ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

Research Progress on the Correlation between Oral Microecological Imbalance and Microscrew Implant Anchorage Failure

Shiting Bi[1,a], Qiaoyu Xu[1,b], Yidan Ma[1,c], Jianguo Liu[1,3,d], Xiaoyan Guan[2,e,*], Suhan Jin[1,2,3,f,*]

[1]School of Stomatology, Zunyi Medical University, Zunyi, China
[2]Department of Orthodontics, Affiliated Stomatological Hospital of Zunyi Medical University, Zunyi, China
[3]Special Key Laboratory of Oral Diseases Research, Higher Education Institution, Zunyi, China
[a]1466550733@qq.com, [b]462254338@qq.com, [c]m651876643@163.com, [d]3596056899@qq.com, [e]2304428043@qq.com, [f]1974007965@qq.com
**Corresponding author*

Abstract: *The interaction between microscrew implants and the oral microbiota has emerged as a research focus in orthodontics in recent years. The application of microscrew implant anchorage may disrupt the ecological balance of normal oral microorganisms. Coupled with the susceptibility to plaque accumulation around the microscrew implant, this can induce peri-implant inflammation and ultimately lead to orthodontic treatment failure. A lack of in-depth understanding of microscrew implant anchorage from the perspective of oral microecology may increase the risk of anchorage loss. This article reviews the intrinsic relationship between microscrew implant anchorage failure and oral microecology, aiming to elucidate the potential mechanisms underlying their interaction and to provide a theoretical reference for reducing the incidence of peri-implantitis and associated failure risks.*

Keywords: *Microscrew implant anchorage, Oral microecology, Orthodontics, Peri-implantitis, Dysbiosis*

1. Introduction

In recent years, with the continuous improvement in living standards, people's aesthetic demands have increasingly risen. Orthodontic treatment, which involves both dental aesthetics and function, has seen a growing demand[1]. Currently, the use of implant anchorage is considered the most common orthodontic treatment modality in clinical practice and is a necessary choice for complex cases, offering both minimally invasive characteristics and cost-effectiveness[2]. The oral cavity is a unique microecosystem that harbors diverse microbial communities, and microbial imbalance is generally recognized as a driving factor for oral diseases[3]. Furthermore, during orthodontic treatment, the presence of orthodontic appliances impedes patients from performing routine oral hygiene, creating favorable conditions for rapid plaque adhesion and accumulation[4]. For patients with microscrew implants, oral microecological imbalance can trigger peri-implant inflammation. In this process, bacterial products within the peri-implant sulcus and various inflammatory factors collectively participate in alveolar bone destruction, which may ultimately lead to the loss of implant anchorage. Therefore, the relationship between implant anchorage and oral microecology has attracted increasing attention from orthodontists in recent years.

Microscrew implant anchorage is a temporary anchorage device that has been widely applied in clinical orthodontics due to its advantages such as small size, ease of operation, and immediate loading capacity[5]. Unlike conventional dental implants, orthodontic microscrew implants typically do not require osseointegration but rely on mechanical interlocking to provide temporary anchorage[6]. Orthodontic microscrews possess unique characteristics in terms of material, design, and insertion site, which render their interaction with the oral microbiota more complex. Regarding material, titanium alloys or stainless steel are commonly used in clinical practice. Although titanium alloys are considered to have good biocompatibility, their surfaces remain prone to the formation of acquired pellicles and microbial colonization. Studies have shown that rough surfaces or unpolished neck designs increase

ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

initial bacterial adhesion and biofilm maturation[7]. In terms of structural design, the transmucosal neck of the microscrew serves as a critical interface connecting the oral environment and bone tissue. Poor surface polishing or the presence of micro-grooves in this area can become a "hotbed" for plaque accumulation, subsequently inducing peri-implant inflammation[8]. Regarding insertion sites, microscrews are often placed in the attached or free gingiva of the alveolar bone, such as the interradicular alveolar ridge of the posterior maxilla and mandible[9]. These regions have complex anatomical structures and narrow interproximal spaces, making daily oral hygiene challenging. Moreover, the presence of orthodontic appliances such as archwires and springs further impedes local cleaning, leading to food debris and plaque retention around the implant neck, thereby disrupting local microecological balance[10]. These factors not only render microscrew implant anchorage susceptible to oral microecological influences but also allow its insertion and use to reciprocally alter the local microenvironment.

