# Microbiological Profile in Periodontitis and Peri-Implantitis: A Systematic Review

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**ABSTRACT:** Understanding the microbiological makeup of peri-implant biofilm could contribute to the discovery of focused treatment strategies, improving the outcome of peri-implantitis management. However, the bacterial profile in diseased periodontal and peri-implant sulci is still unclear. This systematic review aims to analyze the microbiological similarities and differences between diseased periodontal and peri-implant sulci based on the available literature evidence. A thorough search was conducted in electronic databases such as PubMed, Google Scholar, and Cochrane, as well as a manual search employing the eligibility criteria. After a thorough review, studies evaluating the microbial composition acquired from plaque samples obtained from patients with diseased periodontal and peri-implant sulci were chosen. The selected 8 studies evaluated the differences in microbial profile in periodontitis and peri-implantitis. Five studies found a statistically significant variation in the microbial profile between diseased periodontal and peri-implant sulci, while in one study, no changes in the microbiology of inflammatory peri-implant and periodontal sites were observed. In one of the two *in situ* studies, the structure of the transcription level and core species was different in peri-implantitis, whereas the other *in situ* study found that the 16S rRNA-based bacterial profile of both the diseases were different, while the functional genes, taxonomic, and virulence factor mRNA profiles were identical. According to existing studies, significant differences in the biofilm composition of diseased periodontal and peri-implant sulci were observed. Therefore, periodontitis and peri-implantitis have diverse microbial characteristics.

**KEY WORDS:** microflora, microbial profile, periodontitis, periodontal disease, dental implants, peri-implantitis, systematic review

## I. INTRODUCTION

For years, dental implants have grown in popularity and are increasingly being used to replace lost teeth. Even while dental implants have a high success rate, they do fail occasionally. Mechanical and biological issues are the common causes of failure of implant. Biomechanical overloading, inappropriate position/angulation of implant, insufficient bone support or poor bone quality, and the presence of excessive load are the most common causes of mechanical failures.[1–3] Biological failures are caused by bacterial plaque, which causes peri-implantitis by disrupting the balance between the host and the bacteria. Although mechanical complications are likely to be avoided, biological issues are more difficult to avoid.[4,5] As a result, extensive investigation into the microbiota involved in this disease process is required.

Polymicrobial diseases such as peri-implantitis and periodontitis have comparable clinical signs and symptoms. The majority of patients with periodontitis react effectively to treatment and seem to have stable periodontal tissue for a longer period. Clinical therapies for peri-implantitis, including periodontitis treatments, are frequently futile.[6–10] Furthermore, in animal models, peri-implantitis is shown to progress more quickly than periodontitis.[11,12] To further understand the disparities between both the diseases, researchers have analyzed their unique microbiota.

The bacterial profile in diseased peri-implant and diseased periodontal sites have been studied.[13] Peri-implantitis, according to Kumar et al. is a bacterial diverse condition dominated by anaerobic organisms.[14] Furthermore, Dabdoub et al. found that 60% of study population shared less than half of the microbial species between both the disease biofilms,

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implying that the microbial flora belong to separate ecosystems.[15] Several studies, on the other hand, have found a similar microbiological profile in same patients between failed implants and the neighboring teeth.[16–18]

Furthermore, history of periodontitis has found to be a risk indicator for peri-implant diseases. Among individuals who had periodontitis-related tooth loss, a comprehensive review reported a considerably high occurence of peri-implant diseases.[19] Similarly, patients with periodontitis had a high failure rate of implants and crestal bone loss than those with periodontal health. Bacteria that promote periodontal breakdown have been hypothesised to migrate and populate peri-implant sites.[20,21] Researchers have demonstrated that several of the bacterial species may be found in the mouth even after teeth have been lost completely,[22–25] and bacteria can even be found in apparently healed alveolar bone.[26] As a result, oral soft tissues, as well as teeth, may serve as major reservoirs of microorganisms that can proliferate around dental implants. Literature has also highlighted that the presence of periodontopathogens might affect the peri-implant tissues, similar to what has been observed in periodontal tissues.[27–30]

