



Review article

## COMPARATIVE ANALYSIS OF MICROORGANISMS AROUND TEETH AND IMPLANTS IN HEALTH AND DISEASE

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### ABSTRACT

**Aim:** Dental implants are a reliable solution for replacing missing teeth and restoring oral health. Despite their high survival rates, complications such as peri-implant mucositis and peri-implantitis remain common challenges. This underscores the importance of assessing implant success not only in terms of stability but also by preserving healthy peri-implant tissues. The review consolidates recent findings on the peri-implant microbiome and examines its relationship with the dental microbiome under both healthy and diseased conditions.

**Methods:** For the present review a search was conducted on PubMed, Google Scholar, and ScienceDirect.

**Results:** Modern microbial identification techniques have uncovered new species previously unknown or not considered in the context of periodontal infection. It is now understood that the peri-implant biofilm, both in health and disease, is not as similar to that on teeth as previously thought. The implant biofilm is directly influenced by the implant material, the implant-abutment connection, the implant surface, as well as the biofilm present in the patient's mouth.

**Conclusion:** The submucosal microbiota of peri-implantitis lesions has not been extensively studied using culture-independent techniques and our understanding of the microbial profile associated with peri-implantitis remains incomplete.

**Keywords:** peri-implantitis, peri-implant biofilm, peri-implant microbiome,

### BACKGROUND

Dental implants are an effective method for replacing missing teeth and restoring oral health. This approach has shown a high level of predictability, with a survival rate of 90% to 95% over five years [1]. Despite this success, many patients develop peri-implant mucositis and peri-implantitis. Recent studies indicate that the prevalence of peri-implantitis is approximately 20% at the patient level and 11.5% at the implant level [2]. Therefore, the definition of a successful implant goes beyond its mere presence in the mouth. In addition to stability, success requires the maintenance of healthy peri-implant tissues despite the constant microbial challenges of the oral environment [2, 3].

Although peri-implantitis is a complex disease associated with multiple risk factors, it is generally accepted that its initiation results from the interaction between microorganisms and an excessive host response [4, 5]. Dental implants are placed in an oral environment containing both commensal microorganisms and potentially pathogenic microorganisms (pathobionts). Significant research has focused on studying the peri-implant biofilm, its relationship with the periodontal biofilm, and the differentiation of bacterial clusters associated with health and disease. However, there is no definitive consensus on a specific bacterial complex that initiates peri-implant bone loss [6, 7]. In periodontitis, key pathogens present in small amounts can disrupt the periodontal microbiota and lead to dysbiosis [8]. Identifying such key microorganisms in the peri-implant microbial community remains elusive.

### METHODS

For the present review a search was conducted on PubMed, Google Scholar, and ScienceDirect. Only articles written in English were included. The reference lists of the selected articles were subjected to a hand search to identify additional articles.

### RESULTS AND DISCUSSION

The oral cavity hosts over 700 species of microorganisms, 40% of which have never been cultured. Advances in microbiological technologies, bioinformatics, and the wider application of 16S rRNA gene sequencing have improved our understanding of these microorganisms [9]. Results indicate that the periodontal and peri-implant

microbiomes are less similar than previously thought, likely because implants create unique niches within the oral cavity [10, 11, 12]. Dental implants differ from natural teeth in several key architectural aspects, including morphology, surface material, roughness, and energy. Most implants in use today are engineered with moderately rough surfaces, and additional surface modifications have been incorporated into their design to enhance osseointegration [13]. These variations in the structure, surface features, and chemical composition of used materials significantly influence bacterial adhesion and colonization, thereby impacting biofilm formation. As a result, the microbiological profile of peri-implantitis is more heterogeneous and complex, predominantly composed of non-cultivable Gram-negative species, compared to periodontitis [14].

### **Biofilm formation**

Implants have both supra- and submucosal parts exposed to the oral environment. Researchers have drawn conflicting conclusions about the effect of the surface on initial biofilm formation, but it is generally argued that the composition and proportions of the initial colonizers are primarily influenced by the patient's periodontal status [15].

When an implant is placed in the oral cavity, it introduces a new solid surface with distinct physical properties conducive to microbial attachment. These microorganisms might already be present in the mouth or appear subsequently. Salivary proteins and peptides adhere to the implant surface, creating a pellicle. This pellicle acts as a receptor for the adhesins of specific oral bacteria species, which are the early colonizers of the implant surface. These early colonizers, including *Streptococcus*, *Actinomyces*, and *Veillonella*, are similar to those that colonize teeth. Unlike a cleaned tooth surface, which retains remnants of attached microbiota, the implant surface is initially free of endogenous microflora and requires initial colonization by these early bacteria to start forming a complex microcommunity [16, 17].

