**Medical Science**

**To Cite:**
Olszewski J, Uszak S, Rojek J, Jędrzejewska P, Szczawiński T, Ślusarczyk I, Szlajss W, Wieczorek D, Kruk N, Chachuła A. Neuroinflammation in Alzheimer's disease: current evidence and therapeutic targets. *Medical Science* 2026; 30: e168ms3927
doi:

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**Peer-Review History**
Received: 21 March 2026
Reviewed & Revised: 07/April/2026 to 09/August/2026
Accepted: 18 August 2026
Published: 27 August 2026

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

Medical Science
pISSN 2321–7359; eISSN 2321–7367



# Neuroinflammation in Alzheimer's disease: current evidence and therapeutic targets

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## ABSTRACT

*Background/Purpose*: A key part of what causes Alzheimer's disease (AD) is inflammation in the brain. In the past, this was seen as a result of other problems, but now we know that it's a main cause of brain cell damage. This review synthesizes current evidence on about the molecular mechanisms, cellular mediators, and therapeutic strategies that target neuroinflammation in AD. *Methods*: We searched for information using these keywords: neuroinflammation and Alzheimer's disease, microglia in AD, the NLRP3 inflammasome and neurodegeneration, TREM2 in AD, astrocytes and neuroinflammation, the blood-brain barrier in AD, the JAK/STAT pathway and neuroinflammation, and GLP-1 receptor agonists and neurodegeneration. The publication date was the most important factor for articles published between 2022 and 2026. *Findings*: In Alzheimer's disease (AD), two types of cells, microglia and astrocytes, play a key role in causing the inflammation of the brain. This imbalance drives the accumulation of amyloid-beta (Aβ) and promotes abnormal tau hyperactivity. The NLRP3 inflammasome, NF-κB, and JAK/STAT pathways are the main molecular mediators of chronic neuroinflammatory signaling. Therapeutic strategies targeting TREM2, IL-1β/IL-18, GLP-1 receptors, Nrf2, and natural compounds show promise in preclinical and early clinical trials. The blood-brain barrier and immune cell infiltration are important to understand for the treatment of neuroinflammation. *Conclusion*: By synthesizing evidence published between January 2022 and March 2026, this review provides a comprehensive account of the mechanisms underlying neuroinflammation in Alzheimer's disease. These mechanisms are similar to current investigational therapeutic agents, such as small-molecule agonists in Phase 2 clinical trials.

**Keywords**: Alzheimer's disease, neuroinflammation, microglia, NLRP3 inflammasome, TREM2, therapeutic targets.

## 1. INTRODUCTION

Among neurodegenerative disorders, Alzheimer's disease (AD) is the most prevalent disorder worldwide. It causes about 60–70% of all dementia cases and

affects over 55 million people worldwide. This number is expected to triple by 2050 (Heneka et al., 2025; Thakur et al., 2023). AD is marked by two hallmarks: amyloid-beta (Aβ) plaques and neurofibrillary tangles (tau). However, decades of unsuccessful clinical trials targeting the amyloid cascade have led the scientific community to rethink the complex causes of AD (Cummings et al., 2023).

Neuroinflammation occurs when the immune cells in your brain start to react to things that are causing problems in your body. It has become clear that it is an important part of why people with Alzheimer's disease (AD) start to have problems with their memory and thinking. As the resident immune cells of the CNS, microglia play a key role in this process. They sense harmful protein buildup and control future inflammatory responses (Cai et al., 2022; Chen et al., 2024). Reactive astrocytes secrete pro-inflammatory mediators that amplify neuroinflammatory signaling. This process compromises synaptic integrity, thereby impairing the pathways through which neurons exchange information.

Researchers are studying ways to treat neuroinflammation with drugs. These findings have led to new ways to treat the disease, such as using monoclonal antibodies that target TREM2, strategies that block the effect of IL-1β, GLP-1 receptor agonists, and small-molecule inflammasome inhibitors. Right now, some of these drugs are being tested in Phase 2 clinical trials (Burgess et al., 2024; Du et al., 2022). At the same time, scientists are studying if certain molecules in the fluid around the brain and blood can help find Alzheimer's disease earlier and provide more personalized treatments (Lista et al., 2024).