Oral microecology refers to the complex community of microorganisms colonizing the oral cavity, including bacteria, fungi, and viruses, and represents the second most complex microbial community in the human body, second only to the gut[11-13]. Over 700 microbial species have been identified in the human oral cavity. These microorganisms are not randomly distributed but form distinctly different micro-niches across various anatomical sites, including tooth surfaces, gingival sulci, the dorsal tongue, buccal mucosa, hard palate, and soft palate[14]. More importantly, the oral cavity, as the gateway to the digestive tract, can influence systemic health through multiple pathways. Epidemiological and mechanistic studies have confirmed that oral dysbiosis is significantly associated with systemic diseases such as diabetes mellitus, cardiovascular disease, rheumatoid arthritis, adverse pregnancy outcomes, and even Alzheimer's disease[15-18]. The biological basis for this association lies in the fact that oral pathogens and their metabolites can directly disseminate to distant organs through bacteremia or indirectly affect systemic metabolism and immune function by triggering low-grade systemic inflammation. Specifically, oral dysbiosis can activate host immune-inflammatory responses, promote the release of pro-inflammatory cytokines, and subsequently affect distant organ function[19]. For example, periodontal pathogens such as *Porphyromonas gingivalis* have been demonstrated to invade vascular endothelial cells and accelerate the progression of atherosclerosis.

Thus, the relationship between microscrew implant anchorage and oral microecology not only affects the local oral environment and orthodontic treatment outcomes but may also exert potential impacts on overall systemic health through microbial translocation and systemic inflammatory responses. Therefore, gaining an in-depth understanding of the interaction mechanisms between the two holds significant clinical importance.

2. Impact of Microscrew Implants on Oral Microecology

After microscrew implant placement, its material properties, surface characteristics, and insertion site collectively constitute key factors influencing the oral microecology. Implant placement itself creates a new ecological niche within the oral cavity. This artificial structure not only passively accepts microbial colonization but also actively shapes the composition and dynamics of the local microbial community through its physicochemical properties.

2.1. Effects of Material and Surface Characteristics on Microbial Colonization

Regarding material, different implant materials exhibit varying affinities for microorganisms. Studies have shown that titanium alloy surfaces have lower microbial colonization potential compared to stainless steel, while zirconia demonstrates lower colonization capacity for *Prevotella intermedia* and Actinobacillus actinomycetemcomitans than titanium alloy and stainless steel[20]. In an in vivo study comparing three abutment materials—cast titanium, machined titanium, and zirconia—researchers found that cast titanium surfaces exhibited the highest total bacterial counts and species detection rates, whereas zirconia surfaces showed the lowest bacterial colonization[21]. However, some clinical studies have reported that during observation periods of 2 weeks and 3 months, no significant differences were found in the counts of seven representative bacteria including *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, and *Prevotella intermedia* between zirconia and titanium abutments, with both materials demonstrating favorable soft tissue health[22]. This finding suggests that the effect of material itself on microbial colonization may be less significant than that of surface roughness, or that differences may only emerge over longer timeframes.

ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

Regardless of material, surface roughness is a critical factor influencing initial microbial adhesion and plaque maturation. When the surface roughness of an implant exceeds a threshold of Ra=0.2μm, plaque colonization increases significantly[23]. Micro-grooves, cracks, or unpolished areas on the microstructure provide physical protection sites for bacteria, making them more resistant to mechanical cleaning. Further studies have shown that bacterial adhesion exhibits selectivity depending on surface roughness: on smooth surfaces (Ra < 0.3 μm), chitosan coatings significantly reduce bacterial adhesion; whereas on rough surfaces (Ra ≥ 3.0 μm), polylysine coatings demonstrate superior antibacterial effects[24]. Compared to smooth surfaces, rough surfaces such as titanium plasma-sprayed surfaces accumulate significantly more plaque, and conventional tooth brushing is less effective for cleaning them[25].

