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NEUROINFLAMMATION AS A KEY MECHANISM IN THE PROGRESSION OF ALZHEIMER'S: NEW THERAPEUTIC PERSPECTIVES

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Abstract: INTRODUCTION: Alzheimer's disease (AD) is a progressive neurodegenerative condition that causes cognitive decline, memory loss and functional impairment, affecting millions of people worldwide. Neuroinflammation, caused by excessive activation of glial cells such as microglia and astrocytes, has been identified as a central factor in the progression of the disease, exacerbating neuronal degeneration and synaptic dysfunction. **OBJECTIVE:** To analyze neuroinflammation as a key mechanism in the progression of AD, in addition to exploring new therapeutic perspectives that can modulate the cerebral inflammatory response, such as the use of inflammatory cytokine inhibitors and microglia modulators. **METHODOLOGY:** The methodology adopted was a systematic literature review, with the search and analysis of articles published between 2020 and 2024, in the main scientific databases. The search was carried out in the VHL, LILACS, MEDLINE and SCIELO databases, using specific descriptors such as "Neuroinflammation", "Alzheimer's Disease", "Microglia", "Cytokines" and "Anti-inflammatory Therapies". After applying strict inclusion and exclusion criteria, 18 articles were selected that addressed the role of neuroinflammation in AD and its therapeutic implications. Analysis of these studies provided a solid basis on emerging therapeutic approaches to modulating neuroinflammation in AD. **RESULTS AND DISCUSSION:** Neuroinflammation plays a central role in the progression of Alzheimer's disease (AD), accelerating neurodegeneration and cognitive impairment. Chronic activation of microglia and astrocytes generates inflammatory mediators that aggravate oxidative stress, compromise neuronal function and affect synaptic plasticity, which is essential for memory. In addition, neuroinflammation impairs the removal of toxic proteins such as beta-amyloid, favoring the accumulation of amyloid plaques. Neuroinflammation starts early, before the clinical symptoms of AD, and intervening in it could slow down the progression of the disease. Chronic inflammation affects mitochondrial function and the formation of new synaptic connections, which are essential for cognition. **CONCLUSION:** Controlling neuroinflammation and restoring affected cellular processes could be an effective therapeutic strategy for slowing the progression of AD. Therapies such as inflammatory cytokine inhibitors and microglia modulators show promising results, although more clinical studies are needed.

Keywords: Cytokines; Alzheimer's Disease; Microglia; Neuroinflammation; Anti-inflammatory Therapies.

INTRODUCTION

Alzheimer's disease (AD) is a progressive neurodegenerative disorder that affects millions of people around the world and is one of the main causes of dementia among the elderly. It is characterized by gradual cognitive decline, memory loss, difficulty performing everyday tasks and general functional impairment, which significantly impairs the quality of life of patients and their families. Although the exact causes of AD are still not fully understood, recent studies have revealed that, in addition to the classic beta-amyloid plaques and neurofibrillary tau tangles, neuroinflammation plays a central and multifaceted role in the pathogenesis of the disease (Machado *et al*., 2020). Neuroinflammation, in particular, results from the excessive and chronic activation of glial cells, such as microglia and astrocytes, and is strongly associated with the progression of the disease, exacerbating neuronal damage and promoting synaptic dysfunction. The microglia, which normally act to maintain the neuronal environment, when chronically activated, start to release inflammatory mediators that aggrava-

te oxidative stress, mitochondrial dysfunction and neuronal degeneration (Resende *et al.*, 2022). In addition, astrocytes, which have vital support functions for neurons, can contribute to inflammation in the brain and induce a vicious cycle that further aggravates the progression of AD.