## 2. REVIEW METHODS

We searched four big medical databases. The publications from January 2022 and March 2026 were included for the most recent information. Earlier publications were also included to provide historical context.

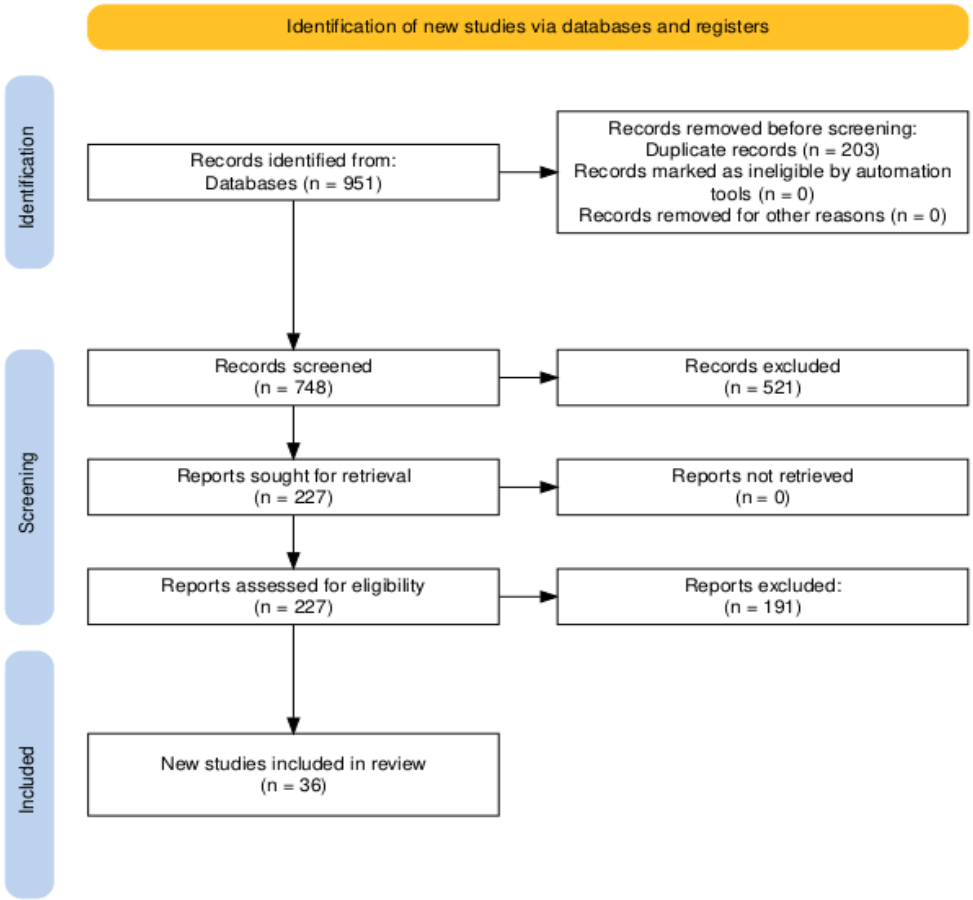


**Figure 1**. PRISMA 2020 flow diagram of study selection process.

The following keywords were used: "Neuroinflammation and Alzheimer's disease," "microglia and Alzheimer's disease," "astrocytes and neuroinflammation and Alzheimer's disease," "NLRP3 inflammasome and Alzheimer's disease," "TREM2 and therapeutic target

and Alzheimer's disease," "blood-brain barrier and neuroinflammation and Alzheimer," "JAK/STAT and neuroinflammation," "Nrf2 and neuroprotection and Alzheimer's disease," "GLP-1 receptor agonists and neurodegeneration," "IL-1β and Alzheimer's disease," "pyroptosis and Alzheimer's disease," and "neuroinflammation biomarkers and Alzheimer's disease.

