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REVIEW ARTICLE

# A Literature Review: Effects of NLRP3 Inflammasome Inhibitor on Alzheimer's Disease

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**ABSTRACT**

Alzheimer is a neurodegenerative disease that is characterized by decreased cognitive abilities and pathological changes in the brain. The prevalence of Alzheimer's disease is rising globally, affecting over 50 million people in 2020, and is projected to reach 152 million by 2050. The NLRP3 inflammasome can be targeted with pharmacological inhibitors that cross the blood-brain barrier (BBB) to treat neurodegenerative and CNS diseases. This literature review evaluates the impact of NLRP3 inflammasome inhibitors on Alzheimer's disease progression, selecting English-language articles published between 2014 and 2024. Relevant biochemistry, immunology, and molecular biology studies were sourced from PubMed, Google Scholar, ScienceDirect, and Web of Science. Using the PRISMA method, 300 initial articles were screened, 261 duplicate articles were excluded, and 39 articles were further assessed based on inclusion and exclusion criteria, resulting in 5 articles reviewed. NLRP3 inflammasome inhibitors, such as anakinra, OLT1177, and β-hydroxybutyrate, can potentially slow Alzheimer's disease progression by reducing neurodegenerative inflammation and amyloid-beta accumulation. These inhibitors can improve cognitive function and decrease neuroinflammatory damage, making NLRP3 a promising therapeutic target for Alzheimer's treatment.

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## 1. Introduction

Alzheimer's disease is a progressive neurodegenerative disorder characterized by a decline in cognitive function, memory, and thinking abilities, eventually disrupting the daily activities of those affected. The prevalence of Alzheimer's is increasing globally, with more than 50 million people worldwide living with the disease in 2020, and it is projected to rise to 152 million by 2050 (Prince *et al*. 2015). In Southeast Asia, the prevalence of Alzheimer's is rapidly increasing, mainly due to demographic and lifestyle changes. For instance, the prevalence of Alzheimer's dementia in Malaysia is estimated to be 8.5% among the elderly population (Alzheimer's Disease Foundation Malaysia, 2020). In Indonesia, the prevalence of Alzheimer's dementia was approximately 1.2 million in 2016, and this number is projected to increase to 4 million by 2050 (Alzheimer's Indonesia, 2019). The impact of this disease is felt by the individuals affected and their families and society at large, given the intensive care needs and significant costs associated with managing this disease (Prince *et al*. 2015).

Alzheimer's is characterized by specific pathological changes in the brain, such as language impairment, executive dysfunction, attention deficits, and progressive decline in cognitive and visuospatial functions (Scheltens, 2015). According to the amyloid cascade hypothesis, amyloid precursor protein (APP) is cleaved by α-secretase and is improperly processed by β-secretase and γ-secretase, creating an imbalance between the production and clearance of amyloid-beta (Aβ) peptides. Aβ peptides accumulate into soluble oligomers, aggregate to form insoluble fibrils in a beta-sheet conformation, and eventually deposit into plaques (Hardy & Selkoe, 2002). Aβ plaques can activate the Nucleotide-binding Oligomerization Domain-like receptor family pyrin domain-containing 3 (NLRP3) inflammasome, leading to the release of pro-inflammatory cytokines such as Interleukin-1 beta (IL-1β), which can exacerbate neuroinflammation and brain tissue damage (Murphy & LeVine, 2010).

The NLRP3 protein is a member of the NOD-like receptor (NLR) family and a critical component of the NLRP3 inflammasome. This inflammasome plays a key role in the body's defense as well as in maintaining cellular balance and health (Qin *et al*., 2024). The NLRP3 inflammasome consists of the NLRP3 protein, procaspase-1, and Apoptosis-associated speck-like protein containing a CARD (ASC). Procaspase-1 acts as an effector within the NLRP3 inflammasome and contains a Caspase activation and recruitment domain (CARD) domain. ASC is a complex composed of two parts, the Pyrin domain (PYD) and CARD, which serve as a bridge between the NLRP3 sensor and the procaspase-1 effector (Wang *et al*., 2020). The NLRP3 inflammasome is essential for the immune response against bacterial, fungal, and viral infections. However, excessive immune responses from the NLRP3 inflammasome can contribute to various inflammatory diseases such as cryopyrin-

associated periodic syndromes (CAPS), Alzheimer's disease, diabetes, gout, autoinflammatory diseases, and atherosclerosis (Kelley *et al*., 2019).