Implant colonization occurs rapidly, with biofilm formation detectable as early as 30 minutes after placement. A study by Fürst et al. investigated the early colonization of titanium implants - from immediately after placement through the first three months following surgery - and compared the developing microbiota with that of adjacent tooth sites in periodontally healthy patients. The study revealed that among the bacterial species examined, only *Veillonella parvula* showed a significant increase in load between 30 minutes and 1 week post-surgery. By week 12, although the composition of bacterial species remained unchanged, the overall bacterial load was higher on natural teeth than on implants. Additionally, key periodontal pathogens - such as *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola* - were more frequently detected at implant sites by week 12. These findings suggest that while the same bacterial species rapidly colonize both teeth and implants, the microbial load differs between these surfaces. [18].

The sequence of events in biofilm formation is similar to that observed on natural teeth. The first colonizers are representatives of the yellow complex

(*Streptococci*) and *Actinomyces*. Their multiplication and co-aggregation result in a dense mass of microorganisms attached either directly to the implant surface or to each other. A second wave of early colonizers follows, adhering to the coaggregates attached to the implant. These are representatives of the green and violet complexes, which also form coaggregates with each other. Representatives of the orange complex form a more loosely attached mass and are dispersed between the implant-associated and epithelium-associated biofilm, which mostly consists of bacteria from the red complex. Species that participate in multiple coaggregates, such as fusobacteria, serve as bridges between early and late colonizers [19, 20].

Currently, there is considerable discussion on how diverse surfaces affect the bacterial species profile associated with them, as well as the mechanisms involved in the initial bacterial colonization of implants and the potential onset of diseases. A study by Herrmann et al. examined typical material and roughness combinations, which represent the composition of an implant/abutment unit, all of which are commonly exposed to the oral cavity. The ceramic abutment material was shown to harbour significantly higher total bacterial cell counts than the titanium abutment. This holds true for both early and mature biofilms. Furthermore, the rough/active implant surface exhibited a significant increase in the total bacterial cell count between early and mature biofilms. Microarray-based DNA hybridization demonstrated the presence of putative periodontal pathogens on both materials, but colonization patterns did not differ significantly among the different surfaces [21].

In another study by Siddique et al., researchers utilized a mixed culture model comprising early, secondary, and late colonizers, which emulate the stages of human oral bacterial biofilm formation. This study analyzed bacterial attachment on two types of dental implant materials, commercially pure titanium (cpTi) and 3% mol yttria-stabilized zirconia (ZrO<sub>2</sub>), with clinically relevant surface treatments (polished, acid-etching, or sandblasting). The findings unveiled a unique composition differing from the planktonic form but remaining consistent across all surfaces. Therefore, within the constraints of this particular model, the dynamics of multi-species bacterial adhesion on cpTi and ZrO<sub>2</sub> are deemed comparable [22].

### **Biofilm associated with peri-implant health and peri-implant disease**

Agerbaek et al. compared samples of 40 bacterial species using DNA-DNA hybridization in periodontally healthy patients with implants. They examined the species found on teeth and implants, including probing depth as a variable. They found an identical microbial profile, but the percentage of sites positive for *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola* was lower in implants with probing depths greater than 5 mm compared to teeth with the same probing depth. The difference in results is explained by the fact that the greater probing depth around implants, in the absence of bone loss and bleeding, may be due to factors such as the diameter of the implant, crown contour, and depth of inser-

tion rather than being related to peri-implant disease [23].

Using conventional DNA probe and cultural analyses, other researchers have identified common periodontopathogenic bacteria at both healthy and diseased implant sites, with no significant variation in species distribution based on the clinical status of the implant [4,24,25]. Persson and Revent used a DNA-DNA checkerboard to analyze the presence of 78 bacterial species in the biofilm of 166 implants with peri-implantitis and 47 healthy implants. They found a cluster of bacteria associated with peri-implantitis sites, including *P. gingivalis*, *Staphylococcus aureus*, *S. anaerobicus*, *S. intermedius*, *S. mitis*, *T. forsythia*, and *T. socranskii*. (26) Additionally, observational studies suggest that peri-implantitis is more frequently associated with opportunistic pathogens such as *Pseudomonas aeruginosa* and *Staphylococcus aureus* (*S. aureus*), as well as fungal organisms (e.g., *Candida albicans*, *Candida boidinii*, *Penicillium spp.*, *Rhodotorula laryngis*, *Paecilomyces spp.*) and viruses (e.g., *human cytomegalovirus*, *Epstein-Barr virus*) [27–31]. This indicates that peri-implantitis involves a complex and heterogeneous infection, and further studies need to be conducted to define the microbiological profile of peri-implant tissues [11, 32, 33].

