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Periodontitis; Oral microbiome; Dysbiosis; Systemic inflammation; Inflammatory biomarkers

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Association Between Periodontal Inflammation, Oral Microbiome Dysbiosis, and Systemic Inflammatory Biomarkers in Adults: A Multicenter Clinical Study

Abstract
Oral microbiome (OM) dysbiosis and periodontal inflammation may be sources of systemic inflammatory activity. Nonetheless, conjunctive clinical evidence that measures periodontal status, changes in microbes, and circulating inflammatory biomarkers (IB) is scarce. To assess the correlation between periodontal inflammation, oral microbiome dysbiosis and systemic inflammatory biomarkers in adults. It was a multicenter retrospective study involving 80 periodontally healthy (n = 20), gingivitis (n = 25), and periodontitis (n = 35) adults. Periodontal parameters were probing pocket depth, clinical attachment loss, bleeding on probing and plaque index. Microbiological results were microbiome dysbiosis index and abundance of Porphyromonas gingivalis, and systemic biomarkers (CRP, IL-6 and TNF-alpha). Correlation and regression analyses were performed. Periodontal parameters, dysbiosis of the microbiome, abundance of P. gingivalis, and systemic inflammatory biomarkers were significantly higher depending on the severity of periodontal disease. It was found that there are strong positive relationships between periodontal parameters and microbiome measures and microbial dysbiosis and systemic inflammatory biomarkers. The association between bleeding on probing and microbiome dysbiosis and CRP, IL-6, and TNF-6 was independent of age and sex, after adjustment. Higher levels of periodontal inflammation and dysbiosis of the oral microbiome were linked to higher levels of systemic inflammatory activity. These results point out mutual periodontal, microbial, and systemic inflammatory mechanisms and suggest additional prospective research of their clinical value.

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INTRODUCTION

Periodontal disease is a common chronic inflammatory disorder that progresses between reversible gingivitis to periodontitis that is characterized by gradual destruction of the tooth-supporting tissues. In addition to local outcomes, ongoing periodontal inflammation has been shown to have systemic biological changes, highlighting the role of oral health in the overall health (Foroughi et al., 2025; Nayak et al., 2025). Clinical indicators, including probing pocket depth (PPD), clinical attachment loss (CAL), bleeding on probing (BOP) and plaque index, are used to determine the burden of disease, whereas periodontal progression demonstrates interrelationships between dental plaque, microbial communities, and host immune reactions (Guo et al., 2024; Kwon et al., 2026). The normal oral microbiome (OM) is in relative balance with the host tissues but any disturbed balance may result in microbial dysbiosis and facilitation of periodontal inflammation. The presence of dysbiotic communities can boost the pathogenic activity, increase immune response, and lead to the destruction of periodontal tissue (Jimenez de Nunzio et al., 2026). The inflammatory mediator changes have also been linked to severe periodontal dysbiosis, which is indicative of interactions between microbial imbalance and host inflammation (Kawamoto et al., 2021). Porphyromonas gingivalis, especially, is the periodontal pathogen that is relevant due to its potential to influence the dysbiotic communities and the ability to alter the host-microbial relationships.

Periodontal pathogens may have a biological role that goes beyond oral cavity. The changes of OM have been linked to hepatic illness, and the directional interaction between periodontal pathogens and systemic microbial conditions is suggested (Dioguardi et al., 2025). Mechanisms of periodontal inflammation have also been associated with mechanisms of non-alcoholic fatty liver disease (Lyu et al., 2025). These findings are in line with the overall idea that periodontal microbial disturbances may engage with systemic inflammatory, metabolic and microbial pathways as opposed to stay within the periodontal tissues. Periodontal inflammation has the potential to affect systemic health due to the spread of microbes, the circulation of microbial products and the release of inflammatory mediators by periodontal tissues. Vascular pathological processes have been yet again linked to OM-mediated inflammation (Gualtero et al., 2023), with the possible linking of periodontal and cardiovascular diseases relying on molecular interactions between immune activation and inflammatory signaling (Dhungana et al., 2025). The systemic inflammatory biomarkers (IB) are thus especially pertinent when assessing periodontal-systemic relationships. C-reactive protein (CRP), interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-alpha) give quantifiable evidence of systemic inflammatory response.