However, the microorganisms that initiate disease namely, peri-implantitis and periodontitis remain debatable based on the available literature evidence. Several investigations have demonstrated the preponderance of microorganisms common in the two diseases[31,32] as well as others that are distinct to peri-implantitis sites.[33–35] All of these investigations used culture, DNA hybridization, PCR, and DNA sequencing to determine the microbial profile. Previous research based on DNA sequencing may have found dead microorganisms, which could explain the contradictory results. As a result, metatranscriptomic *in situ* analysis has recently been performed to investigate the discrepancies in mRNA profiles and core taxa for periodontitis and peri-implantitis etiologies.

Two recent systematic reviews[36,37] on the microbiological composition of peri-implant disease featured in literature search. The microbiological composition of peri-implant disease were compared with implants in healthy condition and with periodontal disease in these studies. Two studies evaluating metatranscriptomic data on the complete microbiota of peri-implantitis[38,39] and comparing it to periodontitis were recently published, although were not considered in earlier evaluations.

This systematic review aims to analyze the microbiological similarities and differences between diseased periodontal and peri-implant sulci based on the available literature evidence.

## II. MATERIALS AND METHODS

A thorough search was conducted in five electronic databases, including PubMed, Google Scholar, Cochrane, EMBASE and Web of Science, as well as manual search, with article selection based on Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) standards. The items relevant to peri-implantitis, periodontitis, and microflora were searched using MeSH (Medical Subject Headings) terminologies, and the search items were concatenated using the Boolean operators (OR, AND). The studies that examined the microbiological profile between periodontitis and per-implantitis were selected for inclusion after searching electronic resources till December 2021. For the literature search, there were no language restrictions.

### A. Inclusion Criteria

Inclusion criteria were: cross-sectional, longitudinal, or clinical trial studies that compared the microbial profile obtained from plaque samples obtained from patients with diseased periodontal and peri-implant sulci.

### B. Exclusion Criteria

Exclusion criteria were: (1) narrative or systematic reviews, meta-analyses, editorials, case reports, case series or animal studies; (2) studies assessing only viruses; and (3) studies without statistical analysis of the microbial findings.

## III. ARTICLE SELECTION

### A. Search Results

Two independent reviewers (AR and SV) used MeSH terms to search PubMed, Google Scholar, Cochrane, EMBASE and Web of Science until December 2021. A manual search was also carried out using the list of references from the selected manuscripts. Two researchers evaluated the title and abstract of the entries found through the initial electronic database searches independently and any disagreements were resolved through discussion. After reviewing the abstracts, the researchers examined the studies that met the inclusion and exclusion criteria and made a final selection of articles. After that, data extraction was carried out.

### B. Studies Included

The study's flow chart is depicted in Fig. 1. There were 626 articles found in the electronic search. Animal studies were the reason for the exclusion of 112 publications. After reviewing the titles and abstracts,