New studies highlight the genus *Prevotella*, which is frequently associated with peri-implantitis [34]. In a study by Song et al., the genus *Prevotella* did not show a significantly higher abundance in peri-implantitis compared to healthy implant sites. However, one unclassified species, *Prevotella* sp. HMSC077E09, along with *P. endodontalis*, was notably more abundant in peri-implantitis sites with established periodontitis. This was in comparison to healthy implant sites within the periodontitis cohort and peri-implantitis sites in the unaffected periodontal environment group. This finding provides novel insights into the unique role these species may play in the disease process of peri-implantitis in the presence of periodontitis [35]. The same research group also found that red complex species and *P. endodontalis* were consistently associated with peri-implantitis, irrespective of periodontal status, as evidenced by their positive correlation with disease-related clinical parameters. In contrast, within the periodontitis-affected cohort, *A. oris*, *S. sanguinis*, *P. propionicum*, and *S. odontolytica* were linked to successful implant outcomes, aligning with their negative correlation with diseased clinical parameters. Moreover, among subjects without periodontitis, *A. naeslundii* was associated with clinically healthy implants, indicated by its taxonomic abundance and its negative correlation with adverse clinical features. Functional pathways related to epithelial cell invasion, such as flagellar assembly, were enriched at peri-implantitis sites. This enrichment was connected to species from the *Treponema*, *Selenomonas*, and *Campylobacter* genera, which, although not dominant in terms of taxonomy, may serve as key triggers in the development of peri-implant disease [35].

In a systematic review and meta-analysis conducted by Sahrman et al., it was emphasized once again that although both periodontitis and peri-implantitis are associated with a polymicrobial subgingival community and its

dysbiosis, recent examinations of the diseases' microbiomes have shown a generally higher prevalence of *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia*, and *Tannerella forsythia* in peri-implantitis biofilms compared to either periodontitis biofilms or healthy implants. Additionally, *Tannerella spp.*, *Parvimonas spp.*, *Fusobacterium spp.*, and *Campylobacter spp.* are frequently detected genera in peri-implant lesions, indicating again that peri-implantitis is driven by a highly complex community through polymicrobial synergy and dysbiosis rather than individual causative pathogens [36].

#### **Biofilm in edentulous patients**

The question of whether partial or total tooth loss affects the subgingival microflora at implants is often raised. Edentulous patients do not have teeth to serve as reservoirs for periodontal pathogens. However, they are not at a lower risk of developing peri-implantitis [37]. Accumulating data indicate that the microorganisms colonizing clinically healthy implants in edentulous patients are similar to those associated with healthy periodontal sites, with a higher frequency of Gram-positive facultative cocci and a lower frequency of Gram-negative anaerobic rods.

The understanding of the microflora of diseased implants in totally edentulous individuals has evolved over recent decades. Early studies did not detect *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis*, attributing this absence to the subgingival environment being the sole habitat for these periodontopathogens [38]. However, both types have been isolated in cases of peri-implantitis developing five or more years after loading in totally edentulous individuals. It is suggested that the soft tissues of these patients harbour periodontal pathogens, likely serving as a source for colonization after implant placement [39]. Research in this area also dispels the myth that bacterial load is automatically neutralized with tooth extraction.

In partially edentulous patients, the remaining dentition is the primary source of microorganisms, but the potential role of soft tissue surfaces and saliva as reservoirs of infection should not be overlooked [21]. It can also be inferred that a greater number of periodontal pathogens will colonize the implants of patients with a history of periodontal infection.

Collectively, the cited above data demonstrates that modern microbial identification techniques have unveiled previously unknown species, underscoring that the peri-implant biofilm - whether in health or disease - is distinctly different from that on natural teeth. The peri-implant sulcus constitutes a unique niche, where factors such as the implant material, implant-abutment connection, implant surface, and the existing oral microbiome all exert a direct influence on the developing biofilm. In partially edentulous individuals, the peri-implant biofilm varies from that of natural teeth in both bacterial composition and load. Moreover, in totally edentulous patients, the biofilm that forms on implants contains species not detected in the mouth prior to implant placement, which may include novel microorganisms or those typically associated with periodontal pockets.

## CONCLUSION

The submucosal microbiota of peri-implantitis lesions has not been extensively studied using culture-independent techniques. Consequently, our understanding of the microbial profile associated with peri-implantitis remains incomplete.

## Data availability statement

The data supporting this narrative review are derived from publicly available articles. These articles can be accessed through online academic databases such as PubMed, Google Scholar, and ScienceDirect. Specific details, including journal names, can be found in the references section of this review.

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