Continuous periodontal microbial stimulation can be a factor in systemic inflammatory signaling via chronic host immune activation. The pathways of dysbiosis-related periodontitis and systemic inflammatory diseases have been associated with cytokine (Irie et al., 2023). Metabolic and cardiovascular complications related to diabetes have also been considered to be related to periodontal inflammation (Fang et al., 2025). Moreover, OM changes were also associated with chronic kidney disease by inflammatory and metabolic pathways (Fang et al., 2026) and initial studies have indicated that oral microbial communities may be connected to reproductive health (Marcickiewicz et al., 2025). Periodontal dysbiosis systemic implications can also be through neurological pathways. The oral-gut-brain axis offers a mechanistic model of the links between periodontal dysbiosis and systemic inflammation (SI) and neuroinflammation (Dumitru et al., 2026). Similar studies on salivary and microbial biomarkers have also been conducted in the associations of periodontal disease with neurodegenerative conditions (Malcangi et al., 2025). Pediatric populations also suggest possible links between oral dysbiosis and systemic inflammatory reactions (Inchingolo et al., 2026), and family patterns of oral–gut dysbiosis and systemic indicators have been found in those with periodontitis (Paz et al., 2026).

Although such evidence is increasing, it is important to have an integrated assessment of periodontal inflammation, OM dysbiosis, as well as systemic IB in the same adult population. Concurrent evaluation can shed light on the idea of increased periodontal load being associated with microbial derailment and SI. Hence, this multicenter study examined how periodontal parameters, microbiome dysbiosis, P. gingivalis abundance, CRP, IL-6, and TNF-a relate and whether periodontal inflammation and microbiome dysbiosis are independently related to systemic inflammatory status following demographic adjustment.

Objectives of the Study

- To compare periodontal inflammation, oral microbiome dysbiosis, and systemic inflammatory biomarkers across healthy, gingivitis, and periodontitis groups.
- To assess the associations between periodontal inflammation, microbiome dysbiosis, and systemic inflammatory biomarkers after adjustment for age and sex.

2. METHODOLOGY

2.1 Study Design and Population

A multicenter retrospective observational study was used to examine the relationship between periodontal inflammation, dysbiosis of the OM, and systemic IB with adults. Retrospective evaluation was done with clinical and laboratory records of 80 eligible participants. The periodontal status was classified into healthy (n = 20), gingivitis (n = 25), and periodontitis (n = 35) groups. The available records were used to extract demographic data, periodontal data, microbiological data, and systemic inflammatory data and placed in comparative, correlation, and multivariate analysis forms.

2.2 Eligibility Criteria

Participants who had full periodontal assessment, OM measurements and systemic inflammatory biomarker data were eligible to participate. Records that were eligible had

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adequate information that could be classified into healthy, gingivitis, or periodontitis. Data on periodontal measurements that were not completed, data on microbiomes that were not provided, the absence of systemic inflammatory biomarker data, and redundant data were eliminated. The eligibility criteria were used consistently across the participating centers to achieve a complete analytical dataset that could be used to examine the interrelationships between periodontal inflammatory burden, OM dysbiosis and systemic inflammatory activity.

2.3 Assessment of Periodontal Inflammation

Probing pocket depth (PPD) and clinical attachment loss (CAL), bleeding on probing (BOP) and plaque index recorded in clinical records were used to assess periodontal inflammatory status. PPD and CAL were measured in millimeters and BOP and plaque index in percentages. Periodontally healthy participants were identified, as well as those with gingivitis and those with periodontitis based on their reported periodontal health. Periodontal inflammatory and destructive burden was characterized by these clinical parameters and assessed its association with changes in the OM and systemic IB.

2.4 Assessment of OM Dysbiosis

The microbiome dysbiosis index and relative abundance of Porphyromonas gingivalis per participant were measured to evaluate the OM characteristics. Microbiome dysbiosis index was a measure of the general level of microbial imbalance, and P. gingivalis abundance was measured as a percentage. Both microbiological parameters were compared among periodontal groups and assessed with respect to periodontal clinical parameters. Their correlations with systemic IB were also analyzed to find out whether there was an increase in the systemic inflammatory activity with an increase in oral microbial dysbiosis across the included participants.

