Biofilm-Induced Immune Modulation in Periodontal and Oral Mucosal Diseases: A Comparative Study

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ABSTRACT:

Oral biofilms are key etiological factors in periodontal and oral mucosal diseases due to their ability to dysregulate host immune responses and perpetuate chronic inflammation. The resulting inflammatory burden contributes significantly to oral disease progression and adversely affects overall health and well-being. This study aimed to evaluate the relationship between biofilm accumulation and immune modulation among healthy individuals and patients with periodontitis or oral mucosal diseases. Clinical periodontal parameters and salivary inflammatory cytokines were assessed and compared across groups. Diseased individuals exhibited significantly greater plaque accumulation, accompanied by elevated salivary levels of IL-1β, TNF-α, and IL-6, compared with healthy controls, indicating an enhanced pro-inflammatory response associated with disease status. These findings highlight the pivotal role of biofilm-driven immune dysregulation in the pathogenesis and progression of oral diseases and reinforce the importance of effective biofilm control in maintaining oral health. Furthermore, the study supports the advancement of Sustainable Development Goal (SDG) 3 (Good Health and Well-being) through disease prevention, SDG 4 (Quality Education) by informing evidence-based oral health promotion, SDG 9 (Industry, Innovation and Infrastructure) through biomarker-driven translational research. Furthermore, advancing knowledge in this area requires collaborative efforts between clinicians, researchers, and public health stakeholders, reflecting the principles of SDG 17 (Partnerships for the Goals).

Keywords: Host–Microbe Interaction, Immune Modulation, Inflammatory Cytokines, Mucosal Immunity, Oral Biofilm, Oral Health, Oral Microbiome, Oral Mucosal Diseases, Periodontal Inflammation, Periodontitis, SDG 3, SDG4, SDG 9, SDG 17.

Introduction

The oral cavity harbours one of the most diverse and complex microbial ecosystems in the human body, comprising highly specialized bacterial, fungal, viral, and archaeal communities that inhabit distinct oral niches. These microorganisms exist predominantly as structured biofilms that colonize the dental surfaces, gingival tissues, tongue dorsum, and oral mucosa. Under physiological conditions, a dynamic and mutually beneficial relationship between the resident oral microbiota and the host immune system maintains oral homeostasis and contributes to tissue health. Disruption of this ecological balance, however, can lead to microbial dysbiosis, characterized by shifts in biofilm composition and function that favour the proliferation of pathogenic species. In the absence of effective host surveillance and biofilm control, these dysbiotic communities can trigger aberrant immune responses and chronic inflammation, resulting in the initiation and progression of periodontal diseases. The intricate interactions between oral biofilms and host immunity are therefore central to the pathogenesis of periodontitis and other inflammatory oral diseases, highlighting the importance of understanding biofilm-mediated immune modulation in maintaining oral health. [1].

The biofilm lifestyle is characterized by highly organized microbial communities embedded within a self-produced extracellular matrix that protects resident microorganisms from environmental challenges and host immune defences. Biofilm-associated microorganisms establish persistent interactions with oral epithelial cells, modulating innate and adaptive immune responses and altering local immune homeostasis. Both acute and chronic infections elicit complex immune reactions involving mast cells, neutrophils, dendritic cells, T lymphocytes, and B lymphocytes. These responses are initiated through the recognition of microbial antigens and pathogen-associated molecular patterns (PAMPs), triggering signaling cascades that regulate cytokine production, antibody release, and inflammatory responses. [2]. Beyond the oral cavity, these interactions are increasingly recognized as components of broader host–microbiome communication networks that influence systemic immune homeostasis [3]. The mucosal immune system plays a critical role in maintaining tolerance to commensal microorganisms while mounting effective responses against pathogens. Oral epithelial cells serve as the first line of defence by providing a physical barrier and secreting antimicrobial peptides, chemokines, and cytokines that regulate local immune responses and microbial homeostasis [4]. Oral microbial biofilms can disrupt mucosal homeostasis by altering epithelial barrier function and modulating host immune signaling through direct interactions with mucosal tissues and the release of microbial metabolites and virulence factors [5]. Persistent biofilm accumulation may impair protective host mechanisms, leading to dysregulated immune responses and chronic inflammation. Among biofilm-associated conditions, periodontitis is one of the most prevalent chronic inflammatory diseases worldwide and is primarily initiated by dysbiotic subgingival biofilms. However, the connective tissue destruction and alveolar bone resorption characteristic of periodontitis are largely driven by an exaggerated host immune-inflammatory response rather than by microbial challenge alone [6]. Biofilm-derived components activate