Studies suggest that neuroinflammation may be a phenomenon that precedes the appearance of the clinical symptoms of AD, with initial signs of microglial activation detected many years before the onset of cognitive deficit, which reinforces the importance of early interventions to try to slow down the progression of the disease and possibly even prevent its onset (Prego *et al.*, 2023). Microglial and astrocyte activation results in the excessive release of pro-inflammatory cytokines such as TNF-α, IL-1β, IL-6, as well as other inflammatory mediators such as chemokines and prostaglandins, which play a key role in worsening neurodegeneration and synaptic dysfunction. This inflammatory environment not only contributes to increased oxidative stress and mitochondrial dysfunction, but also impairs the physiological mechanisms for removing beta-amyloid, favoring its accumulation in the brain and exacerbating the formation of plaques, characteristic of AD (Selinger *et al.*, 2024). The persistence of this inflammatory condition, when chronic, compromises the essential functions of neurons, hindering crucial neuroplasticity processes and directly affecting memory and learning, areas typically compromised in Alzheimer's disease.

The interaction between neuroinflammation and neurodegenerative processes has been widely studied and is currently seen as a crucial pathway for the progression of the disease. Modulation of the inflammatory response has thus emerged as a promising therapeutic strategy for the treatment of AD. Recent research has explored different forms of intervention, including the use of inhibitors of pro-inflammatory cytokines, modulators of microglia, and therapies with essential fatty acids, such as omega-3, known for their anti-inflammatory and neuroprotective properties (Bittencourt *et al.*, 2021). Preclinical trials have shown that these approaches can decrease the expression of inflammatory markers in the brain and promote a more favorable neuronal environment, protecting nerve cells from degeneration and programmed cell death. Studies have also indicated that regulating neuroinflammation can help restore synaptic function and improve learning and memory processes, key areas compromised in AD.

In addition, new emerging therapeutic approaches, such as transcranial stimulation and the use of targeted anti-inflammatory drugs, are being evaluated as possible alternatives to curb the neuroinflammatory process in AD (Edin *et al.*, 2022). Although the prospects are promising, translating these findings into clinical practice presents considerable challenges, which include the need for further studies into the efficacy, safety and long-term effects of these interventions. Another crucial point is the variability in inflammatory responses between individuals, which suggests that personalized treatments may be necessary to optimize therapeutic benefits and minimize adverse effects, since the intensity and duration of neuroinflammation can vary significantly between patients. In addition, the identification of specific biomarkers to monitor inflammatory activity and response to treatments would be a major contribution to clinical practice.

This study therefore seeks to discuss neuroinflammation as a central mechanism in the progression of Alzheimer's, addressing not only the pathological aspects, but also exploring new therapeutic perspectives that could bring real benefits to the treatment of the disease. An in-depth understanding of the inflammatory mechanisms involved in AD and advances in neuroscientific research could provide the necessary basis for the development of

more effective and personalized interventions, allowing for a more targeted and efficient therapeutic approach. With continued advances in the field of neuroscience, it is hoped that new therapies may emerge, not only to slow the progression of neuroinflammation, but also to improve the quality of life of patients diagnosed with Alzheimer's disease, providing them with better prospects for treatment and, possibly, prevention (Freitas *et al.*, 2023).

The aim of this study is to analyze neuroinflammation as a central mechanism in the progression of Alzheimer's disease, exploring its relationship with neurodegeneration, cognitive loss and functional impairment. In addition, it seeks to identify and evaluate new therapeutic perspectives aimed at modulating the brain's inflammatory response, such as the use of inflammatory cytokine inhibitors, microglia modulators and anti-inflammatory compounds, in order to slow down the progression of the disease. The study also aims to provide a deeper understanding of the inflammatory processes involved in Alzheimer's disease, contributing to the development of more effective and personalized therapeutic approaches.

METHODOLOGY

The methodology of this study was structured as a review of the scientific literature, with the aim of analyzing recent advances in the understanding of neuroinflammation as a key mechanism in the progression of Alzheimer's disease (AD) and exploring new therapeutic perspectives aimed at treating the disease. Neuroinflammation, recognized as one of the main factors contributing to neuronal degeneration and cognitive loss in AD, has attracted increasing attention in scientific research. This study sought to identify and compile studies that have investigated the relationship between neuroinflammation and neurodegeneration, with a special focus on modulating the inflammatory response in the brain as a potential therapeutic target for slowing or reversing the progression of the disease. In addition, the review aimed to assess how different therapeutic approaches, such as the use of anti-inflammatory drugs, microglia modulators and essential fatty acid-based therapies, can influence not only the progression of AD, but also improve neuronal survival, promote brain tissue regeneration and restore cognitive function. The study also sought to explore the effectiveness of these therapies in early stages of the disease, given that neuroinflammation can occur before clinical symptoms appear, highlighting the importance of early interventions.

