ISSN 0973-2063 (online) 0973-8894 (print)

Bioinformation 22(7): 4234-4239 (2026)

BIOINFORMATION
Discovery at the interface of physical and biological sciences

BIOMEDICAL INFORMATICS

www.bioinformation.net

Volume 22(7)

Review

Received July 1, 2026; Revised July 31, 2026; Accepted July 31, 2026, Published July 31, 2026

DOI: 10.6026/973206300222434

SJIF 2026 (Scientific Journal Impact Factor for 2026) = 8.478
2022 Impact Factor (2023 Clarivate Inc. release) is 1.9

Edited by P Kangueane

Citation: Gupta *et al.* Bioinformation 22(7): 4234-4239 (2026)

Peri-implant dysbiosis: Current perspectives and microbial dynamics

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ISSN 0973-2063 (online) 0973-8894 (print)
Bioinformation 22(7): 4234-4239 (2026)

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Abstract:
Dental implants are a predictable treatment option for tooth replacement, but peri-implant diseases remain a major challenge to their long-term success. Emerging evidence indicates that these diseases result from peri-implant microbial dysbiosis and disruption of host–microbial homeostasis rather than biofilm accumulation alone. Disease progression results in shift to pathogenic communities, including keystone pathogens and implant-adapted opportunistic species that promote leads to peri-implant bone loss. This review summarizes the current evidence on the composition, dynamics and pathogenic mechanisms of the peri-implant microbiota, with emphasis on implant-specific microbial interactions. Thus, data shows that understanding these microbial changes may facilitate early diagnosis and develop targeted preventive and therapeutic strategies for peri-implant diseases.

Keywords: Dental implants, peri-implantitis, microbial dysbiosis, microbial spectrum

Background:
Dental implants are widely recognised as a predictable and effective treatment modality for replacing missing teeth, with consistently high long-term success rates. Nevertheless, biological complications such as peri-implantitis continue to compromise implant outcomes, with reported failure rates ranging from 5–10% despite advances in implant design and surface characteristics **[1, 2]**. Peri-implantitis is characterised by inflammation of the surrounding hard and soft tissues and is primarily associated with microbial challenge **[1, 3]**. However, the conventional view of peri-implant disease as a purely infectious process is increasingly being reconsidered. Following implant placement, microbial colonisation occurs rapidly, with bacterial adhesion beginning within 30 minutes and early colonisers, predominantly streptococci, establishing themselves within hours **[5, 6]**. These initial microbial events play a critical role in subsequent biofilm development and tissue–implant interactions **[7]**. The peri-implant microflora comprises a complex and dynamic microbial community organised within structured biofilms embedded in an extracellular polymeric matrix, contributing to their persistence and resistance **[4, 8]**. Biofilm formation occurs through ecological succession, beginning with pioneer species such as *Streptococcus* and *Actinomyces*, followed by bridging organisms such as *Fusobacterium nucleatum* and ultimately progressing to colonisation by anaerobic Gram-negative pathogens including *Porphyromonas gingivalis* and *Treponema denticola* **[3, 22]**. Early microbiological studies demonstrated distinct differences between healthy and diseased implant sites. Successful implants were predominantly associated with coccoid forms, whereas failing implants exhibited increased proportions of spirochetes and motile rods **[12, 13]**. Subsequent investigations identified the presence of key periodontopathic microorganisms, including *Peptostreptococcus micros*, *Fusobacterium* spp., *Prevotella intermedia* and *Aggregatibacter actinomycetemcomitans*, in peri-implantitis lesions, thereby reinforcing the infectious nature of the disease and its similarities to periodontitis **[14, 15]**. However, more recent evidence suggests those peri-implant microbiotas are not merely identical to those associated with periodontal disease but instead represent a distinct and more diverse microbial ecosystem. The long-term success of dental implants is closely associated with effective control of microbial biofilm within the peri-implant environment **[3, 10]**. Implant surface characteristics, particularly surface roughness and chemical composition, significantly influence bacterial adhesion as well as the composition of supra- and subgingival microbiota **[11, 12]**. Unlike natural teeth, implants lack a periodontal ligament and possess distinct structural and biological characteristics, rendering peri-implant tissues more susceptible to inflammatory breakdown **[26]**. In addition, opportunistic pathogens such as *Staphylococcus aureus* demonstrate a strong affinity for titanium surfaces, further distinguishing peri-implant infections from classical periodontal disease. Importantly, although microbial biofilm accumulation is a necessary etiological factor, its presence alone is insufficient to initiate disease **[27]**. Current evidence supports the concept that peri-implantitis develops as a consequence of dysbiosis, characterised by a shift in the microbial community toward a pathogenic composition together with disruption of host–microbial homeostasis **[28]**. Advanced high-throughput sequencing and microbial network analyses have further demonstrated significant differences in submucosal microbial communities among healthy peri-implant tissues, peri-implant mucositis and peri-implantitis **[29–31]**. Therefore, it is of interest to evaluate the composition and dynamics of peri-implant microflora, the mechanisms underlying dysbiosis and the transition from health to disease.