epithelial cells, neutrophils, macrophages, and lymphocytes, stimulating the production of pro-inflammatory mediators that amplify local tissue damage. Although antimicrobial peptides and innate immune defense mechanisms play essential roles in maintaining microbial homeostasis, prolonged exposure to dysbiotic biofilms may alter their expression, impair complement-mediated responses, and perpetuate inflammatory pathways, thereby contributing to disease initiation and progression [7].

Emerging evidence highlights the pivotal role of epithelial barrier integrity in regulating host–microbiome interactions and maintaining oral mucosal homeostasis. Disruption of epithelial permeability can facilitate microbial translocation, enhance immune activation, and initiate a self-perpetuating cycle of inflammation and tissue damage [8]. Consequently, barrier-associated immune responses are critical for preserving mucosal health and preventing the development of chronic inflammatory diseases [9]. Bidirectional communication between mucosal surfaces and resident immune cells further governs the balance between protective immunity and pathological inflammation, thereby influencing disease susceptibility and progression [10]. Understanding the mechanisms by which oral biofilms modulate host immune responses is therefore fundamental to elucidating the pathogenesis of periodontal and oral mucosal diseases. A comprehensive evaluation of the complex interactions between polymicrobial biofilms and the host immune system may provide valuable insights into disease mechanisms and facilitate the development of innovative preventive, diagnostic, and therapeutic strategies aimed at restoring microbial and immune homeostasis. Therefore, this study aimed to assess the relationship between biofilm accumulation and immune modulation in healthy individuals and patients with periodontitis or oral mucosal diseases.

Methodology

This cross-sectional observational study was conducted among patients attending the Departments of Periodontology, Oral Medicine and Radiology, and Oral and Maxillofacial Pathology and Oral Microbiology. The primary objective was to evaluate biofilm accumulation and its potential immunomodulatory effects in individuals with periodontal disease and oral mucosal diseases compared with healthy controls.

Participants were recruited consecutively and allocated into one of three study groups based on their clinical diagnosis: (i) healthy controls, (ii) periodontal disease patients, and (iii) patients presenting with oral mucosal diseases. Written informed consent was obtained from all participants prior to enrolment. Demographic information and relevant clinical data were recorded using a standardized data collection form.

A comprehensive periodontal examination was performed by calibrated investigators. Clinical parameters assessed included Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Loss (CAL). Oral biofilm samples were collected aseptically from representative sites using sterile curettes and transported immediately to the laboratory for microbiological analysis under standardized conditions. Unstimulated whole saliva samples were also obtained for the assessment of inflammatory and immune-related biomarkers.

Microbiological analysis was performed to characterize biofilm-associated microbial profiles using conventional culture-based methods and, where applicable, molecular techniques. Host immune responses were evaluated by quantifying selected inflammatory cytokines, including interleukin (IL)-1β, IL-6, and tumour necrosis factor-alpha (TNF-α), together with antimicrobial peptides (AMPs) and other relevant immune mediators. The relationship between biofilm burden and immune modulation was investigated through

correlations between clinical periodontal parameters, microbial findings, and immunological biomarkers.

Data were entered into Microsoft Excel and analysed using appropriate statistical software. Descriptive statistics were used to summarize demographic, clinical, microbiological, and immunological characteristics. Intergroup comparisons were performed using appropriate parametric or non-parametric tests according to data distribution. Correlation analyses were conducted to evaluate associations between biofilm-related parameters and immune biomarkers. Statistical significance was established at $p < 0.05$.

The study protocol was reviewed and approved by the Institutional Ethics Committee and was conducted in accordance with the principles as outlined in the Declaration of Helsinki.

Results

A total of 90 participants were enrolled in the study and equally allocated into three groups: healthy controls (n = 30), periodontitis patients (n = 30), and oral mucosal disease patients (n = 30). Demographic characteristics, clinical periodontal parameters, and salivary inflammatory biomarkers were analysed to investigate the association between biofilm accumulation and host immune responses.