2.5 Assessment of Systemic Inflammatory Biomarkers

The inflammatory status was determined systemically based on the C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) concentrations, which were in the records of the participants. The concentration of CRP was presented in mg/L, and the concentration of IL-6 and TNF- a were presented in pg/mL. The levels of biomarker were compared between healthy, gingivitis, and periodontitis. Their associations to periodontal and microbiome parameters were then assessed to ascertain whether more periodontal inflammatory load and OM dysbiosis were interrelated with more systemic inflammatory action in the study group.

2.6 Statistical Analysis

The data collection and analysis were done using MS Excel. Continuous variables were reported in the form of mean + SD, whereas the categorical variables were reported as frequencies and percentages. Parametric or non-parametric tests were applied to determine the differences between the periodontal groups with pair-to-pair comparisons as needed. Associations between periodontal, microbiome, and systemic variables of inflammation were analyzed using correlation analysis. Independent associations with CRP, IL-6, and TNF- after age and sex adjustment were assessed using regression.

3. RESULTS

3.1 Distribution of Participants According to Periodontal Status

The sample size of the study was 80 adults, including 20 (25.0) periodontally healthy participants, 25 (31.2) participants with gingivitis and 35 (43.8) participants with periodontitis. The overall mean age was 45.65 ± 11.49 years, with 44 females (55.0%) and 36 males (45.0%). There were no significant differences in age among periodontal groups (p = 0.071) and sex distribution was also similar (2 = 1.039, p = 0.595), which means that there were no variations in the demographics among the three groups of study participants (Table 1).

Table 1. Demographic and periodontal-status distribution of participants

Characteristic	Healthy (n=20)	Gingivitis (n=25)	Periodontitis (n=35)	Total (n=80)	p-value
Participants, n (%)	20 (25.0)	25 (31.2)	35 (43.8)	80 (100)	-
Age (years)	41.55 ± 9.20	44.64 ± 10.86	48.71 ± 12.48	45.65 ± 11.49	0.071
Female, n	12	15	17	44	0.595
Male, n	8	10	18	36	0.595

3.2 Periodontal Inflammatory and Microbiological Characteristics

Periodontal and microbiological parameters were more advanced with the severity of the disease. There is an increase of PPD (2.07 +- 0.34 mm to 5.14 +- 0.59 mm) and CAL, BOP and plaque index in periodontitis. The microbiome dysbiosis index increased from 0.83 ± 0.28 to 3.17 ± 0.48, and P. gingivalis abundance from 1.33 ± 0.69% to 11.19 ± 3.72%. Periodontal and microbiological parameters were significantly different between groups (p < 0.001) with more inflammatory and microbial burden as periodontal severity increased (Table 2).

Table 2. Periodontal inflammatory and OM characteristics according to periodontal status

Parameter	Healthy (n=20)	Gingivitis (n=25)	Periodontitis (n=35)	p-value
PPD (mm)	2.07 ± 0.34	2.98 ± 0.40	5.14 ± 0.59	<0.001
CAL (mm)	0.43 ± 0.29	0.84 ± 0.41	4.26 ± 1.07	<0.001
BOP (%)	7.47 ± 3.71	26.72 ± 7.33	52.61 ± 11.88	<0.001

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Figure 1 depicts that periodontal and OM features vary in the healthy, gingivitis, and periodontitis groups.