Inhibitors of the NLRP3 inflammasome pathway target the priming and activation steps in the NLRP3 inflammasome signaling pathway. During NLRP3 inflammasome activation, NLRP3 oligomerizes through its NACHT domain following stimulation by Damage-associated molecular patterns (DAMPs) and Pathogen-associated molecular patterns (PAMPs). NLRP3 then recruits ASC and caspase-1 to form the NLRP3 inflammasome. This inflammasome mediates Gasdermin D (GSDMD) to cause cell membrane rupture and release Interleukin-1 beta (IL-1β) and Interleukin-18 (IL-18), resulting in inflammation and pyroptosis (Zhang *et al*., 2023). Various pharmacological inhibitors of the NLRP3 inflammasome have been developed, such as JC124, Parthenolide, Bay 11-7082, MCC950, MNS, CY-09, Tranilast, OLT1177, Oridonin, and Type I Interferons (IFNs). Some drugs are designed to target IL-1β or IL-18 to treat diseases associated with NLRP3 (Moasses *et al*., 2022). NLRP3 inhibitors can penetrate the Blood-Brain Barrier (BBB) and thus can be utilized for research on neurodegenerative disorders or other diseases related to the central nervous system (CNS) (Barczuk *et al*., 2022).

Despite substantial evidence supporting the potential benefits of NLRP3 inhibitors, a systematic review is still needed to evaluate their role in managing Alzheimer's disease. This review will help sift through the data to understand the effectiveness and mechanisms of NLRP3 inhibitors in managing Alzheimer's. This study establishes the context for a systematic literature review on the role and impact of NLRP3 inhibitors in the treatment of Alzheimer's disease.

## 2. Methods

This literature review aims to evaluate the effects of NLRP3 inflammasome inhibitors on the progression of Alzheimer's disease. The inclusion criteria are as follows: 1. Published in English; 2. Published within the period 2014-2024; 3. The article discusses NLRP3 inflammasome inhibitors concerning the progression of Alzheimer's disease; 4. The article must be relevant to the subjects of biochemistry, immunology, and molecular biology; 5. Open access and open archive. The exclusion criteria are 1. Articles in Indonesian; 2. Published outside the specified time range; 3. Available in repositories, newspapers, and reports.

**Table 1.** Tabel Framework Research Question

| P | E | O | S |
|---|---|---|---|
| Individuals with Alzheimer's | NLRP3 inflammasome inhibitors | Progression of Alzheimer's disease | Retrospective or Prospective Observational Research |

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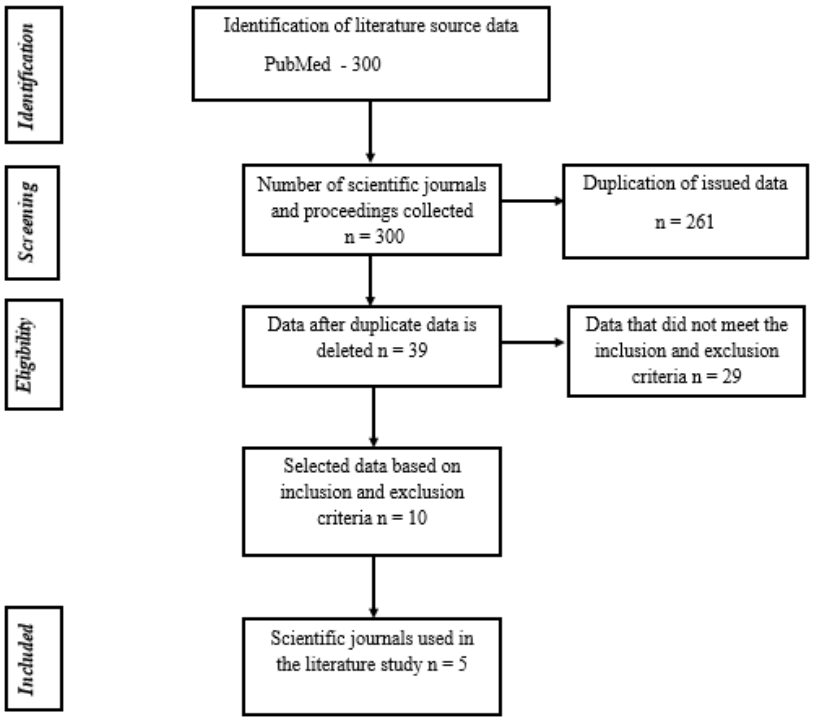
The literature search workflow involves using articles from the years 2014-2024 with limitations to the English language, free full text, and data from the last 10 years. Keywords must appear in the title/abstract. The literature search is conducted through various electronic databases, including PubMed, Google Scholar, ScienceDirect, and Web of Science.