The results of the literature search are shown in a PRISMA 2020 flow diagram (Figure 1). We found a total of 951 records in all the databases we searched (412 in PubMed, 287 in Scopus, 198 in Google Scholar, and 54 in DOAJ). After removing 203 duplicate records, 748 unique records were checked by title and abstract. 521 records were excluded: off-topic, non-English, published befor 2022, or not directly focused on Alzheimer's disease. Full-text screening was performed on 227 articles. They decided not to include 191 of these articles in the study. The reasons these articles were not included were:

- Not peer-reviewed (n=41)
- Not enough information about the methods used (n=34)
- Not related to AD (n=62)
- Included only as a reference to other studies from before 2022 (n=54).

Altogether, 36 studies met all the requirements and are mentioned in this review.

*Inclusion criteria:*

- Research articles that have been reviewed by other scientists and published in English

- Reviews that are written in a way that is easy to understand and that talk about the reasons why brain cells are affected by inflammation in people with Alzheimer's disease

- Studies that report on medicines that target inflammation in the brain in animals or people The following types of studies were not included: conference abstracts without the full text, studies in languages other than English, studies not related to the central nervous system, and studies with incomplete descriptions of the methods used. Ultimately, 36 articles satisfied all inclusion criteria and were incorporated into this review.

## 3. RESULTS AND DISCUSSION

**Neuroinflammation as a Core Pathological Feature of AD**

Neuroinflammation is now seen as a part of many problems with the brain, not just problems with amyloid and tau. Studies now show that it is a key player in the development of Alzheimer's disease (Li et al., 2022). Studies of living brains and brain scans show activated microglia, elevated pro-inflammatory cytokines, and complement system activation in AD brains. This often happens before the symptoms of AD become obvious (Heneka et al., 2025; Gouilly et al., 2023). PET imaging studies link neuroinflammation to problems with brain networks and cognitive decline in AD (Leng et al., 2023).

Neuroinflammation does not happen on its own. It is connected to the build-up of amyloid-beta (Aβ) and tau. These peptides activate glia, leading to pro-inflammatory mediator release and tau hyperphosphorylation and spread. This back-and-forth interaction creates a cycle where neuroinflammation and protein aggregation both get worse, and this cycle keeps getting worse over time. Therefore, drugs that target these pathways have become a promising way to treat the disease and reduce symptoms (Boyd et al., 2022; Gaikwad et al., 2024).

**Microglial Activation: Protective or Pathological?**

Microglia are the brain's main immune cells. How they work depends on the situation (Wei and Li, 2022). Under normal conditions, microglia maintain neural homeostasis through waste clearance, synaptic pruning and trophic support of neurons (Brown et al., 2026; Deng et al., 2024). However, in the AD brain, long-term exposure to Aβ and tau oligomers causes microglia to constantly release chemicals like IL-1β and TNF-α and excessive amounts of ROS. At the same time, the microglia's phagocytic ability is reduced (Chen et al., 2024; Wang et al., 2023).

Recent studies of single-cell genes have identified a microglia (brain cell) phenotype associated with disease in Alzheimer's disease (AD). This is a transitional state with two processes: phagocytosis the process of engulfing and digesting foreign particles and inflammatory response.

In the healthy brain, microglia remain in a surveillant resting state, continuously monitoring the local microenvironment. When the body is exposed to things that can cause neurodegenerative diseases, it triggers a change into Stage 1 DAM (TREM2-independent). To progress to Stage 2 DAM, TREM2 signaling must be fully functional. This is important for protecting the body and clearing protein buildup. Loss-of-function TREM2 mutations impair this protective transition, resulting in sustained neuroinflammation and

progressive synaptic loss. TREM2 is a microglia receptor. It plays a key role in a process called the "DAM transition," during which microglia stop working properly. Some versions of TREM2 that don't work well increase the risk of Alzheimer's disease by making microglia respond poorly to Aβ deposits. One of the main goals in developing treatments for Alzheimer's disease (AD) is to understand and control how microglia, a type of brain cell, can change their function.