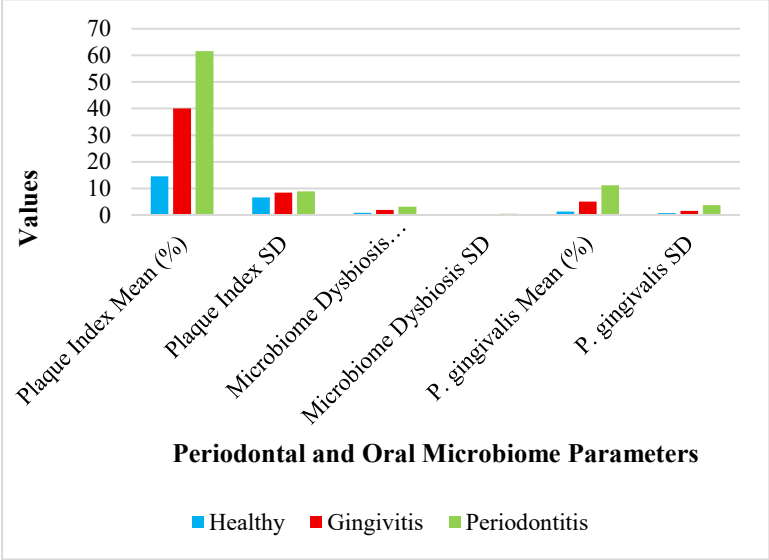

Figure 1. Periodontal and OM parameters according to periodontal status

Figure 1 illustrates that there is a gradual rise in the accumulation of the plaque, microbiome dysbiosis, and the abundance of P. gingivalis as periodontal health, gingivitis, and periodontitis. The subjects with periodontitis always showed the highest changes in the parameters measured as compared to the healthy subjects which were the least. This trend shows that the severity of periodontal diseases is also associated with the increased amount of the plaque and the severe disturbances of the oral microbial environment, which confirms the strong relationship between periodontal inflammation and microbiome dysbiosis among the participants of the study.

3.3 Systemic IB According to Periodontal Status

Periodontal disease severity was associated with progressive increments of systemic IB. CRP rose from 0.80 ± 0.47 mg/L in healthy participants to 5.56 ± 1.29 mg/L in periodontitis, while IL-6 increased from 1.37 ± 0.49 to 7.06 ± 1.83 pg/mL and TNF-α from 4.46 ± 0.94 to 10.52 ± 1.64 pg/mL. There were notable variations in all periodontal samples of each biomarker ($p < 0.001$) with periodontitis having the highest systemic inflammatory load (Table 3).

Table 3. Systemic IB according to periodontal status

Biomarker	Healthy (n=20)	Gingivitis (n=25)	Periodontitis (n=35)	Statistic	p-value
CRP (mg/L)	0.80 ± 0.47	2.63 ± 0.79	5.56 ± 1.29	H = 65.871	<0.001
IL-6 (pg/mL)	1.37 ± 0.49	3.20 ± 1.00	7.06 ± 1.83	H = 64.191	<0.001
TNF-α (pg/mL)	4.46 ± 0.94	6.27 ± 1.35	10.52 ± 1.64	H = 61.348	<0.001

3.4 Association Between Periodontal Inflammation and OM Dysbiosis

Measures of the OM were strongly positively correlated with periodontal inflammatory parameters. PPD was correlated with dysbiosis index (0.856), and P. gingivalis abundance (0.803) and CAL had correlations of 0.817 and 0.809, respectively. BOP correlated with dysbiosis ($\rho = 0.836$) and P. gingivalis ($\rho = 0.760$), while plaque index showed correlations of $\rho = 0.853$ and $\rho = 0.819$. All the relationships were significant at $p < 0.001$.

Figure 2 shows the associations between periodontal clinical parameters and the dysbiosis index of the microbiome and P. gingivalis abundance.

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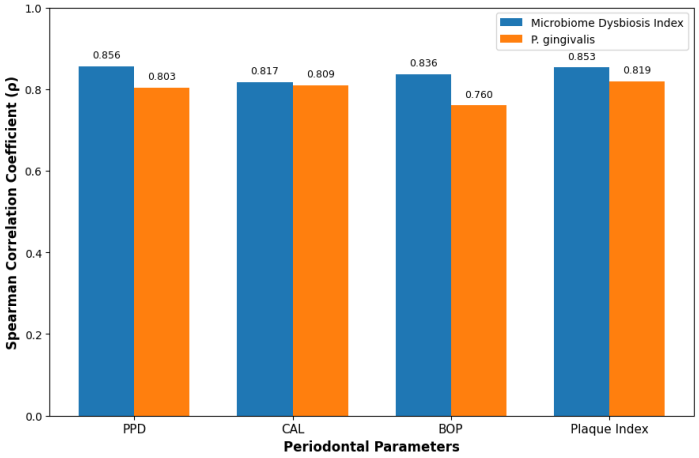

Figure 2. Association between periodontal parameters and OM dysbiosis

All periodontal parameters were strongly positively correlated with both measures of microbiome. The microbiome dysbiosis index significantly correlated with PPD, CAL, BOP and plaque index, with P. gingivalis abundance also correlating similarly as shown in Figure 2. In general, the results suggest that an increase in periodontal inflammatory and clinical burden was strongly linked with an increase in the OM dysbiosis and increase in the relative abundance of the periodontal pathogen P. gingivalis.

