergy, which speeds up synaptic failure and neuronal death (Bai et al, 2022) (Plascencia-Villa & Perry, 2023).

Therapeutic Perspectives and Comparative Results

Despite the poor clinical efficacy of conventional antioxidant and anti-inflammatory medicines, new research suggests more focused approaches. The potential of novel medicines to modify oxidative and inflammatory pathways is being studied, including NLRP3 inhibitors, mitochondrial protectants, and PPAR-γ agonists (Dhapola et al, 2021) (Walker et al, 2023). Furthermore, compared to single-target therapies, combination therapy strategies that concurrently promote mitochondrial biogenesis and reduce chronic inflammation exhibit more promise (Tadokoro et al, 2020) (Tamagno et al, 2021).

Newer research indicates that oxidative stress and inflammation are primary and synergistic drivers of neurodegeneration (Rummel & Butterfield, 2021) (Twarowski & Herbet, 2023), a multi-system disorder that integrates neuronal, metabolic, and immune components, in contrast to previous studies that saw them as secondary phenomena.

Synthesis of Key Results

Overall, the findings support the idea that inflammatory mediators and oxidative stress play a major role in the pathophysiology of dementia. Their interconnectedness offers novel avenues for biomarker identification and therapeutic intervention in addition to explaining the molecular underpinnings of neuronal injury. In line with the objectives of precision medicine, elevated oxidative and inflammatory markers may function as diagnostic markers for early-stage cognitive impairment (Leng & Edison, 2020) (Perluigi et al, 2024). The necessity for integrated, multi-targeted approaches in the fight against dementia is thus highlighted by the convergence of molecular, systemic, and clinical evidence.

DISCUSSION

A thorough theoretical and empirical knowledge of how these systems interact in neurodegenerative processes is provided by the synthesis of recent research on oxidative stress and inflammatory mediators in dementia. The results confirm that oxidative damage and neuroinflammation are both primary, self-reinforcing drivers of dementia pathogenesis, especially in Alzheimer's disease (AD), rather than just secondary outcomes. Theoretically, these findings support the neuroinflammatory-oxidative cascade hypothesis, which holds that long-term oxidative imbalance triggers inflammatory signaling pathways that exacerbate brain damage (Bai et al, 2022) (Leng & Edison, 2020) (Perluigi et al, 2024).

Theoretical Integration and Mechanistic Insight

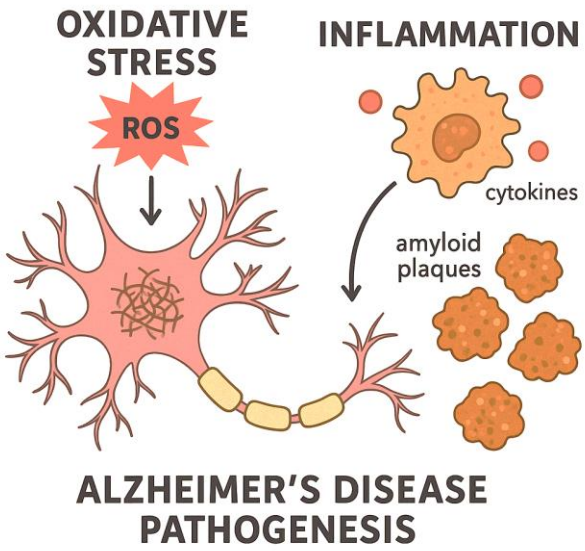

Figure 1. Theoretical Integration and Mechanistic Insight

The findings support well-established hypotheses of oxidative stress-mediated neurodegeneration, which highlight reactive oxygen species (ROS) and mitochondrial dysfunction as key disruptors of neuronal homeostasis (Bhatia & Sharma, 2020) (Rummel & Butterfield, 2021). Because of its high metabolic rate and reliance on oxygen, the brain is vulnerable to oxidative damage, which encourages protein and lipid peroxidation (Birla et al, 2020). These oxidative mechanisms demonstrate that oxidative stress precedes typical AD symptoms by accelerating tau hyperphosphorylation and amyloid-β (Aβ) aggregation (Bai et al, 2022) (Tamagno et al, 2021). The mitochondrial cascade hypothesis, which has long proposed that cellular energy failure causes dementia, is further supported by the observed mitochondrial degradation and compromised antioxidant responses (Perluigi et al, 2024).