**Astrocytic Neuroinflammatory Responses**

Astrocytes are another important cell type involved in neuroinflammation in AD (Rodríguez-Giraldo et al., 2022). In unhealthy conditions, certain cells in the brain (called "astrocytes") change from a protective state to a harmful state. This change releases proteins and other substances that can damage neurons and reduce connections between neurons. This particular cell type has been found in the brains of people with Alzheimer's disease, and it is linked to the buildup of a protein called amyloid and the loss of cognitive abilities (Kim et al., 2024).

Astrocytes affect the balance of glutamate in the brain, and when they malfunction, it disrupts the balance between excitatory and inhibitory signals in the brain, which is a process that is increasingly linked to cognitive symptoms of Alzheimer's disease (AD) (Ranasinghe et al., 2025). Furthermore, reactive astrocytes help maintain synapses by clearing tau oligomers from these structures. Impairment of this clearance mechanism accelerates tau propagation across neural circuits. Changing the way certain brain cells respond from a pro-inflammatory state (A1) to a protective state (A2) could be a helpful treatment, although it is still being studied (Kim et al., 2024; Rodríguez-Giraldo et al., 2022).

**The NLRP3 Inflammasome: A Key Molecular Mediator**

The NLRP3 inflammasome is one of the most studied molecular mechanisms in AD neuroinflammation. The NLRP3 complex comprises the NLRP3 sensor protein, the ASC adapter, and procaspase-1. These include Aβ fibrils, tau oligomers, mitochondrial reactive oxygen species (ROS), and potassium efflux. These are all processes and molecules that are increased or out of balance in the brains of people with Alzheimer's disease (AD). Once it's active, NLRP3 helps other molecules, called cytokines, to mature and be released. These cytokines are IL-1β and IL-18. They contribute to tau hyperphosphorylation and aggregation in the brain (McManus and Latz, 2024).

A cell type death caused by inflammation called pyroptosis has been shown to play a contributes to neuronal loss in AD (AD). This distinct cell death pathway is triggered by activation of a specific set of genes and proteins (Xue et al., 2022; Saad et al., 2026). Some studies on mice with Alzheimer's disease (AD) have shown that using medicines that target a protein called NLRP3 can reduce the buildup of a substance in the brain called Aβ, the uncontrolled folding of a protein called tau, and problems with thinking and memory.

**NF-κB, JAK/STAT, and Nrf2: Signaling Crossroads**

There are three other signaling pathways that play important roles in brain inflammation in people with Alzheimer's disease (Table 1). NF-κB, activated via TLR4 and Aβ signaling, regulates transcription of pro-inflammatory genes such as COX-2, iNOS, and various cytokines. Continuing this process causes inflammation in two types of brain cells, microglia and astrocytes (Li et al., 2022). The JAK/STAT pathway is important in a process called "interferon-driven microglial activation." This pathway is also important in regulating neuroinflammation in AD. On a different note, the Nrf2 antioxidant signaling pathway is like a system that controls these processes, keeping them balanced.

**Table 1**. Neuroinflammation pathways in Alzheimer's disease.

| Pathway | Key Activators | Downstream Effectors | Therapeutic Potential |
|---|---|---|---|
| NF-κB | TLR4, Aβ, TNF-α | IL-1β, IL-6, COX-2, iNOS | High; multiple inhibitors in preclinical testing |
| NLRP3 inflammasome | Aβ, tau, ROS, $K^+$ efflux | Caspase-1, IL-1β, IL-18, pyroptosis | High; MCC950, OLT1177 in trials |
| JAK/STAT | IFN-γ, IL-6, IL-18 | Microglia M1 activation, STAT3 | Moderate; JAK inhibitors in development |
| Nrf2 | Oxidative stress | HO-1, NQO1, anti-inflammatory genes | High; curcumin, flavonoids |

When activated, Nrf2 reduces oxidative stress, suppresses NF-κB activity, and promotes the expression of antioxidant or cell-protective genes. This helps to control the inflammatory response in both microglia and astrocytes (Saha et al., 2022). Research shows that curcumin and related flavonoids can protect nerve cells, at least in part, by activating a pathway called Nrf2.