The demographic characteristics of the study population are summarized in Table 1. The mean age was 32.4 ± 6.8 years in the healthy control group, 45.7 ± 8.2 years in the periodontitis group, and 42.3 ± 7.5 years in the oral mucosal disease group. Participants with periodontitis and oral mucosal diseases were older than healthy controls.

Sex distribution was comparable across the study groups. Males represented 53.3% (n = 16) of the healthy control group, 60.0% (n = 18) of the periodontitis group, and 56.7% (n = 17) of the oral mucosal disease group, while females accounted for 46.7% (n = 14), 40.0% (n = 12), and 43.3% (n = 13), respectively. No marked differences in sex distribution were observed among the groups.

Table 1: Demographic Characteristics of the Study Population

Variable	Healthy Controls (n=30)	Periodontitis (n=30)	Oral Mucosal Disease (n=30)
Mean Age (years)	32.4 ± 6.8	45.7 ± 8.2	42.3 ± 7.5
Male, n (%)	16 (53.3)	18 (60.0)	17 (56.7)
Female, n (%)	14 (46.7)	12 (40.0)	13 (43.3)

The comparison of clinical periodontal parameters across the study groups is presented in Table 2. Significant differences were observed in plaque accumulation and gingival inflammation among healthy controls, periodontitis patients, and oral mucosal disease patients.

The mean Plaque Index (PI) was significantly higher in the periodontitis group (2.34 ± 0.54) compared with the oral mucosal disease group (1.89 ± 0.47) and healthy controls (0.82 ± 0.31) ($p < 0.001$). Healthy participants exhibited the lowest plaque scores, whereas individuals with periodontitis demonstrated the greatest biofilm accumulation.

A similar trend was observed for the Gingival Index (GI). The highest mean GI was recorded in the periodontitis group (2.51 ± 0.49), followed by the oral mucosal disease group (1.76 ± 0.42), while healthy controls showed the lowest values (0.75 ± 0.28). Differences among the groups were statistically significant ($p < 0.001$), indicating progressively greater gingival inflammation in association with disease status.

Overall, both plaque accumulation and gingival inflammation were significantly elevated in participants with periodontal and oral mucosal diseases compared with healthy controls, with the highest values consistently observed in the periodontitis group.

Mean probing pocket depth (PPD) differed significantly among the study groups (Table 2). Healthy controls exhibited the lowest mean PPD (2.1 ± 0.4 mm), consistent with periodontal health. In contrast, the periodontitis group demonstrated a markedly greater mean PPD (5.8 ± 1.2 mm), indicative of substantial periodontal breakdown. Participants with oral mucosal diseases showed intermediate PPD values (3.2 ± 0.8 mm), which were higher than those observed in healthy controls but lower than those recorded in the periodontitis group. The intergroup differences were statistically significant ($p < 0.001$).

Collectively, the clinical periodontal findings demonstrated progressively higher levels of plaque accumulation, gingival inflammation, and periodontal tissue destruction in the disease groups compared with healthy controls. Among all groups, patients with periodontitis consistently exhibited the most severe clinical periodontal impairment.

Table 2: Clinical Periodontal Parameters Among Study Groups

Parameter	Healthy Controls	Periodontitis	Oral Mucosal Disease	p-value
Plaque Index	0.82 ± 0.31	2.34 ± 0.54	1.89 ± 0.47	<0.001
Gingival Index	0.75 ± 0.28	2.51 ± 0.49	1.76 ± 0.42	<0.001
Probing Pocket Depth	2.1 ± 0.4	5.8 ± 1.2	3.2 ± 0.8	<0.001

The salivary concentrations of inflammatory cytokines are presented in Table 3. Significant differences in cytokine expression were observed among the study groups.

Mean salivary IL-1β levels were significantly higher in the periodontitis group (52.7 ± 10.6 pg/mL) than in the oral mucosal disease group (43.8 ± 8.9 pg/mL) and healthy controls (18.4 ± 4.2 pg/mL) ($p < 0.001$). Similarly, TNF-α concentrations differed significantly across groups, with the highest levels detected in periodontitis patients (39.4 ± 7.8 pg/mL), followed by oral mucosal disease patients (31.6 ± 6.7 pg/mL) and healthy controls (12.1 ± 3.5 pg/mL) ($p < 0.001$).