Neuroinflammation is reaffirmed as a major pathogenic element in AD in tandem with oxidative imbalance. Cytokines including IL-1β, IL-6, and TNF-α are secreted in excess when microglia and astrocytes are activated, which interferes with synaptic communication and neuronal survival (Leng & Edison, 2020) (Novoa et al, 2022). Numerous research' findings indicate that inflammatory mediators may initially provide protection by aiding in the removal of Aβ) (nevertheless, long-term activation turns harmful and causes persistent damage to neurons (De Oliveira et al, 2021) (Thakur et al, 2022). Recent molecular studies have shown that intracellular stress signaling promotes inflammation and apoptosis by activating the NLRP3 inflammasome and NF-κB signaling pathways (Dhapola et al, 2021) (Tamagno et al, 2021).

Systemic Implications and Interdependence

According to an integrative interpretation of the reviewed data, oxidative stress and inflammation are related in both directions. A vicious pathogenic cycle is created when

proinflammatory cytokines increase ROS production and ROS function as signaling molecules that cause microglial activation (Novoa et al, 2022) (Tadokoro et al, 2020). Cumulative neuronal death and progressive cognitive impairment result from this cyclical interaction (Plascencia-Villa & Perry, 2023) (Twarowski & Herbet, 2023). Additionally, research shows that systemic inflammation—caused by vascular dysfunction or peripheral infections—can worsen neurodegeneration by compromising the blood-brain barrier (BBB), which permits peripheral immune cells and inflammatory mediators to enter the brain (Lopez-Rodriguez et al, 2021) (Walker et al, 2023).

This holistic approach has significant theoretical implications. Instead than viewing dementia as a condition just affecting the central nervous system, it presents it as a multi-system disorder. By incorporating immunological and metabolic aspects, this paradigm change broadens the field's understanding of AD and informs the creation of comprehensive treatment strategies (Xie et al, 2022).

Comparative Perspectives and Knowledge Development

Current study offers a more comprehensive model that includes redox and inflammatory dysregulation as key pathways in dementia, in contrast to previous literature that mostly focused on amyloid and tau hypotheses (Bai et al, 2022) (Perluigi et al, 2024). This shift represents a theoretical advancement in the knowledge of AD pathogenesis, suggesting that oxidative-inflammatory crosstalk is both a cause and an effect of amyloid-β buildup (Tamagno et al, 2021). Additionally, results from molecular and clinical research show that single-target treatments like conventional antioxidants and NSAIDs are insufficient, highlighting the need for multi-targeted therapies that concurrently address oxidative and inflammatory processes (Dhapola et al, 2021) (Walker et al, 2023).

Recent developments in experimental therapies, such NLRP3 inhibitors, PPAR-γ agonists, and enhancers of mitochondrial biogenesis, offer intriguing approaches to reduce cytokine-mediated neurotoxicity and ROS generation (Tadokoro et al, 2020) (Thakur et al, 2022). But despite these promising findings, pharmacokinetic hurdles, disease progression heterogeneity, and patient response variability continue to hinder translation into successful therapeutic outcomes (Novoa et al, 2022) (Tamagno et al, 2021).

Important Restrictions and Prospects

Although the studied literature offers a thorough insight, a number of limitations should be acknowledged. The majority of research is based on preclinical or in vitro models, which do not accurately reflect the complexity of human neurodegeneration despite being mechanistically instructive (Bai et al, 2022) (Rummel & Butterfield, 2021). Furthermore, the development of universal therapeutic benchmarks and diagnostic consistency are complicated by individual differences in oxidative and inflammatory indicators (Perluigi et al, 2024). There is also a lack of longitudinal human studies linking oxidative and inflammatory biomarkers with disease progression, which restricts the predictive power of current findings (Lopez-Rodriguez et al, 2021).

In order to clarify the temporal links between oxidative and inflammatory changes, future research should give priority to integrative longitudinal investigations that integrate neuroimaging, omics technologies, and molecular assays. Additionally, investigating

precision medicine frameworks customized to each patient's redox and immunological profile may enhance treatment results and diagnostic accuracy (Leng & Edison, 2020) (Walker et al, 2023).