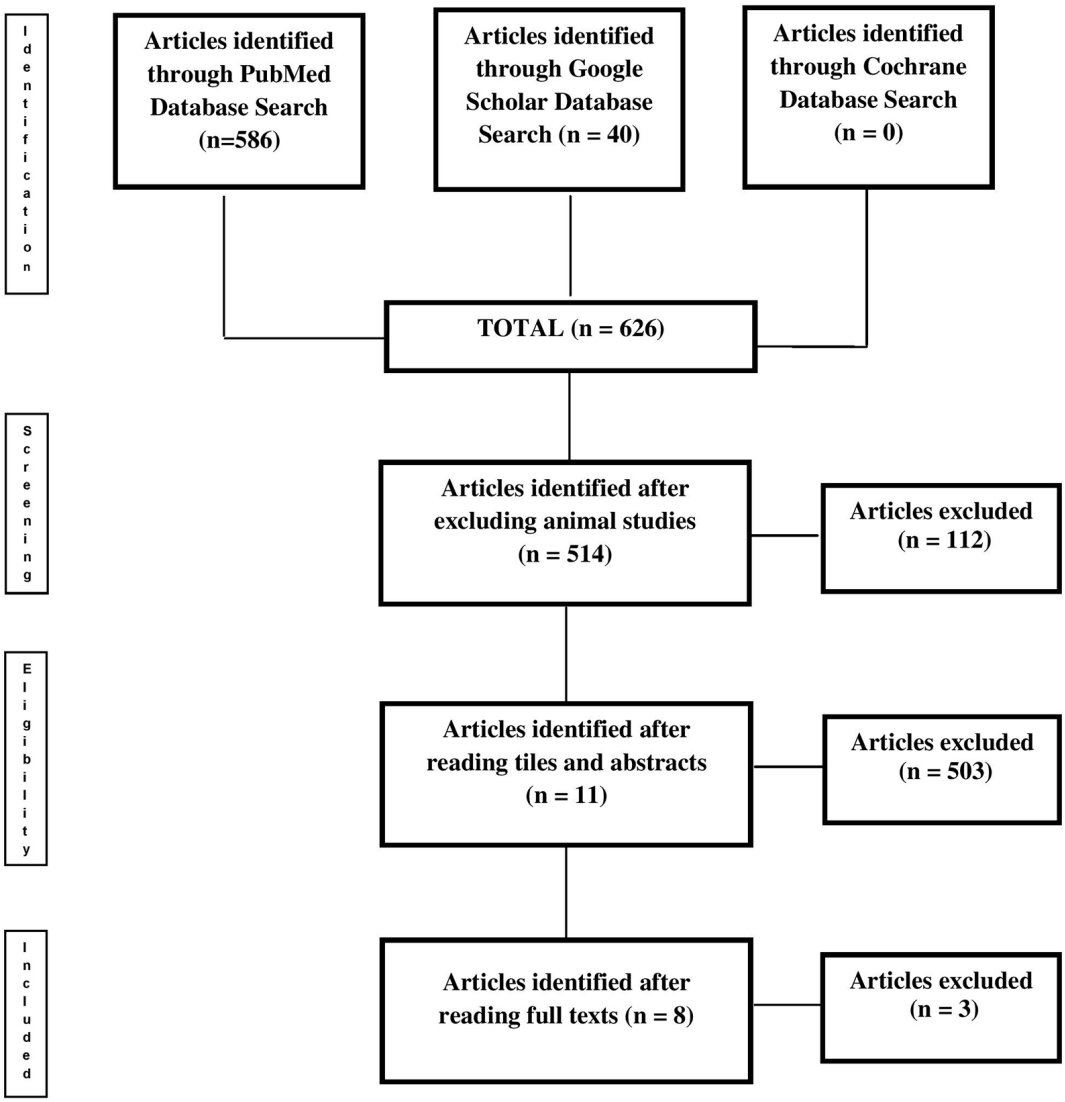


**FIG. 1:** Study flowchart

503 items were eliminated, leaving 11 for full-text reading. No additional articles were chosen during the manual search. After reading the complete text, three papers were eliminated because they did not include statistical analysis comparing the microbiological profile of periodontitis and peri-implantitis. As a result, the current systematic review contained 8 articles.

## IV. DATA EXTRACTION

The data of the selected studies was extracted and summarized as follows: (1) reference, (2) study design, (3) sample, (4) microbiological analysis, (5) data expression and statistical analysis, (6) microorganisms in periodontitis, (7) microorganisms in peri-implantitis, and (8) conclusion (Table 1).