Subsequently, articles are selected using English keywords such as "*NLRP3 inflammasome inhibitor,*" "*Alzheimer's disease,*" *and* "*neuroinflammation.*" The found articles are then screened to remove duplicates and articles with the same title. The remaining articles will be further selected based on the pre-defined inclusion and exclusion criteria. The outcome of this process is a set of articles that meet the criteria and will be the subject of analysis in the literature review.

## 3. Main Body

The PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) method is used to select the literature. The PRISMA Flow Diagram in this study is presented in Figure 1.

**Figure 1.** PRISMA *Flow Diagram*



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Based on the identification of relevant literature using the PRISMA method, a total of 300 articles were found for further analysis. A total of 261 articles were excluded due to data duplication, leaving 39 articles that passed the selection process. The final step involved reviewing according to the inclusion and exclusion criteria, resulting in 10 references with abstracts and full texts. The final result includes 5 articles to be reviewed in the literature study.

**Table 2.** Extraction Article

| No | Author/Title/Year | Result |
|---|---|---|
| 1. | Liang T, Zhang Y, Wu S, Chen Q, Wang L, *et al. The Role of NLRP3 Inflammasome in Alzheimer's Disease and Potential Therapeutic Targets.* Front. Pharmacol. 2022 | NLRP3 inflammasome inhibitors like anakinra improve Alzheimer's disease models by reducing neurodegenerative inflammation and Aβ buildup. These inhibitors prevent inflammasome activation, decrease pro-inflammatory cytokines (IL-1β, IL-18), and lead to significant cognitive improvements ($P < 0.05$). This suggests that targeting NLRP3 inflammasome could be an effective therapy for Alzheimer's by reducing inflammation and promoting Aβ clearance. |
| 2. | Lonnemann N, Hosseini S, Marchetti C, Skouras D B, Stefanoni D, D'Alessandro A, Dinarello C A, Korte M, *et al. The NLRP3 inflammasome inhibitor OLT1177 rescues cognitive impairment in a mouse model of Alzheimer's disease.* Proceedings of the National Academy of Sciences of the United States of America. 2020 | The impact of the NLRP3 inflammasome inhibitor OLT1177 on cognitive function was assessed in an Alzheimer's mouse model. Administering OLT1177 (7.5 g/kg for three months) significantly improved learning and memory in APP/PS1 mice ($P = 0.008$). The treatment also reduced microglial activation, levels of pro-inflammatory cytokines (IL-1β, IL-6, TNF-α), and Aβ plaque burden, demonstrating its therapeutic potential in Alzheimer's pathology. |
| 3. | Bai H, Zhang Q, *et al. Activation of NLRP3 Inflammasome and Onset of Alzheimer's Disease.* Front. Immunol. 2021 | NLRP3 inflammasome inhibitors β-hydroxybutyrate (BHB) and OLT1177 effectively treat Alzheimer's in the 5xFAD mouse model. BHB (100 mg/kg for 28 days) and OLT1177 (10 mg/kg for 6 months) reduced NLRP3 activation, IL-1β, caspase-1 levels, and pathological plaques, improving cognitive function and reducing neuroinflammation ($P < 0.05$). These results underscore the potential of NLRP3 inhibitors in Alzheimer's therapy. |

| | | |
|---|---|---|
| 4. | Sharma B, Satija G, Madan A, Garg M, Alam MM, Shaquiquzzaman M, *et al. Role of NLRP3 Inflammasome and Its Inhibitors as Emerging Therapeutic Drug Candidate for Alzheimer's Disease: a Review of Mechanism of Activation, Regulation, and Inhibition*. Inflammation. 2023 | NLRP3 inflammasome activation contributes to neuroinflammation and disease pathology. Previous research shows that natural compounds like Picrorhiza kurroa and Baicalin, as well as some synthetic inhibitors, effectively reduce NLRP3 activation, alleviate microglial inflammation, and lower pro-inflammatory cytokines. These findings suggest that targeting NLRP3 inflammasome could be an effective therapeutic strategy for managing Alzheimer's and slowing disease progression. |
| 5. | Daniels, MJD, Auty JR, Schilling T, Spencer NG, Watremez W, Fasolino V, *et al. Fenamate NSAIDs inhibit the NLRP3 inflammasome and protect against Alzheimer's disease in rodent models*. Nat. Commun. 2016 | Fenamate NSAIDs, such as mefenamic acid, significantly inhibit the NLRP3 inflammasome and protect against Alzheimer's in the 3xTgAD mouse model. Treatment with mefenamic acid for 28 days improved cognitive function, as measured by new object recognition tests, and reduced microglial activation and IL-1β production, indicating reduced inflammation. These findings suggest that inmate NSAIDs could be a promising therapeutic strategy for neurodegenerative inflammation associated with Alzheimer's. |