Research and Clinical Practice Consequences

This synthesis concludes by highlighting the interdependence and targetability of oxidative stress and inflammatory mediators as key pathways in the development of dementia. Redefining AD as a multifactorial condition fueled by systemic biochemical and immunological dysregulation is its theoretical and practical relevance. It is possible to build next-generation therapies that can reduce or stop neurodegeneration by incorporating oxidative and inflammatory pathways into both diagnostic and therapeutic models. As a result, this literature advances both scientific knowledge and translational opportunities in dementia research.

CONCLUSION

This integrative qualitative analysis concludes that oxidative stress and inflammatory mediators are interdependent, primary mechanisms in the pathogenesis of dementia, particularly Alzheimer's disease. The findings provide a deeper understanding of how molecular imbalances in reactive oxygen species (ROS), mitochondrial dysfunction, and neuroinflammatory signaling converge to trigger neuronal injury and cognitive decline. These results substantiate and extend existing theories such as the oxidative-inflammatory cascade and mitochondrial cascade hypotheses, positioning them within a more holistic neurobiological framework that includes systemic immune contributions. The study advances prior research by demonstrating that oxidative and inflammatory pathways operate synergistically, not sequentially, and that their chronic interaction perpetuates neurodegenerative progression. Beyond theoretical enrichment, these findings carry significant academic and clinical implications, emphasizing the necessity of multi-targeted therapeutic approaches that address both oxidative imbalance and chronic inflammation. In broader social and cultural contexts, this understanding reinforces the urgency of preventive interventions through lifestyle, nutrition, and neuroprotective strategies aimed at mitigating oxidative and inflammatory burdens in aging populations. However, the study also acknowledges limitations arising from the reliance on secondary data and the predominance of preclinical models, which may not fully capture human complexity. Future investigations should adopt longitudinal, cross-disciplinary designs integrating molecular, clinical, and behavioral dimensions to better elucidate the temporal dynamics of oxidative–inflammatory interactions and foster the development of personalized therapeutic paradigms in dementia research.

Based on this qualitative study, researchers are encouraged to examine the relationship between oxidative stress and inflammation using multidimensional, longitudinal, and triangulated approaches, while practitioners apply preventive strategies such as antioxidant nutrition, anti-inflammatory lifestyles, and early detection to reduce dementia risk. Policymakers should support community-based brain health programs that emphasize dietary, environmental, and behavioral regulation. Cross-cultural studies are

also needed to strengthen evidence for precision prevention and innovative therapies against neurodegenerative diseases.

REFERENCES

Abraham, D., P. P. (2024). A Methodological Framework for Descriptive Phenomenological Research. Western Journal of Nursing Research, 47, 125–134. https://doi.org/10.1177/01939459241308071

Ahmad, S., Yang, W., Orellana, A., Frölich, L., De Rojas, I., Cano, A., Boada, M., Hernández, I., Hausner, L., Harms, A., Bakker, M., Cabrera-Socorro, A., Amin, N., Ramirez, A., Ruiz, A., Van Duijn, C., & Hankemeier, T. (2024). Association of oxidative stress and inflammatory metabolites with Alzheimer's disease cerebrospinal fluid biomarkers in mild cognitive impairment. Alzheimer's Research & Therapy, 16. https://doi.org/10.1186/s13195-024-01542-4

Altahrawi, A., James, A., & Shah, Z. (2025). The role of oxidative stress and inflammation in the pathogenesis and treatment of vascular dementia. Cells, 14. https://doi.org/10.3390/cells14080609

Bai, R., Guo, J., Ye, X., Xie, Y., & Xie, T. (2022). Oxidative stress: The core pathogenesis and mechanism of Alzheimer's disease. Ageing Research Reviews, 77. https://doi.org/10.1016/j.arr.2022.101619

Bandaranayake, P. (2024). Application of Grounded Theory Methodology in Library and Information Science Research: An Overview. Sri Lanka Library Review. https://doi.org/10.4038/sllr.v38i2.70

Belotto, M. (2018). Data Analysis Methods for Qualitative Research: Managing the Challenges of Coding, Interrater Reliability, and Thematic Analysis. The Qualitative Report. https://doi.org/10.46743/2160-3715/2018.3492