Peri-implant biofilm dynamics and dysbiosis:
Biofilm formation within the peri-implant environment is a highly organised and dynamic process that plays a central role in the initiation and progression of peri-implant diseases. Following implant exposure to the oral cavity, a conditioning film derived from salivary proteins and glycoproteins rapidly adsorbs onto the implant surface, thereby facilitating bacterial adhesion and coaggregation **[1]**. Early colonisation is predominantly mediated by Gram-positive facultative organisms, particularly *Streptococcus* species such as *S. viridans*, *S. mitis* and *S. oralis*, which establish the initial biofilm architecture. Subsequently, interbacterial adhesion and coaggregation mechanisms promote the recruitment of secondary colonisers, including *Actinomyces* species and other bridging organisms, leading to the development of a complex

multilayered biofilm embedded within an extracellular polymeric matrix composed of polysaccharides such as levans and dextrans **[2]**. This structured biofilm provides mechanical stability and enhances microbial survival by increasing resistance to environmental challenges and host defence mechanisms. The high microbial load in saliva, estimated at approximately 10^7 bacteria per millilitre, further contributes to rapid biofilm maturation **[3]**. A critical aspect of peri-implant biofilm development is ecological succession, whereby early colonisers modify the local microenvironment to favour the establishment of anaerobic Gram-negative pathogens. This transition reflects a shift from a symbiotic microbial community to a dysbiotic state characterised by enrichment of keystone pathogens such as *Porphyromonas gingivalis*, which can modulate host immune responses and disrupt microbial homeostasis. These pathogens act synergistically with other microbial species to enhance virulence and promote tissue destruction. Unlike natural tooth surfaces, implant materials particularly titanium possess distinct physicochemical properties that significantly influence microbial adhesion and biofilm composition. Titanium implants are coated by a thin oxide layer (approximately 2–5 nm) with amphoteric properties, enabling interactions with both positively and negatively charged molecules **[21, 24]**. The adsorption of salivary and crevicular fluid-derived proteins onto this oxide layer through covalent, ionic and hydrogen bonding creates a favourable substrate for bacterial attachment **[21]**. Furthermore, surface roughness and surface energy play decisive roles in enhancing microbial retention and biofilm maturation, thereby increasing susceptibility to peri-implant inflammation **[24, 25]**. In addition, implant-specific microbial patterns have been identified, with opportunistic pathogens such as *Staphylococcus aureus* demonstrating a strong affinity for titanium surfaces and the implant–abutment interface, thereby contributing to persistent colonisation and potential infection **[26]**. These findings indicate that peri-implant biofilms are not merely analogous to dental plaque but rather constitute a distinct microbial ecosystem shaped by material properties and local environmental conditions. Collectively, the interactions among microbial communities implant surface characteristics and host immune responses drive the transition from biofilm formation to dysbiosis, ultimately playing a pivotal role in the pathogenesis of peri-implant diseases.