A comparable pattern was observed for IL-6. Mean IL-6 concentrations were greatest in the periodontitis group (28.3 ± 5.9 pg/mL), intermediate in the oral mucosal disease group (24.7 ± 5.2 pg/mL), and lowest among healthy controls (8.6 ± 2.7 pg/mL). The differences among the groups were statistically significant ($p < 0.001$).

Overall, patients with periodontitis and oral mucosal diseases exhibited significantly elevated salivary levels of IL-1β, TNF-α, and IL-6 compared with healthy controls, with the highest concentrations consistently observed in the periodontitis group.

Table 3: Comparison of Salivary Inflammatory Cytokines Among Study Groups

Biomarker	Healthy Controls	Periodontitis	Oral Mucosal Disease	p-value
IL-1β (pg/mL)	18.4 ± 4.2	52.7 ± 10.6	43.8 ± 8.9	<0.001
TNF-α (pg/mL)	12.1 ± 3.5	39.4 ± 7.8	31.6 ± 6.7	<0.001
IL-6 (pg/mL)	8.6 ± 2.7	28.3 ± 5.9	24.7 ± 5.2	<0.001

The association between biofilm accumulation and inflammatory activity was evaluated by correlating Plaque Index (PI) scores with salivary IL-1β concentrations (Table 4). Correlation analysis demonstrated a strong positive relationship between PI and IL-1β levels ($r = 0.68$, $p < 0.001$).

Higher plaque scores were associated with increased salivary IL-1β concentrations, indicating a significant relationship between biofilm burden and inflammatory biomarker expression. The observed correlation remained statistically significant, suggesting that greater biofilm accumulation was accompanied by enhanced local inflammatory activity.

Table 4: Correlation Between Plaque Index and IL-1β Levels

Variables	Correlation Coefficient (r)	p-value
Plaque Index vs IL-1β	0.68	<0.001
Variables	Correlation Coefficient (r)	p-value

The present study demonstrated that patients with periodontitis and oral mucosal diseases exhibited significantly higher plaque accumulation, gingival inflammation, probing pocket depth, and salivary inflammatory cytokine levels compared with healthy controls. Periodontitis patients consistently showed the highest clinical and immunological values among all groups. Furthermore, a significant positive correlation between plaque accumulation and IL-1β expression highlighted the close relationship between microbial biofilms and host inflammatory responses.

These findings collectively suggest that biofilm-induced immune modulation plays a critical role in the initiation and progression of periodontal and oral mucosal disease

Discussion

The present study investigated the relationship between oral biofilm accumulation, clinical periodontal status, and inflammatory immune responses in healthy individuals, patients with periodontitis, and patients with oral mucosal diseases. The findings demonstrated significantly greater plaque accumulation, gingival inflammation, probing pocket depth, and salivary concentrations of pro-inflammatory cytokines in the disease groups compared with healthy controls. These results support the concept that oral biofilms play a central role in modulating host immune responses and contribute to the pathogenesis of both periodontal and oral mucosal diseases.

Oral biofilms are highly organized polymicrobial communities that exist in dynamic interaction with host tissues. Under physiological conditions, a balanced relationship between the resident oral microbiota and the host immune system is essential for maintaining oral homeostasis. Disruption of this equilibrium through alterations in microbial composition, increased biofilm maturation, or ecological shifts within the oral environment can lead to dysbiosis, triggering inflammatory pathways that promote tissue damage and disease progression. Emerging evidence indicates that dysbiotic biofilms not only increase the microbial burden but also modify host immune regulation, thereby sustaining chronic inflammation and impairing tissue repair mechanisms.

The significantly higher plaque index observed in the periodontitis and oral mucosal disease groups in the present study is consistent with previous reports identifying biofilm accumulation as a primary etiological factor in the initiation and progression of oral inflammatory diseases. Increased biofilm burden provides a persistent source of microbial antigens, virulence factors, and pathogen-associated molecular patterns that stimulate epithelial cells and immune effector cells,

resulting in the production of inflammatory mediators. These findings are in agreement with those of Ebersole et al. [1], who reported that alterations in the oral microbiome profoundly influence immune regulatory pathways and contribute to the development and persistence of chronic inflammatory periodontal diseases.