Bhatia, V., & Sharma, S. (2020). Role of mitochondrial dysfunction, oxidative stress and autophagy in progression of Alzheimer's disease. Journal of the Neurological Sciences, 421. https://doi.org/10.1016/j.jns.2020.117253

Bingham, A. (2023). From Data Management to Actionable Findings: A Five-Phase Process of Qualitative Data Analysis. International Journal of Qualitative Methods, 22. https://doi.org/10.1177/16094069231183620

Birla, H., Minocha, T., Kumar, G., Misra, A., & Singh, S. (2020). Role of oxidative stress and metal toxicity in the progression of Alzheimer's disease. Current Neuropharmacology, 18, 552–562. https://doi.org/10.2174/1570159x18666200122122512

De Oliveira, J., Kucharska, E., Garcez, M., Rodrigues, M., Quevedo, J., Moreno-Gonzalez, I., & Budni, J. (2021). Inflammatory cascade in Alzheimer's disease pathogenesis: A review of experimental findings. Cells, 10. https://doi.org/10.3390/cells10102581

Dhapola, R., Hota, S., Sarma, P., Bhattacharyya, A., Medhi, B., & Reddy, D. (2021). Recent advances in molecular pathways and therapeutic implications targeting neuroinflammation for Alzheimer's disease. Inflammopharmacology, 29, 1669–1681. https://doi.org/10.1007/s10787-021-00889-6

Doyle, L., M., C., Keogh, B., Brady, A., McCann, M. (2019). An overview of the qualitative descriptive design within nursing research. Journal of Research in Nursing, 25, 443–455. https://doi.org/10.1177/1744987119880234

Fanlo-Ucar, H., Picón-Pagès, P., Herrera-Fernández, V., Ill-Raga, G., & Muñoz, F. (2024). The dual role of amyloid beta-peptide in oxidative stress and inflammation: Unveiling their connections in Alzheimer's disease etiopathology. Antioxidants, 13. https://doi.org/10.3390/antiox13101208

Fife, S., G., J. (2024). Deductive Qualitative Analysis: Evaluating, Expanding, and Refining Theory. International Journal of Qualitative Methods, 23. https://doi.org/10.1177/16094069241244856

Güler, E. (2025). Comparative analysis of oxidative stress, inflammatory markers, and neurotrophic factors in healthy controls, vascular dementia, and Alzheimer's dementia. Haydarpasa Numune Training and Research Hospital Medical Journal. https://doi.org/10.14744/hnhj.2025.93276

Hsu, Y., Huang, Y., Tsai, S., Kuo, Y., Lin, J., Ho, H., Chen, J., Hsia, K., & Sun, Y. (2023). Efficacy of probiotic supplements on brain-derived neurotrophic factor, inflammatory biomarkers, oxidative stress, and cognitive function in patients with Alzheimer's dementia: A 12-week randomized, double-blind active-controlled study. Nutrients, 16. https://doi.org/10.3390/nu16010016

Jimenez, S., B.-M., J., De La Torre, R. (2024). How do university libraries contribute to the research process? The Journal of Academic Librarianship. https://doi.org/10.1016/j.acalib.2024.102930

Kalpokaite, N., R., I. (2018). Demystifying Qualitative Data Analysis for Novice Qualitative Researchers. The Qualitative Report. https://doi.org/10.46743/2160-3715/2019.4120

Leng, F., & Edison, P. (2020). Neuroinflammation and microglial activation in Alzheimer disease: Where do we go from here? Nature Reviews Neurology, 17, 157–172. https://doi.org/10.1038/s41582-020-00435-y

Lopez-Rodriguez, A., Hennessy, E., Murray, C., Nazmi, A., Delaney, H., Healy, D., Fagan, S., Rooney, M., Stewart, E., Lewis, A., Barra, N., Scarry, P., Riggs-Miller, L., Boche, D., Cunningham, M., & Cunningham, C. (2021). Acute systemic inflammation exacerbates neuroinflammation in Alzheimer's disease: IL-1β drives amplified responses in primed astrocytes and neuronal network dysfunction. Alzheimer's & Dementia, 17, 1735–1755. https://doi.org/10.1002/alz.12341