3.5 Independent Associations with Systemic IB

When age and sex were factored in, BOP and microbiome dysbiosis were independent predictors of CRP, IL-6, and TNF-alpha (all $p < 0.001$). These biomarkers had 75.9, 82.1 and 75.9 percent variance explained by the respective models with the highest explanatory capacity being IL-6. These results imply that independent relations of periodontal inflammatory burden and microbiome dysbiosis with demographically adjusted systemic inflammatory status were observed (Table 4)

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Table 4. Multivariable associations of periodontal inflammation and microbiome dysbiosis with systemic IB

Outcome	Predictor	Adjusted β	95% CI	p-value	R^2
CRP	BOP (%)	0.045	0.023–0.068	<0.001	0.759
CRP	Dysbiosis Index	0.952	0.499–1.406	<0.001	0.759
IL-6	BOP (%)	0.077	0.053–0.101	<0.001	0.821
IL-6	Dysbiosis Index	0.975	0.484–1.465	<0.001	0.821
TNF-α	BOP (%)	0.066	0.036–0.096	<0.001	0.759
TNF-α	Dysbiosis Index	1.243	0.637–1.848	<0.001	0.759

3.6 Association Between OM Dysbiosis and Systemic IB

Systemic IB were strongly positively correlated with OM dysbiosis. The microbiome dysbiosis index correlated with CRP ($\rho = 0.839$), IL-6 ($\rho = 0.861$), and TNF-α ($\rho = 0.820$). P. gingivalis abundance was similarly associated with CRP ($\rho = 0.797$), IL-6 ($\rho = 0.766$), and TNF-α ($\rho = 0.713$). Each of the correlations was statistically significant ($p < 0.001$), which proved that an increased OM disruption was always followed by an increase in the concentration of circulating markers of inflammation in the subjects of the study.

Figure 3 shows that microbiome dysbiosis and P. gingivalis abundance are associated with systemic IB such as CRP, IL-6 and TNF-alpha.

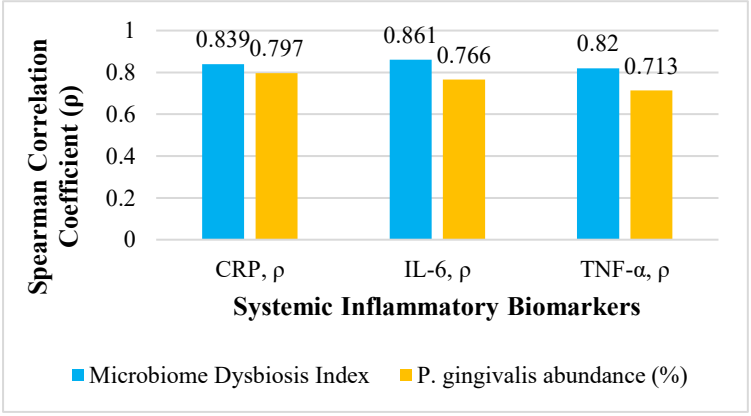

Figure 3. Association of OM dysbiosis with systemic IB

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There were significant positive relationships between changes in the OM and IB in the body. The index of microbiome dysbiosis showed a steady and greater relationship with CRP, IL-6, and TNF-α compared to P. gingivalis abundance as shown in Figure 3. Microbiome dysbiosis was found to correlate with IL-6 the most. On the whole, the results indicate that an increase in oral microbial dysbiosis correlated with an increase in systemic inflammatory activity, which showed that periodontal microbial imbalance is related to SI.

4. DISCUSSION

The current research showed that periodontal inflammation, OM dysbiosis, and inflammatory activities in the body were significantly linked. Participants who had periodontitis had the highest PPD, CAL, BOP, plaque index, dysbiosis index, and P. gingivalis abundance. There was also increasing progression of CRP, IL-6, and TNF-a with periodontal severity. The positive associations between periodontal parameters and microbiome measures showed that the rise in clinical disease burden was associated with an increase in microbial disturbance which supported the idea of interrelationship between periodontal inflammation, dysbiosis, and systemic inflammatory condition.