The observation that patients with oral mucosal diseases also exhibited higher plaque scores than healthy controls suggests that biofilm-associated immune activation may not be restricted to periodontal tissues alone. Rather, oral biofilms may influence broader mucosal immune networks, contributing to inflammatory processes across different oral disease states. This concept aligns with the growing recognition of the oral cavity as an integrated mucosal ecosystem in which host–microbiome interactions play a critical role in maintaining tissue homeostasis and determining disease susceptibility.

The oral epithelium acts as an important barrier that protects deeper tissues from invasion by microorganisms. Microorganisms within a biofilm can impact epithelial barrier integrity and modulate immune signaling pathways. Takiishi et al. emphasized the critical role of epithelial barrier function in regulating immune homeostasis and preventing chronic inflammatory responses [2]. Similarly, Partida-Rodríguez et al. stated that microbial communities have direct and ongoing interactions with mucosal immune systems, shaping both local- as well systemic immunity [3]. Ahluwalia et al. actually showed that microbial dysbiosis can impair barrier function and favour the development of inflammatory diseases via immune deregulation [4]. This could account for the increased inflammatory responses that we observed in both disease groups of this study.

Oral inflammation and periodontal tissue destruction are reflected by the markedly increased gingival index in addition to probing pocket depth among periodontitis patients. Microbial products derived from biofilms trigger host cells to secrete pro-inflammatory mediators, promoting immune cell recruitment and amplifying the inflammatory response. Ghosh et al. described the impacts of interactions between microbial communities and host immunity on disease susceptibility and progression of inflammation [5]. The results of the current study are consistent with these latter findings indicating more biofilm accumulation to correlate with clinical severity of disease.

Host immune responses are key contributors to periodontal tissue destruction. While microbial biofilms trigger the inflammatory response, a large proportion of tissue destruction are due to host immune overactivity or dysregulation. McCormick et al. says that chronic exposure to periodontal pathogens is responsible for the persistent activation of innate and adaptive immune responses, which results in connective tissue destruction and bone resorption [6]. In line with the finding of immune-mediated tissue destruction related to disease progression, significantly deeper probing pocket depths have been observed in periodontitis patients participating in fuzzy forest combined analysis.

Oral antimicrobial peptides (AMPs) and innate immune defenses play critical roles in maintaining oral microbial homeostasis. However, persistent biofilm challenge can alter the expression and protective functions of these molecules. As reported by Diamond et al., AMPs are small cationic peptides that possess both antimicrobial and immunomodulatory properties, contributing to the regulation of host defense and inflammatory responses [7]. Disruption of these protective mechanisms may promote biofilm persistence and sustained inflammation, potentially contributing to the elevated inflammatory biomarker levels observed in the disease groups.

The present study demonstrated significantly higher salivary concentrations of IL-1β, TNF-α, and IL-6 in patients with periodontitis and oral mucosal diseases compared with healthy

controls. These cytokines are key mediators of inflammatory signaling and play pivotal roles in immune regulation and tissue destruction. Previous studies by Pardo-Camacho et al. have shown that impaired epithelial barrier function enhances cytokine production and immune activation [8], while Allaire et al. emphasized the importance of epithelial barrier integrity in regulating host–microbial interactions and limiting excessive inflammatory responses [9]. Accordingly, the elevated cytokine levels observed in the present study may reflect the consequences of chronic biofilm exposure and disrupted mucosal homeostasis.

Dynamic interactions between microbial communities and host immune cells are essential for maintaining oral health; however, dysregulation of these interactions can drive chronic inflammation. Andrews et al. demonstrated that microbial signals play a crucial role in shaping mucosal immune responses and that disturbances in these regulatory pathways contribute to inflammatory disease development [10]. The markedly elevated cytokine levels identified in the disease groups further suggest that biofilm-associated immune dysregulation is an important mechanism underlying the pathogenesis of periodontal and oral mucosal diseases.Human studies investigating innate and adaptive immune mechanisms in periodontal disease also support the findings of the present study. Buckley et al. discussed the significance of immune regulation at mucosal surfaces in evolution and cause cytokine-mediated responses to regulate microbial populations [11]. Suk et al. which indicated that host inflammatory responses were important determinants of the severity and progression of periodontal disease [12]. Similarly, Montreekachon et al. have shown that dysregulated production of cytokines plays a major role in the pathogenesis of chronic inflammatory munosal diseases [13].

