

CHRONIC PERIODONTITIS AND ALZHEIMER'S DISEASE: INTERACTIONS BETWEEN ORAL DYSBIOSIS, NEUROINFLAMMATION AND COGNITIVE DEGENERATION FACTORS - LITERATURE REVIEW

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RESUMO

ABSTRACT Introduction: Periodontitis, characterized as a multifactorial chronic inflammatory disease resulting from dental biofilm dysbiosis and an exacerbated host immunoinflammatory response, has been extensively investigated not only for its local impacts but also for its possible relationship with systemic diseases, including Alzheimer's disease (AD). Recent evidence suggests that periodontal pathogens and periodontal inflammatory mediators may contribute to neuroinflammatory and neurodegenerative processes. Objective: To analyze the relationship between chronic periodontal inflammation, mechanisms of oral dysbiosis, and their possible contribution to the progression of Alzheimer's disease. Materials and Methods: This study consists of a narrative literature review, conducted using publications indexed in the PubMed database over the last two years. The search terms used were "periodontitis" and "Alzheimer's," including original articles, systematic reviews, and meta-analyses. Results: Periodontitis develops from an imbalance in the oral biofilm, favoring the proliferation of pro-inflammatory microbial agents, mainly *Porphyromonas gingivalis*, capable of deregulating immunological mechanisms, perpetuating local inflammation, and releasing endotoxins such as lipopolysaccharides (LPS). LPS are components of the outer membrane of periodontopathogenic Gram-negative bacteria and are released into the systemic circulation, playing a central role in inducing inflammatory responses through the activation of Toll-like receptor 4 (TLR4). Recurrent exposure to LPS promotes systemic inflammation, microglial activation, and neuroinflammation. The identification of these biomolecules in brain regions of patients with Alzheimer's disease reinforces their possible participation in the formation of beta-amyloid plaques, tauopathies, and neurodegenerative progression, associating them with cognitive decline. Furthermore, genetic factors linked to inflammation, such as alterations in genes of pro-inflammatory cytokines, were identified in common in periodontitis and Alzheimer's disease, reinforcing the relationship between the two.

Conclusion: Chronic periodontal inflammation has the potential to play a modifiable risk factor for Alzheimer's disease through mechanisms involving oral dysbiosis, systemic dissemination of inflammatory mediators, and possible brain invasion by periodontal pathogens. Thus, preventive and therapeutic strategies aimed at maintaining periodontal health can contribute not only to the preservation of oral health but also to the reduction of systemic inflammation and the risk of progression of neurodegenerative diseases, such as AD.

Palavras-chave: Periodontitis; Alzheimer's Disease; Chronic Inflammation; Oral Dysbiosis.