## V. RESULTS

The selected eight studies compared the microbial profile around diseased periodontal and peri-implantitis sulci. Six of the investigations were cross-sectional, while the other two were *in situ*. In terms of microbial profile analysis, one study used culture, three studies used PCR, two studies used pyrosequencing and two studies used metatranscriptomic analysis. Three studies looked at the prevalence of specific periodontal pathogens, while one looked at the overall number of bacterial taxa, two looked at the core microbiota at the genus level, and the other two looked at metatranscriptomic analyses *in situ*.

Regarding the microbiological data, out of six cross-sectional studies, five studies found a statistically significant variation in the microbial profile between periodontal disease and peri-implant disease, while one of the study reported no changes in the microbiology of inflammatory peri-implant and periodontal sites. In one of the two *in situ* studies, the structure of the transcription level and core species was different in peri-implant disease, whereas the other *in situ* study found that the 16S rRNA-based bacterial profile of both the diseases were different, while the functional genes, taxonomic, and virulence factor mRNA profiles were identical.

Table 2 shows quality of the included studies, according to the STROBE statement. All articles selected defined title and abstract, background, objectives, study design, setting, criteria for participant selection, variables, data sources, statistical methods, outcome data, main results, key results and interpretation. Six studies mentioned about the quantitative variables and descriptive data. Also, six studies discussed about the generalizability of the study results and analysed confounder adjustment and subgroup analysis. Limitations of the study and funding details was mentioned in five studies. None of the selected studies mentioned about the methods used to avoid bias and sample size calculation. None of the articles considered in this study satisfied all of the STROBE criteria. Three articles complied with 19 items; another two with 18 items, one article with 17 items, and two articles with 14 items. Thus, all articles included in this review satisfied 75% of the items assessed.

## VI. DISCUSSION

The prevalence of implant failure owing to peri-implantitis is increasing in parallel with the increased use of dental implants. Oral biofilm is the key causative factor for peri-implant tissue inflammation. Because bacterial biofilm is the cause of both periodontitis and peri-implantitis, the treatment for both is the same.[40,41] However, the success of peri-implantitis therapy is not commendable. Gaining a better understanding of the nature of peri-implant biofilm could contribute to the discovery of focused treatment strategies, improving the outcome of peri-implantitis management. However, the bacterial profile in diseased periodontal and peri-implant sulci is still unclear.

Three studies looked at the prevalence of certain periodontal pathogens. *B. forsythus* and *P. gingivalis* were more frequent in periodontitis sites, while spirochetes, *P. micros*, and fusobacterium were more frequent around peri-implant diseased sulci, according to Listgarten et al.[42] Another study[43] assessed the presence of *P. gingivalis*, *A. actinomycetemcomitans*, *T. forsythia*, *P. intermedia*, *C. rectus*, *T. denticola*, and around inflamed periodontal and peri-implant sulci. The levels of *A. actinomycetemcomitans* and *P. gingivalis* were in comparison between diseased periodontal sulci and diseased peri-implant sulci.