Microorganisms associated with biofilms have been reported to produce numerous virulence factors that activate inflammatory signaling pathways. Dickinson et al. have found that fungal metabolites and signaling molecules can modulate host cellular responses, contributing to inflammation or tissue damage [14].

More recently, Phanrungsuwan et al. demonstrated how the interactions between microbial biofilms and immune cells are intricate, that is critical for chronic inflammatory diseases and modulation of immunity [15]. These observations also confirm our biological mechanisms observed in the current study.

Poor oral health and related chronic diseases has persisted globally since at least the 2000s, with periodontal disease still being one of the most frequently diagnosed inflammatory non-communicable conditions worldwide [3]. Epidemiological studies by Eke and Thornton-Evans revealed that periodontal disease represents a considerable burden among adults [16]. Righolt et al. reported socio-economic and health impacts of untreated oral diseases [17]. Moreover, oral diseases were recognised as significant contributors to disability and impaired quality of life, ranked third globally [18]. These findings emphasize the importance of understanding biofilm-host interactions in regard to better prevention and management strategies for diseases.

Dental plaque biofilm was first conceptualized by P.D Marsh, who emphasized that disease is not purely a consequence of the presence per se of different microorganisms [19]. This ecological plaque hypothesis is still valid and offers a conceptual framework for the association established between biofilm accumulations, immune dysregulation as identified in this study.

As Van Dyke pointed out, the host's inflammatory responses are a key driver of periodontal disease progression as opposed to microbial burden alone [20]. In the same manner, systemic and environmental factors are also responsible for immune-mediated periodontal destruction [21], as highlighted by

Iacopino. Kikuchi go on to further prove that the inflammation mediators took a role in metabolic and tissue destroying. [22] These mechanisms could illustrate the greater probing pocket depth and inflammatory biomarkers corresponding to periodontitis in our study.

Previous studies done by Gafar showed that the clinical importance of plaque accumulation in periodontal disease severity [23]. Karimbux placed the understanding of host and microbial interactions at the forefront of contemporary periodontal research and education [24]. Kamen also wrote that periodontal disease can be controlled only by inhibiting both biofilm and the inflammatory reaction of host [25]. Those observations are consistent with the current findings and suggest that a theoretically effective therapeutic must antagonize both microbial biofilms and host immune pathways.

In summary, current evidence shows a compelling association between immune activation and biofilm accumulation in the etiology of periodontal disease and probably other mucosal diseases. The also notably elevated clinical parameters and inflammatory cytokine levels as well, provide evidence that biofilm-induced immune modulation is important for the disease initiation or progression since there was a positive correlation between plaque index and IL-1β expression. An improved understanding of these interactions may enable the identification and application of targeted therapeutic approaches that restore microbial eubiosis, immune homeostasis, whilst minimizing tissue destruction and disease burden.

Advancement of Knowledge

This study advances current understanding of the role of oral biofilms in modulating host immune responses in periodontal and oral mucosal diseases. The observed association between biofilm accumulation and elevated pro-inflammatory cytokine levels highlights the contribution of immune dysregulation to disease progression and supports the development of integrated therapeutic approaches targeting both microbial biofilms and host inflammatory pathways. The findings align directly with Sustainable Development Goal (SDG) 3: Good Health and Well-Being by contributing to the prevention, diagnosis, and management of chronic oral inflammatory diseases and their broader health implications. The study also supports SDG 9: Industry, Innovation and Infrastructure through its contribution to translational research on biofilm–host interactions and the development of innovative biomarkers, diagnostic tools, and therapeutic strategies. Furthermore, it advances SDG 4: Quality Education by strengthening evidence-based knowledge in periodontology, oral pathology, microbiology, and immunology, thereby supporting academic research and professional training. The findings also contribute to SDG 17: Partnerships for the Goals by fostering interdisciplinary collaboration among various dental, microbiological, immunological, and public health researchers.

Conclusion

The present study confirmed the association between increased oral biofilm burden and heightened inflammatory and immune responses in periodontal and oral mucosal diseases. Compared with healthy controls, affected individuals exhibited significantly greater periodontal clinical parameters and elevated levels of pro-inflammatory cytokines. These findings underscore the pivotal role of biofilm-driven immune dysregulation in disease pathogenesis and highlight the importance of effective biofilm control in the prevention and management of oral inflammatory diseases, also aligning with SDG's 3, 4, 9, and 17.

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