Novoa, C., Salazar, P., Cisternas, P., Gherardelli, C., Vera-Salazar, R., Zolezzi, J., & Inestrosa, N. (2022). Inflammation context in Alzheimer's disease, a relationship intricate to define. Biological Research, 55. https://doi.org/10.1186/s40659-022-00404-3

Pappolla, M., Martins, R., Poeggeler, B., Omar, R., & Perry, G. (2024). Oxidative stress in Alzheimer's disease: The shortcomings of antioxidant therapies. Journal of Alzheimer's Disease, 101, S155–S178. https://doi.org/10.3233/jad-240659

Perluigi, M., Di Domenico, F., & Butterfield, D. (2024). Oxidative damage in neurodegeneration: Roles in the pathogenesis and progression of Alzheimer disease. Physiological Reviews, 104(1), 103–197. https://doi.org/10.1152/physrev.00030.2022

Plascencia-Villa, G., & Perry, G. (2023). Roles of oxidative stress in synaptic dysfunction and neuronal cell death in Alzheimer's disease. Antioxidants, 12. https://doi.org/10.3390/antiox12081628

Pratt, M. (2025). On the Evolution of Qualitative Methods in Organizational Research. Annual Review of Organizational Psychology and Organizational Behavior. https://doi.org/10.1146/annurev-orgpsych-111722-032953

Rummel, N., & Butterfield, D. (2021). Altered metabolism in Alzheimer disease brain: Role of oxidative stress. Antioxidants & Redox Signaling, 35, 987–1005. https://doi.org/10.1089/ars.2021.0177

Scarian, E., Viola, C., Dragoni, F., Di Gerlando, R., Rizzo, B., Diamanti, L., Gagliardi, S., Bordoni, M., & Pansarasa, O. (2024). New insights into oxidative stress and inflammatory response in neurodegenerative diseases. International Journal of Molecular Sciences, 25. https://doi.org/10.3390/ijms25052698

Tadokoro, K., Ohta, Y., Inufusa, H., Loon, A., & Abe, K. (2020). Prevention of cognitive decline in Alzheimer's disease by novel antioxidative supplements. International Journal of Molecular Sciences, 21. https://doi.org/10.3390/ijms21061974

Tamagno, E., Guglielmotto, M., Vasciaveo, V., & Tabaton, M. (2021). Oxidative stress and beta amyloid in Alzheimer's disease: Which comes first? Antioxidants, 10. https://doi.org/10.3390/antiox10091479

Thakur, S., Dhapola, R., Sarma, P., Medhi, B., & Reddy, D. (2022). Neuroinflammation in Alzheimer's disease: Current progress in molecular signaling and therapeutics. Inflammation, 46, 1–17. https://doi.org/10.1007/s10753-022-01721-1

Togia, A., M., A. (2017). Research Methods in Library and Information Science. https://doi.org/10.5772/intechopen.68749

Twarowski, B., & Herbet, M. (2023). Inflammatory processes in Alzheimer's disease—Pathomechanism, diagnosis and treatment: A review. International Journal of Molecular Sciences, 24. https://doi.org/10.3390/ijms24076518

Vila-Henninger, L., D., C., Van Ingelgom, V., Caprioli, M., Teuber, F., Pennetreau, D., Bussi, M., Gall, C. (2022). Abductive Coding: Theory Building and Qualitative (Re)Analysis. Sociological Methods & Research, 53, 968–1001. https://doi.org/10.1177/00491241211067508

Vogrinc, D., Kramberger, G., Emeršič, A., Čučnik, S., Goričar, K., & Dolžan, V. (2023). Genetic polymorphisms in oxidative stress and inflammatory pathways as potential biomarkers in Alzheimer's disease and dementia. Antioxidants, 12. https://doi.org/10.3390/antiox12020316

Walker, K., Page, L., Terrando, N., Duggan, M., Heneka, M., & Bettcher, B. (2023). The role of peripheral inflammatory insults in Alzheimer's disease: A review and research roadmap. Molecular Neurodegeneration, 18. https://doi.org/10.1186/s13024-023-00627-2

Xie, J., Van Hoecke, L., & Vandenbroucke, R. (2022). The impact of systemic inflammation on Alzheimer's disease pathology. Frontiers in Immunology, 12. https://doi.org/10.3389/fimmu.2021.796867