**TABLE 1:** Characteristics and summary of included studies

| Sl. No | Ref. | Study design | Sample (*n*) | Microbiological analysis | Data expression and statistical analysis | Microorganisms in periodontitis | Microorganisms in peri-implantitis | Conclusion |
|---|---|---|---|---|---|---|---|---|
| 1 | Listgarten et al.[42] (1999) | Cross-sectional | 41 periodontitis patients 41 peri-implantitis patients | Culture | Comparison of detection frequency of *B. forsythus*, *Spirochetes*, *P. micros*, *P. gingivalis*, *C. rectus*, *P. intermedia*, *Fusobacterium* sp., *A. actinomycetemcomitans*, *E. corrodens* Chi-square test | Frequently detected microorganisms were: *B. forsythus* 83%, *Spirochetes* 79%, *Fusobacterium* 80%, *P. micros* 51%, *P. gingivalis* 59%, *E. corrodens* 37% | Frequently detected microorganisms were: *B. forsythus* 59%, *Spirochetes* 54%, *Fusobacterium* 41%, *P. micros* 39%, *P. gingivalis* 27% | Some periodontal bacteria were detected at much higher frequencies in peri-implantitis than in periodontitis ($P < 0.05$) |
| 2 | Cortelli et al.[43] (2012) | Cross-sectional | 50 periodontitis patients 50 peri-implantitis patients | PCR | Comparison of prevalence of *T. denticola*, *P. gingivalis*, *T. forsythia*, *C. rectus*, *P. intermedia*, *F. nucleatum*, *A. actinomycetemcomitans* Chi-square test | Higher frequency of all six species | Higher frequency of *P. gingivalis* and *A. actinomycetemcomitans* | There were significant differences between periodontitis and peri-implantitis in the six species studied ($P < 0.05$) |
| 3 | Koyanagi et al.[47] (2013) | Cross-sectional | 6 patients with both periodontitis and peri-implantitis | PCR | Comparison of number of bacterial taxa Mann-Whitney *U* test | Total number of bacterial taxa: 148 | Total number of bacterial taxa: 192 | Per-implantitis sites had a higher prevalence than periodontitis sites ($P < 0.05$) |
| 4 | Maruyama et al.[44] (2014) | Cross-sectional | 20 patients with both periodontitis and peri-implantitis | Pyrosequencing | Comparison of relative abundances of each bacterial taxon Wilcoxon signed-rank test | The proportion of *Peptostreptococcaceae* sp. and *Desulfomicrobium orale* was significantly higher in periodontitis than in peri-implantitis | The proportions of *Prevotella nigrescens* were significantly higher in peri-implantitis than in periodontitis | Both peri-implantitis and periodontitis are polymicrobial diseases that are caused by different microorganisms ($P < 0.05$) |

**TABLE 1:** (*continued*)

| Sl. No | Ref. | Study design | Sample (*n*) | Microbiological analysis | Data expression and statistical analysis | Microorganisms in periodontitis | Microorganisms in peri-implantitis | Conclusion |
|---|---|---|---|---|---|---|---|---|
| 5 | Schaumann et al.[46] (2014) | Cross-sectional | 7 patients with both periodontitis and peri-implantitis | Pyrosequencing | Comparison of taxa on a genus level Wilcoxon signed-rank test | Most abundant genera: *Prevotella*, *Streptococcaceae*, TG5 | Most abundant genera: *Rothia*, *Streptococcaceae*, *Porphyromonas* | In the same subject, there were no significant microbiological changes on a genus level between diseased peri-implant and periodontal sites ($P > 0.01$) |
| 6 | Zhuang et al.[45] (2014) | Cross-sectional | 22 patients with both periodontitis and peri-implantitis | PCR | Comparison of prevalence of *T. denticola*, *P. gingivalis*, *P. intermedia*, *F. nucleatum*, *A. actinomycetemcomitans*, *S. aureus* between tooth-implant pairs per individual Student's *t*-test | High levels of *P. gingivalis*, *F. nucleatum*, *A. actinomycetemcomitans* | High levels of *A. actinomycetemcomitans* | On the basis of periodontal or peri-implant disease status, there are differences in the involvement of some of the pathogens for both the diseased conditions ($P < 0.05$) |

**TABLE 1:** (*continued*)

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 7 | Shiba et al.[38] (2016) | *In situ* | 12 patients with both periodontitis and peri-implantitis | Metatranscriptomic analysis | Comparison of taxonomic profile, functional potential profile, virulence factor profile, co-occurrence networks, and interacting core taxa Wilcoxon signed-rank test | Operational taxonomic units: 62.3 ± 20.3; microbial taxa identified: 164 | Operational taxonomic units: 58.5 ± 21.8; microbial taxa identified: 150 | In peri-implantitis, microbial networks were more complex than in periodontitis ($P < 0.05$) |
| 8 | Komatsu et al.[39] (2020) | *In situ* | 21 patients with both periodontitis and peri-implantitis | Metatranscriptomic analysis | Comparison of taxonomic profile, functional potential profile, virulence factor profile, co-occurrence networks, and interacting core taxa Wilcoxon signed-rank test | Operational taxonomic units: 319.1 ± 86.6 | Operational taxonomic units: 357.9 ± 110 | A number of core species and high-expression genes in the co-occurrence networks were found to be specific to peri-implantitis ($P < 0.05$) |

