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World Congress on

Dementia, Alzheimer's, Parkinson's & Neurodegenerative Disorders

July 09-10, 2026 | Frankfurt, Germany

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Lactoferrin and Altered Immune Response: Novel Insights into the Link Between Periodontitis and Alzheimer's disease

Periodontal disease has been associated with an increased risk of developing Alzheimer's disease. Traditionally, this relationship has been attributed to chronic systemic inflammation and/or the hematogenous dissemination of oral bacteria, both of which may potentiate neuroinflammatory processes. In this context, multiple studies have suggested that periodontal pathogens and their inflammatory mediators could contribute to the cerebral accumulation of β-amyloid plaques—a pathological hallmark of Alzheimer's disease—thereby establishing a plausible biological link between the two conditions. The identification of early biomarkers for Alzheimer's disease has become a public health priority in light of the global rise in dementia prevalence. Within this framework, salivary lactoferrin—an immunomodulatory glycoprotein primarily produced by neutrophils—has been shown to exhibit a marked reduction from the earliest stages of the disease, positioning it as a potential early diagnostic biomarker for Alzheimer's disease. Conversely, lactoferrin is overexpressed in the general population affected by periodontitis. In parallel, several hematological indices derived from routine blood counts, such as the neutrophil-to-lymphocyte ratio (NLR) and the systemic immune-inflammation index (SII, calculated from neutrophil, lymphocyte, and platelet counts), have been proposed as accessible markers of peripheral inflammation in both dementia and periodontal disease.

Our findings indicate that, overall, neither NLR nor SII shows significant differences between patients with dementia and healthy controls, nor between individuals with Alzheimer's disease and those with non-Alzheimer dementias. However, within the non-Alzheimer dementia subgroup, the presence of periodontitis is associated with a significant increase in both NLR and SII, attributable to elevated circulating neutrophil counts. In contrast, in Alzheimer's disease, periodontal status does not modify inflammatory indices, suggesting the presence of early immune dysregulation. The absence of a detectable peripheral inflammatory response in patients with Alzheimer's disease and concomitant periodontitis—despite the chronic inflammatory burden imposed by the latter—together with the early deficit in salivary lactoferrin, supports the hypothesis of an early systemic immune alteration. This pattern may reflect dysfunction in the activation or regulation of innate immunity, particularly within the neutrophil lineage, thereby limiting the peripheral expression of inflammation. In this context, reduced lactoferrin levels may function not only as an early biomarker of Alzheimer's disease but also as a potential indicator of immune dysregulation capable of modulating the biological interplay between periodontal inflammation and the neurodegenerative process.

Keywords: Alzheimer's disease, periodontitis, lactoferrin, innate immune dysregulation

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Biography

I am a researcher in special care dentistry, with a particular focus on patients with dementia and other cognitive impairments. My research focuses on identifying salivary biomarkers for the early diagnosis of Alzheimer's disease. My work also centers on improving oral healthcare strategies and clinical protocols for vulnerable populations.

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