Notably, the surface characteristics of microscrews not only influence initial bacterial adhesion but also determine the structure and maturation process of the biofilm. The grooves and pores of rough surfaces can serve as refuges for bacteria, protecting them from shear forces and mechanical cleaning. Therefore, in the design of microscrew implants, surface polishing of the transmucosal neck is crucial. Maintaining a highly smooth surface in this region can effectively reduce plaque accumulation and lower the risk of peri-implantitis[23]. In recent years, researchers have attempted to enhance the antimicrobial properties of microscrews through surface modification technologies such as silver/hydroxyapatite nanoparticle coatings and zinc oxide nanoparticle coatings[26]. Animal studies have demonstrated that Ag/HA-coated microscrews exhibit the highest antibacterial activity and bone area fill rate, while sustained-release fisetin-loaded PLGA coatings inhibit inflammatory cell infiltration and bone resorption, thereby improving implant stability[27]. However, a clinical randomized controlled trial showed that chlorhexidine hexametaphosphate antimicrobial nanoparticle coatings did not significantly improve the success rate or stability of microscrews over a short observation period, suggesting that surface modification strategies require further optimization[28].

2.2. Temporal Dynamics of Plaque Colonization and Microbial Composition

Microbial colonization around microscrew implants occurs extremely rapidly. Studies have shown that initial colonizing bacteria can be detected within 24 hours after implant placement, with *Streptococci* being the predominant early colonizers[29]. A study by de Freitas et al. further confirmed this finding. The study monitored microbial colonization dynamics in 15 microscrews over a 3-month period and found that from baseline to 24 hours, the colonization of Streptococcus species increased significantly, constituting the primary initial colonizing microbiota. Over time, the relative proportion of anaerobic bacteria in the total microbial community gradually increased, suggesting that a microenvironment conducive to anaerobic bacterial growth is established around the implant[30].

A study employing 16S rRNA gene sequencing longitudinally analyzed microbial community changes in 8 orthodontic patients at 1 week and 1 month after microscrew placement. It was found that at 4 weeks after implantation, the α-diversity of the microbiota in the peri-implant sulcular fluid decreased significantly, while the relative abundance of periodontal pathogens such as *Porphyromonas gingivalis*, Treponema denticola, and Fusobacterium nucleatum increased. Notably, although clinical examinations revealed no obvious signs of inflammation, the microbial community had already undergone significant changes, indicating that microbial-level alterations may precede clinical manifestations[31].

2.2.1. Differences Between Peri-Implant and Healthy Periodontal Microbiota

Regarding the composition of colonizing microbiota, the microbial community of the peri-implant sulcus of microscrew implants exhibits both overlap with and differences from that of adjacent healthy periodontium. A large-sample study by Mishra et al. on 102 microscrews showed that the proportions of Staphylococci, opportunistic pathogens of the Enterobacteriaceae family including *Klebsiella*, *Acinetobacter*, *Enterobacter, Pseudomonas*, and *Citrobacter*, and anaerobic cocci such as *Parvimonas micra* were significantly higher in peri-implant samples compared to gingival crevicular fluid samples. The study also found that the isolation frequency of *Veillonella* was significantly elevated in the peri-implant microbiota[32]. Of note, the detection rates of classically recognized periodontal pathogens such as *Porphyromonas gingivalis*, Treponema denticola, and Tannerella forsythia were relatively low around microscrew implants. This phenomenon may be related to the relatively short duration of microscrew use, typically only a few months, as classical periodontal pathogens require longer microbial succession for colonization. Additionally, age influences microbial composition: in younger patients aged ≤14 years, higher detection rates of Citrobacter and Parvimonas micra were observed around microscrews compared to the >14-year age group[32].

ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

2.2.2. Microbial Differences Between Successful and Failed Implants

In cases of implant failure, the enrichment of specific microbial groups is particularly prominent. Mishra et al. demonstrated significantly higher detection rates of *Staphylococci*, *Enterococci*, and *Parvimonas micra* in failed microscrews. Specifically, among failure cases in the >14-year age group, the detection rates of *Staphylococci* with P=0.008, Enterococci with P=0.011, and Parvimonas micra with P<0.001 were significantly higher than those in the successful group[32]. These data strongly suggest that these microorganisms may be closely associated with the loss of implant stability. However, differing viewpoints exist in the academic community regarding the causal relationship between bacterial colonization and implant failure[33]. Apel et al., analyzing 76 microscrews including 8 failed ones, found no significant differences in total bacterial load or species composition between failed and successful groups, only observing higher detection rates of *Actinomyces viscosus* and Campylobacter gracilis in the successful group[33]. Ferreira et al., using scanning electron microscopy to morphologically examine bacterial biofilms on the surfaces of 12 microscrews including 7 successful and 5 failed ones, found extensive bacterial colonization on the heads and transmucosal necks of all successful and failed microscrews, but none of the failed cases exhibited bacterial detection on the body, the intraosseous portion. The study concluded that no direct association was found between failure and bacterial colonization on implant surfaces[34].

2.2.3. Ecological Succession Mechanisms of Peri-Implant Microbiota

The insertion of microscrew implants creates a new artificial ecological niche. This change may affect the local microenvironment through the dysbiosis-inflammation axis. Studies have found that nitrate-reducing bacteria and Saccharibacteria (TM7) remain stable around microscrews and exhibit an antagonistic relationship with periodontal pathogens. Nitrate-reducing bacteria convert dietary nitrate into nitrite and nitric oxide, potentially playing a protective role in maintaining oral microecological homeostasis. Saccharibacteria, as ultramicrobacteria that depend on host bacteria such as Schaalia odontolytica for survival, their stability following microscrew implantation suggests potential involvement in microecological self-regulatory mechanisms[31]. This finding provides new insights for exploring biological strategies to maintain peri-implant microecological balance.

2.3. Impact of Insertion Site and Cleaning Difficulty on Microecology

Microscrews are often placed in regions such as the interradicular alveolar ridge of the posterior maxilla and mandible, the buccal shelf, or the infrazygomatic crest. The anatomical characteristics of these sites determine the difficulty of cleaning: some implants may be covered by buccal soft tissues or positioned posteriorly, making them inaccessible to routine oral hygiene tools. Clinical studies have confirmed that after microscrew implantation, patients' plaque index, probing depth, and bleeding on probing index significantly increase and tend to rise over time[35].

The choice of insertion site is an important determinant of the peri-implant microecology. When microscrews are placed in attached gingiva, the surrounding keratinized mucosa can form a favorable soft tissue seal, helping to reduce plaque accumulation and inflammatory reactions. In contrast, when placed in movable mucosa, the peri-implant soft tissue stability is poorer, prone to inflammatory hyperplasia or cratering, creating conditions for plaque retention[36]. Additionally, posterior regions adjacent to the parotid duct openings have higher local salivary flow rates, theoretically providing stronger self-cleaning ability, though this also implies that the microbial profile in this area may differ from that of the anterior oral cavity. When implants are covered by soft tissue or located in areas that are difficult to clean, plaque retention time is prolonged, the proportion of anaerobic bacteria further increases, and inflammatory risks consequently rise[37].

Researchers believe that microscrews, as foreign bodies in the oral cavity, not only themselves serve as surfaces for plaque adhesion but also impede patients from performing routine oral hygiene, leading to plaque accumulation and local inflammatory reactions. The presence of orthodontic appliances such as archwires, springs, and ligature rings further exacerbates this predicament: the narrow gaps between these appliances and the microscrew head become retention sites for food debris and plaque, which are difficult to clean effectively with conventional toothbrushes[38]. Therefore, patients need to use auxiliary tools such as interdental brushes, single-tuft brushes, or water flossers for meticulous cleaning around microscrews, imposing high demands on patient compliance and operational skills.