The NLRP3 inflammasome inhibitor holds significant potential in slowing the progression of Alzheimer's disease. Studies have shown that NLRP3 inflammasome inhibitors, such as anakinra, OLT1177, and β-hydroxybutyrate (BHB), can reduce neurodegenerative inflammation and amyloid-beta (Aβ) accumulation, key characteristics of Alzheimer's pathology (Dempsey *et al.*, 2017). This reduction in inflammasome activation leads to decreased release of pro-inflammatory cytokines like IL-1β, IL-6, and TNF-α, which contributes to improved cognitive function and reduced neuroinflammation (Voet *et al.*, 2019). According to research by Casares *et al.* (2023), APP/PS1 model mice treated with Anakinra showed significant improvements in cognitive capacity. This suggests that NLRP3 inflammasome inhibition could be an effective therapeutic approach for Alzheimer's through mechanisms that reduce inflammation and enhance Aβ clearance.

Previous research indicates that the NLRP3 inflammasome plays a role in Alzheimer's pathogenesis through neuroinflammatory pathways. Inhibiting the inflammasome with BHB and fenamate NSAIDs, such as mefenamic acid, can reduce microglial inflammation and improve cognitive function (Melnikov *et al.*, 2017). Inhibition of the inflammasome can decrease Aβ accumulation and microglial activation. OLT1177 has been shown to

improve learning and memory in APP/PS1 mice (Kuwar *et al*., 2021). Natural compounds like Picrorhiza kurroa and Baicalin can inhibit NLRP3 inflammasome activation and alleviate microglial inflammation (Liu *et al*., 2024). The consistency of these findings strengthens the evidence that the NLRP3 inflammasome is a promising target for therapeutic intervention in Alzheimer's treatment.

The reviewed studies face several limitations, including small sample sizes, limited observation durations, and variability in animal models. Some studies may also be affected by selection and measurement biases, and the use of transgenic mouse models may not fully reflect human Alzheimer's pathology. Future research should involve more robust study designs, such as randomized controlled trials with larger sample sizes and longer durations. Developing more accurate animal models and conducting in-depth studies on molecular mechanisms and interactions with other Alzheimer's therapies are also crucial.

## 4. Conclusion

Inhibiting the NLRP3 inflammasome holds significant potential for slowing the progression of Alzheimer's disease. NLRP3 inhibitors such as anakinra, OLT1177, and β-hydroxybutyrate (BHB) have been shown to reduce neurodegenerative inflammation and amyloid-beta (Aβ) accumulation, key features of Alzheimer's pathology. This is confirmed by previous research conducted by Cassares *et al.* and Kuwar *et al.* which stated that NLRP3 inhibitors such as anakinra and OLT1177 can improve cognitive function in animal models of APP/PS1 mice. This reduction in inflammasome activation leads to decreased release of pro-inflammatory cytokines like IL-1β, IL-6, and tumor necrosis factor-alpha (TNF-α), which in turn can improve cognitive function and reduce neuroinflammation-related damage.

Additionally, data from various studies support that inhibiting the NLRP3 inflammasome not only reduces Aβ accumulation but also enhances cognitive function in Alzheimer's disease animal models. Natural compounds such as Picrorhiza kurroa and Baicalin also show similar effects in alleviating microglial inflammation. These findings reinforce the evidence that targeting the NLRP3 inflammasome is a promising therapeutic approach for Alzheimer's, with the potential to improve cognitive conditions and reduce inflammation in patients. Although there have been many studies that prove that NLRP3 inhibition influences the progression of Alzheimer's disease, just a few studies have discussed the effectiveness, side effects, and long-term effects of using NLRP3 inhibition on the progression of Alzheimer's disease. In the future, it is hoped that further research will be conducted regarding the effectiveness and side effects of using NLRP3 inhibitors in the treatment of Alzheimer's disease.

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**Conflict of interest**

The authors declare that there are no conflicts of interest that could affect the integrity of this research. The study was conducted with good faith and high professionalism, free from external influences. The authors have no affiliations or financial interests that could impact the results of this research. The findings are presented objectively, based on data analysis and the research conducted.

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