Such results were in line with prior evidence supporting the association between OM changes and the severity of periodontitis and SI (Plachokova et al., 2021). The diagnostic value of microbiome biomarkers in periodontitis was also supported by the progressive microbial dysbiosis of periodontal groups (Radu et al., 2025). The interactions between periodontal inflammation and microbial changes were also in line with the reported relationships between dysbiosis, inflammatory cytokines, and altered host responses (Pirih et al., 2021) indicating that microbial imbalance and inflammatory activity are tightly related factors of periodontal disease. The results obtained regarding the relationship between oral dysbiosis and systemic IB could also be compared to findings on the association between changes in oral microbial communities and immune-inflammatory reactions (Soffritti et al., 2021). Dysbiosis of oral microbiota has also been identified as being involved in systemic inflammatory pathways involved in inflammatory bowel disease (Qi et al., 2022). In addition, the possible applicability of oral microbial profiles to systemic diseases has been highlighted earlier (Thomas et al., 2021), and dysbiosis has been shown to be a potential non-invasive microbial biomarker in the context of systemic diseases (Zhang et al., 2020).

Adjusted age and sex-adjusted BOP and microbiome dysbiosis were independently correlated with CRP, IL-6, and TNF-alpha. The models accounted for a large amount of variation in systemic IB with the best explanatory capacity being noted with IL-6. These results suggest that clinical periodontal inflammation and microbial dysbiosis were complementary to each other when it came to systemic inflammatory status. However, the relationships that were observed should not be construed as cause and effect since the retrospective study design was capable of establishing relationships but it could not tell the temporal orientation between periodontal, microbial and systemic inflammatory changes.

The results indicate that inflammation in the periodontal area and microbiome dysbiosis could be of interest in assessing periodontal disease. Their regular interactions with systemic IB imply that periodontal diseases could be relevant beyond local oral inflammation. BOP and the microbiome dysbiosis index can be some of the informative indicators of inflammatory burden. Combining clinical periodontal and microbiological measures may thus enhance the characterization of periodontal to systemic inflammatory connections, but additional validation is needed before these can be suggested to be used in systemic inflammatory risk evaluation in standard clinical practice. A number of limitations must be noted. This retrospective design did not allow establishing temporal connections and causality. The study sample (n=80) might not be representative of larger and more diverse populations. Even though, the multivariate analysis has controlled the age and sex, possible confounding factors like smoking, metabolic disorders, medications, diets and lifestyle factors were not included. Moreover, P. gingivalis alone was not an adequate measure of the complexity of the OM, which is an interaction of various interacting microbial communities related to periodontal health and disease.

Future research ought to employ larger prospective multicenter cohorts to elucidate temporal and causal connections amid periodontal inflammatory state, microbiome dysbiosis, and SI. Microbiome profiling in depth must be integrated to describe changes in microbes at community levels other than single pathogens. Other behavioral, metabolic, and clinical confounders are to be taken into account as well. Such longitudinal and interventional studies into whether periodontal therapy and restoration of microbial balance can decrease CRP, IL-6 and TNF- a would be of great interest in establishing whether the periodontal-systemic inflammatory relationships observed are of clinical significance and whether they can be modified.

5. CONCLUSION

The current multicenter retrospective study revealed that there was a distinct relation between periodontal inflammation, OM dysbiosis, and systemic inflammatory activity in adults. The severity of periodontal disease increased with increased probing pocket depth, clinical attachment loss, bleeding on probing, and accumulation of plaque, along with gradual increases in microbiome dysbiosis and P. gingivalis abundance. The highest levels of CRP, IL-6, and TNF-alpha were also observed among participants with periodontitis suggesting that there is a consistent relationship between the severity of periodontal disease and systemic inflammatory load. Positive associations between periodontal parameters and microbiome measures were also strong and enough evidence that the higher periodontal inflammation was, the closer microbial disruption. Likewise, systemic IB were positively correlated with microbiome dysbiosis and abundance of P. gingivalis. Notably, after controlling age and sex, bleeding on probing and microbiome dysbiosis were still significantly related to CRP, IL-6, and TNF-

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alpha, indicating their possible applicability to systemic inflammatory condition. On the whole, these results indicate that periodontal inflammation, oral microbial imbalance, and SI have an interrelated association. Causal inference is, however, not possible with the retrospective design. More prospective and longitudinal studies with complete microbiome profiling and clinical confounders should be conducted to better understand these associations and whether periodontal therapy and recovery of microbial homeostasis can help in decreases in systemic inflammatory load.

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