Transition from health to disease:

Peri-implant tissues in health are characterised by a stable and symbiotic microbial community predominantly composed of Gram-positive facultative cocci and rods, which contribute to biofilm homeostasis and maintenance of tissue integrity **(Table 1) [7]**. These microbial communities resemble those present around periodontally healthy teeth and are associated with minimal inflammatory response. However, this equilibrium is highly susceptible to disruption by local and systemic factors, resulting in a shift toward a dysbiotic microbial profile. One of the distinctive features of peri-implant microbiology is the influence of implant material on microbial colonisation. In contrast to natural teeth, titanium implant surfaces exhibit a selective affinity for opportunistic pathogens such as *Staphylococcus aureus*, which is not commonly associated with the microbiota of natural teeth. This organism shows a strong ability to adhere to titanium surfaces through surface adhesins known as microbial surface components recognising adhesive matrix molecules (MSCRAMMs), which interact with host proteins including fibrinogen, fibronectin, collagen, vitronectin and laminin **[8, 10]**. Following implant placement, rapid coating of the implant surface with host-derived extracellular matrix components establishes a competitive environment, often described as a "race for the surface," between host cells and microbial colonisers. Successful adhesion and persistence of *S. aureus* have been associated with clinical manifestations such as bleeding on probing and suppuration **[9]**, highlighting its potential role in the initiation of peri-implant inflammation (**Table 2**). As dysbiosis progresses, a well-recognised transition occurs in the microbial composition, shifting from predominantly Gram-positive facultative organisms to Gram-negative anaerobic species. This shift signifies progression from peri-implant health to peri-implant mucositis and ultimately peri-implantitis. The microbial profile associated with peri-implantitis demonstrates a greater prevalence of pathogenic complexes described by Socransky, particularly the red complex species including *Porphyromonas gingivalis*, *Tannerella forsythia* and *Treponema denticola*, together with orange complex organisms such as *Fusobacterium nucleatum* and *Prevotella intermedia* (**Table 3**) **[11]**. These microorganisms exhibit enhanced virulence through synergistic interactions, thereby increasing inflammatory burden and promoting tissue destruction. Importantly, this transition is not solely attributable to microbial accumulation but also reflects disruption of host–microbial homeostasis. Keystone pathogens such as *P. gingivalis* play a crucial role by modulating host immune responses and promoting a dysbiotic environment that favours proliferation of pathogenic species. The resulting inflammatory cascade contributes to connective tissue breakdown and progressive peri-implant bone loss. The differences in microbial composition between health and disease are further illustrated in **Figure 1**, which demonstrates a distinct shift from a commensal-dominated microbiota in healthy peri-implant tissues to a pathogen-rich microbial community in peri-implantitis, as described by Al-Ahmad *et al.* (2018) **[28]**. This microbial transition highlights the dynamic nature of peri-implant biofilms and reinforces dysbiosis as a central mechanism in the pathogenesis of peri-implant disease.

Table 1: Health-associated bacteria

Bacteria	Role
Actinomyces naeslundii	Early colonizer; maintains biofilm stability and promotes oral health.
Corynebacterium spp.	Common in healthy peri-implant sites; contributes to microbiome balance.
Rothia spp.	Found in health and mucositis; may help buffer transition to disease.

Veillonella parvula	Present in early biofilms; supports biofilm maturation with Streptococci.
Neisseria spp.	Abundant in healthy sites; indicates balanced microbial environment.

Table 2: Mucositis-associated (early disease) bacteria

Bacteria	Role
Capnocytophaga spp.	Found in low abundance; involved in early dysbiosis.
Campylobacter rectus	Pathogenic; participates in early inflammation.
Parvimonas micra	Anaerobe contributing to early tissue damage.
Peptostreptococcus spp.	Minor presence in health; increased in mucositis.
Prevotella nigrescens	Initiates inflammation and immune response.
Neisseria spp.	Transitional role from health to mucositis.