The research was conducted between August and December 2024, using the main scientific databases, which are essential for obtaining high-quality peer-reviewed articles. The databases consulted included the Virtual Health Library (VHL), *Latin American and Caribbean Literature in Health Sciences* (LILACS), *Medical Literature Analysis and Retrieval System Online* (MEDLINE) and *Scientific Electronic Library Online* (SCIELO), which are widely recognized for their scope and relevance in the field of health sciences. To ensure the relevance and comprehensiveness of the results, specific Health Sciences Descriptors (DeCS) were used, such as "Neuroinflammation", "Alzheimer's Disease", "Microglia", "Glial Cells", "Cytokines" and "Anti-inflammatory Therapies". These key terms were chosen because of their central relevance to the study of neuroinflammation and its direct relationship with AD. In addition, the additional terms "Modulation of Microglia", "Innovative Therapies" and "Treatment of Alzheimer's Disease" were also incorporated into the search, using the Boolean operator "*AND*" to ensure that the articles selected were directly related to the central focus of the review, providing a rigorous and highly specific selection of relevant studies.

The inclusion criteria for this systematic review were carefully defined to ensure the high quality and relevance of the studies analyzed. Full articles were included, with free access in the selected databases, published between 2020 and 2024, a period that covers the most recent advances in the field of research into neuroinflammation in AD. The studies included in the review needed to directly address the role of neuroinflammation in Alzheimer's disease, especially with a focus on modulating the inflammatory response and its impact on disease progression. In addition, to ensure accessibility and understanding of the results, the articles had to be published in widely understood languages, such as Portuguese, English or Spanish. The exclusion criteria were strict and included the elimination of incomplete articles, articles with restricted or paid access, duplicate articles in the databases, systematic reviews and studies that did not directly address the proposed topic. This process of inclusion and exclusion was fundamental in ensuring that only articles with substantial and relevant data were considered in the final analysis.

The initial search resulted in a total of 1,520 articles, reflecting the breadth of research available on neuroinflammation in AD. After applying the inclusion and exclusion criteria, the sample was reduced to 134 articles that met the established requirements. Each of these articles was then subjected to a detailed and careful analysis, involving assessment of the methodological quality, the results presented and the relevance of the conclusions to the central theme of the study. This process resulted in the final selection of 18 articles, considered suitable for in-depth analysis of neuroinflammation in AD and its therapeutic implications. During the evaluation, the articles were organized into specific categories according to the main topics covered. These categories included the role of neuroinflammation in the progression of AD, the modulation of microglia as a therapeutic target, anti-inflammatory and immunomodulatory therapies, and new therapeutic approaches, such as the use of drugs that stimulate neuroplasticity and neuronal regeneration.

In addition, the selected articles were grouped based on the different therapeutic approaches adopted to treat AD. Among the approaches analyzed were the use of inhibitors of pro-inflammatory cytokines, modulators of microglia, and combined therapies aimed at restoring neuronal function and reducing neuroinflammation in the brain. Categorizing these studies enabled a more detailed analysis of the most recent advances in the development of more effective, less toxic and more targeted treatments for AD, highlighting the crucial role of neuroinflammation as a promising therapeutic target in the fight against Alzheimer's disease. In addition, the methodology adopted allowed a critical analysis of the benefits and limitations of each therapeutic approach, considering the complexity of inflammatory mechanisms and the need for personalized treatments to maximize clinical benefits. The resulting systematic review provides a solid evidence base on neuroinflammation as a key factor in the progression of AD, as well as pointing to the most promising therapeutic perspectives for treating the disease. This detailed analysis contributes to a broader understanding of the opportunities and limitations of anti-inflammatory therapies in AD, providing a solid basis for future research and innovative therapeutic developments.