**TABLE 2:** Quality assessment of the included studies according to STROBE statement

| Question | Ref. | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | **Listgarten et al.[42] (1999)** | **Cortelli et al.[43] (2012)** | **Koyanagi et al.[47] (2013)** | **Maruyama et al.[44] (2014)** | **Schaumann et al.[46] (2014)** | **Zhuang et al.[45] (2014)** | **Shiba et al.[38] (2016)** | **Komatsu et al.[39] (2020)** |
| Title and abstract | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Background | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Objectives | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Study design | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Setting | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Participants | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Variables | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Data sources | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Bias | No | No | No | No | No | No | No | No |
| Study size | No | No | No | No | No | No | No | No |
| Quantitative variables | No | Yes | Yes | Yes | No | Yes | Yes | Yes |
| Statistical methods | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Descriptive data | Yes | Yes | Yes | Yes | No | No | Yes | Yes |
| Outcome data | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Main results | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Other analyses | No | Yes | Yes | Yes | No | Yes | Yes | Yes |
| Key results | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Limitations | No | No | No | Yes | Yes | Yes | Yes | Yes |
| Interpretation | Yes | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Generalizability | No | Yes | Yes | Yes | No | Yes | Yes | Yes |
| Funding | No | Yes | No | Yes | No | Yes | Yes | Yes |

However, other analyzed species were more common in periodontal disease rather than peri-implant disease. Similarly, Maruyama et al.[44] found that the proportion of *Prevotella nigrescens* in peri-implantitis was significantly greater as compared with periodontal disease, but the detection frequency of *Desulfomicrobium orale* and *Peptostreptococcaceae* sp. were more in inflamed periodontal sulci than in peri-implant sulci. These studies indicated that there exists a bacterial variation in both the diseases. Similarly, Zhuang et al.[45] highlighted that there was difference in the involvement of some of the pathogens for periodontitis and peri-implantitis. In contrary, Schaumann et al.[46] reported that there was no significant microbiological differences on a genus level between both the diseases in the same subject.

A total of 333 unique taxa were discovered when the prevalence of bacteria in periodontitis and peri-implantitis was investigated utilising a real-time polymerase chain reaction. There were 192 and 148 taxa found at the peri-implant disease and periodontal disease sites, respectively. When compared with periodontitis, the bacterial composition of peri-implant disease was diversified. Bacteria such as *Streptococcus* spp. and *Fusobacterium* spp. were found in both the diseases, however, *Parvimonas micra* was found in only in diseased peri-implant site. When compared with periodontitis, the biofilm in peri-implant disease had a diverse bacterial population.[47]

Recently, systematic reviews on the microbial profile of peri-implant disease have underlined the relevance of using metagenomic and metatranscriptomic techniques to analyze the microbial profile. This systematic review looked at two recent metatranscriptomic studies on the bacterial profile of peri-implantitis. By performing a metatranscriptomic analysis at diseased peri-implant site and diseased periodontal site in the same individuals, Shiba et al.[38] examined the bacterial species associated with each disease *in situ*. The microbial compositions of the two groups differed based on 16S rRNA sequences. Furthermore, bacterial distribution at the genus level differed across samples from each subject. The number of functional genes in the two diseases, however, did not differ significantly when mRNA profiles were compared. Furthermore, no variations in mRNA abundance of any virulence genes were found between both the diseases, according to the research. Moreover, the peri-implantitis microbiome had more complex microbial networks than the periodontal disease microbiome. In peri-implant disease, the red complex species *P. gingivalis*, *T. denticola*, and *T. forsythia* were associated with each other whereas limited association was noted among *P. gingivalis* and *T. denticola* in periodontal disease.