The blood-brain barrier (BBB) is a special cell type membrane that controls what goes into and out of the brain. Emerging evidence indicates a bidirectional relationship between BBB integrity and neuroinflammation. This reaction can be both the cause and the result of Alzheimer's disease (AD). High protein levels indicate a broken BBB. These signs are linked to how bad Alzheimer's disease (AD) and cognitive impairment (CI) are. BBB disruption allows immune cells from the body to enter the brain. These cells can cause inflammation and brain damage (Zhang et al., 2024; Ly et al., 2023; Zhang and Yan, 2023).

**Emerging Therapeutic Targets**

***TREM2-Targeting Strategies***

TREM2 has become a top target for treatments in AD neuroinflammation (Larson et al., 2022; Mirescu et al., 2024). The TREM2-activating monoclonal antibody AL002 is being studied in the INVOKE-2 Phase 2 trial. This trial is randomized, double-anonymized, and placebo-controlled. Participants are patients with early AD. The trial found that the drug is safe and effective. This was seen in an early analysis of the study (Burgess et al., 2024; Mayorga et al., 2024). VGL101 is an antibody that targets CSF1R and indirectly activates TREM2. VGL101 is being studied in a trial called IGNITE (Lynch et al., 2024).

**Table 2**. Selected therapeutic agents targeting neuroinflammation in Alzheimer's disease

| Agent | Target | Mechanism | Stage |
|---|---|---|---|
| AL002 (antibody) | TREM2 | Microglial activation, Aβ phagocytosis | Phase 2 (INVOKE-2) |
| VGL101 | CSF1R/TREM2 | Microglial survival and function | Phase 2 (IGNITE) |
| VG-3927 | TREM2 | Small-molecule agonist | Phase 1 |
| MCC950 | NLRP3 | Inflammasome inhibitor | Preclinical/early clinical |
| OLT1177 | NLRP3 | Inflammasome inhibitor | Phase 1/2 |
| Liraglutide/Semaglutide | GLP-1R | Anti-inflammatory, neuroprotective | Phase 2/3 |
| IL-18 binding protein | IL-18 | Cytokine neutralization | Preclinical |
| Curcumin derivatives | Nrf2/NF-κB | Antioxidant, anti-inflammatory | Preclinical |

***Cytokine-Targeting and Immunomodulatory Approaches***

In preclinical models of Alzheimer's disease (AD), targeting pro-inflammatory cytokines has shown benefits in protecting the brain (Table 2). Experts discovered that a protein called IL-18BP could help reduce inflammation in the brain, as well as the loss of brain cells and memory problems in a mouse model of Alzheimer's disease. Therefore, IL-18 might be a good choice for medicines that treat Alzheimer's disease (Hong et al., 2026).

A study done by Zhao et al., (2026) found that using specific antibodies to block IL-1β improved memory problems and tau pathology in a model of Alzheimer's disease with high homocysteine levels. As a result, this research supports the idea that the NLRP3-IL-1β axis is a good target for treatment. Research is being done on ways to treat diseases using a process called pyroptosis. This process can be studied in two different ways: by looking at the effects that happen before or after a specific step in the process, which is called "caspase-1" and "gasdermin D." Another research area is studying how microRNAs can help control these processes.

***GLP-1 Receptor Agonists***

A type of drug called GLP-1R agonists was first made to treat type 2 diabetes. It has since been shown to have many benefits, including protecting nerve cells and reducing inflammation. These effects are important for people with Alzheimer's disease. Recent reviews

suggest that GLP-1R agonists may help reduce brain inflammation, improve brain insulin resistance, and show early signs of improving cognitive function in patients with Alzheimer's disease (Du et al., 2022). Right now, there are many Phase 2 and Phase 3 trials looking at whether liraglutide, semaglutide, and related compounds can slow down the progression of Alzheimer's disease. Some of these trials also look at neuroinflammation as a secondary outcome (Du et al., 2022; Cummings et al., 2023).