RESULTS

A review of the literature revealed a strong and consistent association between neuroinflammation and the progression of Alzheimer's disease (AD), highlighting neuroinflammation as one of the central mechanisms responsible for the acceleration of neurodegeneration and cognitive loss observed in this disease. Recent studies confirm that the chronic activation of microglia and astrocytes is a major factor in the amplification of brain inflammation, leading to a worsening of the clinical condition of patients with AD (Santos *et al*., 2024). Neuroinflammation, often a response to cell damage, can occur long before the onset of cognitive symptoms, showing that therapeutic interventions aimed at controlling the brain's inflammatory response can have a significant impact in the early stages of the disease (Souza *et al*., 2023). The excessive activation of glial cells, such as microglia, in response to the accumulation of pathological proteins, such as beta-amyloid and tau tangles, generates an excessive release of inflammatory mediators, such as TNF-α, IL-1β and IL-6. These mediators, in turn, exacerbate oxidative stress and trigger a cascade of neurotoxic events that accelerate neuronal damage and cell death, leading to progressive cognitive dysfunction.

In addition, the review identified that neuroinflammation is also closely related to mitochondrial dysfunction, a phenomenon widely observed in AD patients. The activation of microglia not only amplifies inflammation, but also negatively affects the functioning of mitochondria, compromising energy homeostasis in neuronal cells (Rocha *et al*., 2022). This mitochondrial impairment results in greater production of free radicals and an increase in oxidative stress, factors that exacerbate neurodegeneration. The inability of the brain's immune system, especially microglia, to adequately eliminate beta-amyloid due to the chronic inflammatory response, is a key factor in the accumulation of amyloid plaques in the brain, one of the classic pathological signs of AD (Teixeira *et al*., 2020). This vicious cycle of inflammation and accumulation of neurotoxic proteins results in an increasingly toxic environment for neurons, impairing synaptic function and, consequently, memory and other cognitive functions.

Another significant aspect that the review highlighted is the relationship between neuroinflammation and the loss of synaptic plasticity, an essential factor for the brain's adaptation to new experiences and the consolidation of memories. Persistent inflammation in the brains of Alzheimer's patients has been associated with a decrease in the brain's ability to form new synaptic connections, a fundamental process for learning and memory (Xavier *et al*., 2021). Excessive production of inflammatory mediators such as IL-1β and TNF-α can induce synapse dysfunction, negatively affecting communication between neurons (Melo *et al*., 2022). These inflammatory mediators not only damage nerve cells, but also inhibit the activation of processes necessary for neuroplasticity, limiting the brain's ability to adapt and learn. As a result, neuroinflammation becomes a crucial factor in the progression of cognitive and behavioral symptoms in AD, making it an important therapeutic target for new treatments.

The review also indicated that neuroinflammation interferes with other essential cellular processes, such as the removal of toxic proteins in the brain, including beta-amyloid. Persistent inflammation activates cellular mechanisms that impede the effectiveness of the brain's cleansing systems, such as autophagy, which facilitates the accumulation of these toxic proteins (Bezerra *et al*., 2024). According to Silva *et al*. (2024), neuroinflammation not only increases oxidative stress, but also alters the function of glial cells, which are respon-

sible for removing cellular debris and misfolded proteins. This inefficiency in removing protein fragments is one of the main factors contributing to the formation of amyloid plaques, aggravating neurodegeneration. Therefore, neuroinflammation appears to be both a consequence and a driver of the formation of these plaques, creating a self-perpetuating cycle that accelerates the progress of the disease.