Similarly, Komatsu et al.[39] used metatranscriptomic network analysis to assess the gene transcription activity in peri-implant disease and periodontal disease. The metagenomic-based microbial co-occurrence network had a wider range of species and relationships than the metatranscriptomic-based network. In the co-occurrence network, *Prevotella denticola* and *Solobacterium moorei* displayed higher level of activity and were specific to peri-implant disease. Furthermore, the gene activity of plasmin receptor/glyceraldehyde-3-phosphate dehydrogenase was greater in peri-implant disease. These changes in activity may add to the intricacy of the peri-implantitis microbiota and help distinguish between the two diseases' clinical manifestations.

Overall, the microbiological analysis method and study design varied greatly among the studies that were chosen. While each methodology has its own set of benefits and drawbacks, the outcomes of various microbiological processes did not allow to compare the outcome parameters. In the last few decades, there are more advances in genetic analysis which enabled more extensive bacterial analysis, whereas culture-dependent approaches were previously used. Other modern assays, such as pyrosequencing, also enabled for the examination of a broad spectrum of bacteria. Furthermore, because different microbiological assays focus on different targets such as total bacterial load quantification, identification of core microbiota or assessment of specific periodontopathogens, comparing data obtained from various studies using different microbial assays is difficult.

Both periodontitis and peri-implantitis are inflammatory disorders that last for a long time. Despite the fact that bacterial plaque is the primary cause of both diseases, a number of aggravating

or risk factors exist, including systemic disorders, smoking, stress, hereditary, hormonal influence, malnutrition, gender and age.[48–57] These variables have been found to have a considerable impact on periodontal or peri-implant disease progression. The results and microbiological profile may have been influenced by the lack of control of these variables in the included trials. Also, six out of eight studies[38,39,44–47] assessed the microbial findings in the same patients with both peri-implant disease and periodontal disease and the remaining two studies[42,43] evaluated the bacterial profile in inflamed periodontal and peri-implant sites among different patients. This form of heterogeneity may impact the microbial profile due to the influence of patient-related factors.

In recent years, our knowledge about bacteria in the development of periodontitis and peri-implantitis has shifted dramatically. The character of the disease is determined by the interaction between the host, bacteria and the environment, rather than by the specific group of microorganisms, as emphasized by the concept of polymicrobial synergy and dysbiosis.[58] Among the studies included in this systematic review, three studies[42,43,45] assessed the proportion of specific pathogens rather than assessing the entire microbiome. In view of the new idea of disease pathophysiology, this could be a limitation.

In the formation of dental plaque, the topography and chemical composition of implant surfaces are crucial factors. Surface energy, roughness, material stability, crystallographic characteristics and surface chemistry, in particular, are factors that enhance biofilm attachment.[59] However, none of the investigations specified the type of implant utilized or the impact of implant material on the microbial profile.

Within the limitations, the study highlights that peri-implant disease represents a diverse bacterial profile as compared with periodontal disease. Heterogeneity in terms of diagnostic criteria for periodontitis and peri-implantitis, microbial analysis, study design (within patients or different patients) and confounders did not allow comparison of data, thus meta-analyses may be questionable due to potential bias. Another problem is the sample size estimation, which may understate results because to the low frequency of peri-implant disease, which necessitates bigger sample size to get substantial differences. Despite the fact that metatranscriptomic analysis may evaluate a wider range of bacteria and has higher sensitivity, both of the investigations in this review looked at the microbiome in periodontitis and peri-implantitis samples from the same patients. As a result, the conclusions of the study did not accurately reflect the population.

## VII. CONCLUSION

According to existing studies, significant differences in the biofilm composition of diseased periodontal and peri-implant sulci were observed. Therefore, periodontitis and peri-implantitis have diverse microbial characteristics. These findings should aid in the diagnosis and development of targeted therapeutic approaches for peri-implantitis, improving the prognosis of peri-implantitis management.

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