***Natural Compounds and Exercise Mimetics***

Curcumin derivatives have similar effects, reducing the effects of Aβ on activated microglia and the buildup of tau in preclinical models. This is likely due to their antioxidant and anti-inflammatory properties (Sawikr et al., 2024). Exercise mimetics are compounds that have the same effects on the brain and body as exercise does. Research shows that these compounds could be used to treat Alzheimer's disease. In animal models, exercise-induced exerkines change microglia from a pro-inflammatory state to a homeostatic state and stop NLRP3 inflammasome activation. All of this suggests that signaling molecules from the body can control central neuroinflammatory processes (Zhao, 2024).

**Neuroinflammatory Biomarkers: Toward Precision Medicine**

Table 3 shows the identifying specific molecules in the blood and cerebrospinal fluid (CSF) is essential for correctly diagnosing and monitoring AD (Lista et al., 2024). YKL-40, GFAP, sTREM2, and PDGFRβ are all proven brain inflammation markers for Alzheimer's disease (Lista et al., 2024; Cicognola et al., 2023). Neuroinflammatory PET imaging can show significant differences in neuroinflammation during the early stages of AD. But this variability can help doctors make better treatment plans for each patient (Gouilly et al., 2023; Leng et al., 2023).

**Table 3**. Neuroinflammatory biomarkers (indicators of nerve cell inflammation) in Alzheimer's disease

| Biomarker | Compartment | Biological Significance | Clinical Utility |
|---|---|---|---|
| YKL-40 | CSF, plasma | Astrocytic activation | Disease staging, progression monitoring |
| GFAP | Plasma, CSF | Reactive astrogliosis | Aβ burden correlation |
| sTREM2 | CSF | Microglial activation | DAM transition marker |
| PDGFRβ | CSF, plasma | BBB pericyte damage | BBB disruption severity |
| TSPO (PET) | Brain (imaging) | Activated microglia | Neuroinflammation mapping |

## 4. CONCLUSION

The evidence here shows that neuroinflammation is a driver of Alzheimer's disease, not just a result. Two types of brain cells, microglia and astrocytes, together create a continuous inflammatory response that speeds up the buildup of a protein called Aβ, the formation of a protein called tau that is abnormally folded, the loss of connections between neurons, and the death of neurons. This is caused by a long-term imbalance in the NLRP3 inflammasome, NF-κB, and JAK/STAT pathways. Disrupting the blood-brain barrier makes these processes worse by allowing immune cells from the body to enter the brain tissue and by impairing the brain's ability to remove waste. The world of medicine is changing quickly. TREM2-activating antibodies, inflammasome inhibitors, GLP-1R agonists, cytokine-targeting biologics, and natural anti-inflammatory compounds are all showing promise in early tests. The success of these strategies critically depends on validated neuroinflammatory biomarkers enabling patient stratification and individualized treatment planning.

Future research should focus on: (1) Understanding how neuroinflammation changes from a protective response to a harmful one over time; (2) Testing ways to treat neuroinflammation by targeting multiple causes at once; (3) Including blood tests for biomarkers in clinical trials for Alzheimer's disease; and (4) Finding out how peripheral inflammation, metabolic disorders, and central nervous system (CNS) neuroinflammation together increase the risk of Alzheimer's disease and make it worse.

**Acknowledgments**

The authors thank all researchers whose published work provided the foundation for this review.

**Authors' Contributions**

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

**Informed consent**

Not applicable. This review does not contain any studies with human participants performed by the authors.

**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 interest, competing financial interests or personal relationships that could have influenced the work reported in this paper.

**Data and materials availability**

The data supporting the findings of this review are included in the manuscript as well as the referenced publications.

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