With regard to therapies for the treatment of AD, the review pointed to various therapeutic strategies focused on modulating neuroinflammation as promising. The use of inhibitors of inflammatory cytokines, such as TNF-α, IL-1β and IL-6, has shown potential to reduce the inflammatory response and improve cognitive function in experimental models (Siqueira *et al*., 2021). Research into microglia modulators has also shown that it is possible to regulate the activation of these cells in order to reduce the harmful effects of neuroinflammation and protect neurons. The application of therapies based on essential fatty acids, such as omega-3, has also been identified as a viable strategy, as these compounds have proven anti-inflammatory properties that can mitigate neuroinflammation and promote neuroplasticity (Levada *et al*., 2024). These approaches aim to create a more favorable environment for neuronal health, potentially slowing the progression of AD and improving patients' quality of life.

Finally, the review identified emerging therapies that are being evaluated for the modulation of neuroinflammation in AD, such as non-invasive transcranial stimulation and the use of anti-inflammatory drugs. Preliminary studies suggest that transcranial stimulation may have a modulating effect on brain activity, promoting a reduction in inflammation in the brain and improving cognitive function in AD patients (Lino *et al*., 2024). Although these treatments are still in the experimental phase, the first results indicate that, if successful, they could offer a new line of treatment for patients. The need for more randomized, controlled clinical trials, however, is essential to validate the efficacy and safety of these innovative therapies, especially in more advanced stages of the disease.

DISCUSSION

A review of the literature on neuroinflammation in Alzheimer's disease (AD) confirms the growing evidence that neuroinflammation is one of the central mechanisms that accelerates the neurodegeneration and cognitive loss associated with the disease. Chronic activation of microglia and astrocytes has been shown to be a major factor in amplifying brain inflammation, exacerbating the progression of symptoms and the clinical picture of AD (Santos *et al*., 2024). The study by Souza *et al*. (2023) highlights that neuroinflammation can begin long before the onset of clinical symptoms, which suggests that the inflammatory response plays a significant role in the early pathogenesis of AD. This reinforces the importance of early therapeutic interventions focused on controlling neuroinflammation, with the aim of slowing down the progression of the disease. Microglia, activated by the presence of pathological proteins such as beta-amyloid and tau tangles, release inflammatory mediators which, in addition to exacerbating oxidative stress, trigger a cascade of neurotoxic events that accelerate neuronal damage, thus compromising progressive cognitive function (Souza *et al*., 2023; Santos *et al*., 2024).

In addition, one of the most important findings of the review was the relationship between neuroinflammation and mitochondrial dysfunction. Activated microglia not only amplify the inflammatory response, but also directly affect the function of mitochondria, which are essential for energy production in nerve cells (Rocha *et al*., 2022). This

mitochondrial dysfunction results in greater production of free radicals and an increase in oxidative stress, factors that aggravate neurodegeneration. Failure in mitochondrial function compromises the repair capacity of neuronal cells, making them more vulnerable to damage and cell death (Teixeira *et al*., 2020). This vicious circle, in which inflammation perpetuates mitochondrial dysfunction and vice versa, creates a neurotoxic environment that accelerates the progression of the disease and impairs the patient's cognitive health. According to Rocha *et al*. (2022), improving mitochondrial function could therefore represent a promising therapeutic strategy to combat neurodegeneration in AD.

Another crucial point identified in the review is the impact of neuroinflammation on synaptic plasticity, one of the processes most affected in AD. Neuroplasticity is fundamental for the formation and consolidation of memories, and its loss is directly associated with the cognitive deficits observed in AD patients. Prolonged neuroinflammation in the brain alters the function of synapses, making the learning and memorization process more difficult (Xavier *et al*., 2021). Excessive production of inflammatory mediators such as IL-1β and TNF-α not only damages nerve cells, but also interferes with communication between neurons, impairing the formation of new synaptic connections. Melo *et al*. (2022) point out that chronic inflammation inhibits neuroplasticity by affecting essential processes for regeneration and brain adaptation. With decreased synaptic plasticity, the brain's ability to form new memories and adapt to new experiences is severely limited, which accelerates the progression of cognitive symptoms.