Table 3: Peri-implantitis-associated bacteria

Bacteria	Role
Fusobacterium nucleatum	Keystone coaggregator; supports pathogenic biofilm maturation.
Porphyromonas gingivalis	Major pathogen; suppresses host immunity, causes bone loss.
Tannerella forsythia	Member of red complex; promotes inflammation and epithelial destruction.
Treponema denticola	Invasive spirochete; produces cytotoxins and degrades tissue.
Prevotella intermedia	Pro-inflammatory; contributes to tissue destruction.
Enterobacteriaceae (e.g., E. coli)	Opportunistic pathogens; increase biofilm complexity and resistance.

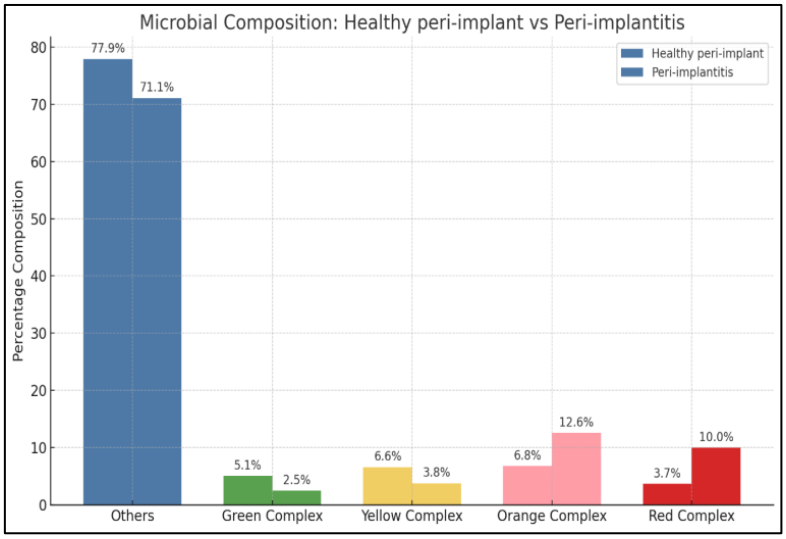

Figure 1: Healthy peri-implant versus peri-implantitis

Microbiologic evidence:

Microbiologic evidence supporting biofilm-associated peri-implant infections is derived from early human studies analysing plaque samples from diseased implant sites. Implants with increased probing depths demonstrated a reduced proportion of coccoid forms and a significantly higher prevalence of spirochetes, indicating a shift toward a more anaerobic and pathogenic microbial profile **[12, 13]**. This transition in microbial morphology provides evidence of the association between biofilm composition and peri-implant disease progression **[14]**. Although biofilm formation around dental implants follows a pattern broadly comparable to that observed on natural teeth **[16]**, important differences exist in the initial colonisation dynamics. On natural tooth surfaces, biofilm development begins rapidly due to the presence of residual indigenous microbiota, with colonisation occurring within 24–26 hours **[17]**. In contrast, implant surfaces are initially devoid of a resident microbial community, making early colonisers critical in conditioning the surface and facilitating subsequent microbial succession **[18]**. Following exposure to the oral environment, implant surfaces are rapidly coated by an acquired pellicle, with formation occurring as early as 30 minutes **[19]**. The composition of this pellicle differs from that on enamel due to variations in protein adsorption, including reduced albumin binding, which may influence early bacterial adhesion patterns **[21]**. Initial colonisation is dominated by Gram-positive cocci and rods, particularly *Streptococcus* and *Actinomyces* species, which establish the primary biofilm structure **[20]**. Subsequent colonisation involves incorporation of anaerobic Gram-negative pathogens such as *Porphyromonas gingivalis* and *Prevotella intermedia* through coaggregation mechanisms, leading to increased biofilm complexity and pathogenicity **[9, 11]**. This microbial succession closely parallels the transition observed in periodontitis and reinforces the role of biofilm maturation in disease development **[27, 28]**. Collectively, these findings support the concept that peri-implantitis is not merely associated

with bacterial presence but is driven by qualitative changes in biofilm composition characterised by increased microbial diversity, pathogenicity and host immune modulation **[29–31]**.