The review also highlights how neuroinflammation interferes with the processes of removing toxic proteins, such as beta-amyloid, which are characteristic of AD. Microglia play a key role in eliminating these proteins, but their efficiency is impaired by chronic inflammatory activation (Bezerra *et al*., 2024). When neuroinflammation persists, the ability of the brain's immune system to clear cellular debris and misfolded proteins decreases considerably, which facilitates the accumulation of beta-amyloid, one of the classic pathological signs of the disease. Silva *et al*. (2024) argue that this accumulation is largely caused by the failure of microglia to perform their "cleaning" function, which directly contributes to the formation of amyloid plaques, intensifying neurodegeneration. The persistence of this cycle of inflammation and accumulation of toxic proteins creates an environment that is increasingly neurotoxic and detrimental to neuronal health, becoming a key factor in the accelerated progression of AD.

In terms of therapies, the review pointed to various therapeutic strategies focused on modulating neuroinflammation, which have shown promising results in the treatment of AD. The use of inhibitors of inflammatory cytokines, such as TNF-α, IL-1β and IL-6, has shown potential to reduce the inflammatory response and improve cognitive function in experimental models (Siqueira *et al*., 2021). These therapies aim to reduce the damaging effects of chronic inflammation on the brain, providing a more favorable environment for neuronal survival and, consequently, slowing the progression of symptoms. In addition, research into microglia modulators suggests that regulating the activation of these cells may be an effective strategy for mitigating the effects of neuroinflammation (Levada *et al*., 2024). The use of anti-inflammatory compounds, such as essential fatty acids, have been shown to have a neuroprotective effect, with properties that can improve neuroplasticity and reduce inflammation in the brain, providing a promising therapeutic approach for AD.

Finally, the review identified emerging therapies that are being evaluated for the treatment of neuroinflammation in AD, such as non-invasive transcranial stimulation and the use of anti-inflammatory drugs. Preliminary studies indicate that transcranial stimulation can have positive effects on reducing inflammation in the brain, promoting an improvement in the cognitive function of AD patients (Lino *et al*., 2024). Although these treatments are still in the experimental stages, the initial results are promising, suggesting that, if successful, they could represent a new line of treatment for AD patients. However, as highlighted by Siqueira *et al*. (2021), it is essential that more randomized controlled clinical trials are conducted to validate the efficacy and safety of these therapies, especially in more advanced stages of the disease. Personalizing treatment, taking into account the variability in the inflammatory response between patients, will be key to maximizing the therapeutic benefits and minimizing the possible adverse effects of these new approaches.

CONCLUSION

Neuroinflammation plays a central role in the progression of Alzheimer's disease, acting as a key mechanism in the acceleration of neurodegeneration and cognitive loss. The chronic activation of glial cells, such as microglia, in response to the accumulation of pathological proteins, results in an excessive release of inflammatory mediators that aggravate oxidative stress and compromise neuronal function. This vicious cycle of inflammation and cell damage contributes significantly to the cognitive dysfunction observed in patients, indicating that modulating the inflammatory response may be a crucial therapeutic strategy for slowing down the progression of the disease.

In addition, the review suggests that neuroinflammation also affects essential cellular processes, such as the removal of toxic proteins in the brain and synaptic plasticity, fundamental factors for preserving cognitive functions. The failure of cellular clearance mechanisms and the decreased ability to form new synaptic connections are closely related to the cognitive deficits observed in Alzheimer's disease. These findings reinforce the importance of therapeutic interventions aimed not only at reducing inflammation, but also at restoring cellular functionality and synaptic plasticity.

Finally, emerging therapies that focus on modulating neuroinflammation have great potential for the treatment of Alzheimer's disease. Strategies that include the use of inflammatory cytokine inhibitors, microglia modulators and anti-inflammatory compounds have shown promising results in experimental models. However, more clinical studies are needed to validate the effectiveness of these approaches in humans, especially in more advanced stages of the disease. As research progresses, it is hoped that new therapies can be developed, offering patients a better quality of life and slowing down the impact of Alzheimer's disease on the brain.

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