Prevention of biofilm formation:

Biofilm accumulation around dental implants represents a primary etiological factor in the development of peri-implant diseases; however, disease initiation is more accurately attributed to dysbiosis rather than mere plaque presence **[27, 28]**. Consequently, preventive strategies must focus not only on mechanical disruption of biofilm but also on maintaining host–microbial equilibrium **[29]**. Regular maintenance and systematic monitoring remain fundamental for long-term implant success **[7]**. Clinical and radiographic assessments, including evaluation of plaque accumulation, bleeding on probing, probing depth and peri-implant bone levels, are essential for early detection of disease progression **[7, 10]**. Structured protocols such as Cumulative Interceptive Supportive Therapy (CIST) and the Peri-Implant Mucositis Index (PIMI) provide a standardised framework for risk assessment and timely intervention **[9]**. Despite the widespread use of titanium implants, their intrinsic antibacterial properties are limited when compared with noble metals such as gold **[21]**. This limitation has encouraged the development of advanced surface modification strategies aimed at reducing bacterial adhesion and biofilm maturation **[23]**. These include antibiotic coatings, incorporation of antimicrobial peptides and alterations in surface nanotopography, all of which have showed potential in modulating early microbial colonisation and preventing establishment of pathogenic biofilms **[24, 25]**. Importantly, preventive approaches should also consider the dynamic interactions between implant surfaces, microbial communities and host immune responses **[27, 28]**. Strategies targeting early colonisation, disrupting biofilm architecture and modulating host inflammatory pathways are likely to be more effective in preventing the transition from peri-implant health to disease **[29–31]**. Adjunctive oral hygiene measures play a supportive role in controlling peri-implant biofilm and preventing the transition toward dysbiosis **[7, 9]**. The use of antimicrobial mouth rinses, particularly chlorhexidine, may aid in reducing microbial load and plaque accumulation; however, prolonged use should be approached cautiously because of potential adverse effects, including alterations in titanium surface characteristics and disruption of oral microbiome balance **[21, 23]**. Professional maintenance is equally critical in preserving peri-implant health **[7]**. The use of non-metallic or plastic-coated instruments during supportive periodontal therapy is recommended to minimise surface damage, as surface alterations can enhance bacterial adhesion and promote biofilm retention **[24, 25]**. Regular professional debridement, combined with patient education emphasising meticulous plaque control and adherence to maintenance protocols, is essential for long-term stability of peri-implant tissues **[9]**. Importantly, both patient-driven and professional interventions should be regarded as components of a broader strategy aimed at maintaining microbial homeostasis rather than merely reducing plaque accumulation **[27, 28]**. Sustained disruption of pathogenic biofilms, together with preservation of implant surface integrity, remains central to preventing peri-implant disease progression **[29–31]**.

Future perspectives:

Despite significant advances in implant therapy, biofilm formation on implant surfaces remains a persistent and unresolved biological challenge **[22, 27]**. Future strategies should focus on shifting from conventional plaque-control approaches toward targeted modulation of the peri-implant microbiome and host response **[28, 29]**. Emerging technologies such as antimicrobial photodynamic therapy (aPDT), bioactive surface coatings, antimicrobial peptides and nanotopographic modifications show promise in preventing bacterial adhesion and disrupting established biofilms while maintaining biocompatibility **[23–25]**. In addition, microbiome-based approaches, including probiotics and host-modulation therapies, may provide novel opportunities for restoring microbial balance and preventing dysbiosis. Future research should prioritise well-designed longitudinal clinical and microbiological studies to establish standardised preventive and therapeutic protocols **[29–31]**. Greater emphasis is also required on identifying specific microbial signatures and host-response markers that may enable early diagnosis and risk stratification of peri-implant disease **[30, 31]**. A deeper understanding of the complex interactions among implant surfaces, microbial communities and host immunity will be essential for the development of more predictable and biologically driven treatment strategies **[27–31]**.

Conclusion:

Peri-implant diseases results from microbial dysbiosis driven by complex interactions between biofilms, implant surfaces and the host immune response. Advances in understanding peri-implant microbiota and implant-specific microbial interactions may improve earlier diagnosis and more targeted prevention and treatment can be provided. Thus, future research should focus on personalized therapeutic strategies and long-term maintenance to improve implant health and longevity.

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