Differences in microbial composition also exist among different insertion sites. Studies have shown that the predominant bacterial genera differ between maxillary and mandibular microscrews, potentially related to differences in local salivary flow rate, temperature, oxygen concentration, and the

ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

microbial reservoir of adjacent natural teeth. Furthermore, some scholars have pointed out that peri-implant regions are generally difficult to self-clean, making them prone to plaque accumulation[32, 39]. It is noteworthy that in the early stage (1-2 weeks) after microscrew implantation, plaque accumulation is dominated by aerobic and facultative anaerobic bacteria. However, as plaque maturation time increases, the proportion of Gram-negative anaerobic bacteria gradually rises[40, 41], a succession process closely associated with an increased risk of peri-implantitis.

2.4. Microecological Impact during Microscrew Implantation and Healing

The process of microscrew implantation itself triggers a host inflammatory response. If bacterial invasion occurs during or after implantation, the inflammatory response will be further exacerbated, leading to loss of implant stability[32]. Furthermore, oral microecological imbalance may impede the normal healing process after microscrew implantation. The microcrack hypothesis proposes that due to the mismatch in elastic modulus between bone tissue and microscrew implants, microcracks form at the bone-implant interface after microscrew placement[42]. Under normal healing conditions, these microcracks can be repaired through calcium phosphate-induced bone mineralization. However, if microbial invasion occurs during the repair process, it can interfere with the mineralization process, affecting the quality and efficiency of osseointegration[43]. This further illustrates the important role of microecological factors in the early stage of implant healing.

3. Oral Microecological Imbalance Leading to Microscrew Implant Anchorage Failure

3.1. Microecological Factors: Enrichment of Pathogenic Microbiota and Virulence Factors

Dysbiosis is the initiating factor for peri-implant lesions. In failed microscrew implant cases, the detection rates of *Staphylococci*, Enterococci, and Parvimonas micra are significantly higher in failed microscrews compared to successful ones, suggesting a close association between these microorganisms and loss of implant stability[44]. As plaque maturation time increases, the microbial community structure undergoes dynamic succession. Studies have shown that the early-stage microbiota, dominated by aerobic and facultative anaerobic bacteria, gradually transitions to a community dominated by Gram-negative anaerobic bacteria[40, 41]. 16S rRNA gene sequencing studies have further revealed that at 4 weeks after microscrew implantation, the α-diversity of the peri-implant sulcular fluid microbiota significantly decreases, while the relative abundance of periodontal pathogens such as *Porphyromonas gingivalis*, Treponema denticola, and Fusobacterium nucleatum increases[45].

These pathogenic bacteria produce various virulence factors that directly damage peri-implant tissues[46]. Lipopolysaccharide, a major component of the Gram-negative bacterial outer membrane, activates host immune cells and induces the release of large quantities of inflammatory factors[47]. Adhesion factors such as fimbriae and lectins facilitate bacterial colonization on implant surfaces and epithelial cells, forming biofilms that are difficult to eradicate[48]. Proteases and toxins directly degrade the extracellular matrix, compromising the integrity of soft tissues[49]. Furthermore, acidic end products and toxic small molecules generated by bacterial metabolism can directly suppress osteoblast activity and interfere with normal bone metabolism[50].

3.2. Host Factors: Dysregulation of Immune-Inflammatory Responses

The host immune response to pathogenic bacteria is a protective reaction. However, excessive or persistent inflammation can become a major driver of tissue destruction. When dysbiosis occurs, pattern recognition receptors, primarily Toll-like receptors, in peri-implant tissues recognize pathogen-associated molecular patterns on pathogenic bacterial surfaces. This recognition activates downstream inflammatory signaling pathways such as NF-κB and MAPK, thereby initiating a cascade of inflammatory responses[51, 52]. During this process, a large number of pro-inflammatory cytokines and chemokines are released. Various inflammatory mediators, including IL-1β, IL-6, IL-8, and TNF-α, can be detected in the peri-implant sulcular fluid (PMICF) around microscrew implants[53]. Among these, IL-1β and TNF-α are core factors mediating bone resorption: they not only directly activate osteoclasts but also stimulate osteoblasts and stromal cells to express RANKL, further enhancing osteoclast differentiation and activity[54, 55]. IL-8 primarily exerts chemotactic effects, recruiting neutrophils to the site of inflammation. These recruited neutrophils, while releasing reactive oxygen species and proteases to eliminate pathogens, also cause oxidative damage and matrix degradation in surrounding tissues[56].

ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

Matrix degradation is mainly mediated by matrix metalloproteinases (MMPs). Under the stimulation of inflammatory factors such as TNF-α and IL-1β, MMP-9 expression is significantly upregulated, leading to the degradation of various extracellular matrix components. Studies have confirmed that MMP-9 levels in PMICF are positively correlated with clinical indicators such as plaque index, bleeding on probing index, and probing depth, making it an important biomarker reflecting peri-implant tissue health status[56, 57].

3.3. Microbiota-Host Interactions: Positive Feedback Loop and Loss of Implant Stability

A complex bidirectional interaction exists between microecological imbalance and host inflammatory responses. The two mutually reinforce each other, forming a positive feedback loop that ultimately leads to progressive destruction of peri-implant tissues.

On the one hand, dysbiosis activates the host inflammatory response. Bacteria and their products such as lipopolysaccharide activate immune cells through Toll-like receptors, initiating an inflammatory cascade. While eliminating pathogens, this process also alters the local microenvironment. Inflammatory exudates provide novel nutrient sources for specific pathogens. Tissue hypoxia and an acidic environment favor the growth of anaerobic and opportunistic bacteria. The living space for commensal bacteria is further compressed[39, 58]. This alteration of the microenvironment in turn exacerbates dysbiosis, forming a vicious cycle characterized by dysbiosis exacerbating inflammation and inflammation further worsening dysbiosis[59, 60]. On the other hand, inflammation disrupts the soft tissue seal around the implant. A biological seal, consisting of epithelial attachment and connective tissue cuff, exists between the microscrew and surrounding soft tissue[61]. When dysbiosis and inflammation persist, the epithelial attachment loosens, and the expression of intercellular tight junction proteins is downregulated, leading to biological seal failure[62]. Once an effective seal is lost, more bacteria can invade the deeper portions of the implant, further activating immune responses and accelerating bone resorption[63].

At the level of bone metabolism, inflammatory factors mediate bone resorption by regulating the RANKL/RANK/OPG system[64, 65]. Pro-inflammatory cytokines promote the high expression of RANKL by osteoblasts and activated T cells while inhibiting the expression of OPG, leading to an elevated RANKL/OPG ratio[66]. RANKL binds to RANK on the surface of osteoclast precursor cells, activating the NF-κB and MAPK signaling pathways[67], thereby promoting the differentiation, activation, and survival of osteoclasts, ultimately accelerating alveolar bone resorption. As bone resorption progresses, the effective osseointegration length of the microscrew gradually decreases, mechanical stability declines, and eventually loosening and loss occur[68]. Of note, once this positive feedback loop is established, it is often difficult to reverse with antibacterial therapy alone[69]. Therefore, early identification of signs of dysbiosis during the initial stage of microscrew implantation, and interruption of the vicious cycle of microbiota-inflammation, are of great significance for preventing peri-implantitis and implant failure.

4. Discussion and Conclusions

Current research on the relationship between microscrew implant anchorage and oral microecology still presents several key controversies and gaps.

The causal relationship remains unclear. Most existing studies are cross-sectional in design, which can only confirm a correlation between dysbiosis and peri-implantitis but cannot distinguish between cause and effect[70]. It remains unknown whether the enrichment of specific pathogenic bacteria triggers inflammation, or whether the inflammatory microenvironment selectively promotes the growth of these bacteria[71]. Clarifying this question is critical for guiding the development of prevention strategies. Longitudinal tracking of dynamic succession is lacking. The majority of studies have focused on microbial characteristics at the mature stage, while little is known about the dynamic changes in the microbiome during the continuous transition from health to mucositis and then to peri-implantitis[72]. As a result, the key nodes driving disease progression remain difficult to identify[45]. Heterogeneity in research findings also warrants attention. Mishra et al. reported significant associations of *Staphylococci*, Enterococci, and Parvimonas micra with implant failure[32], whereas Apel et al. and Ferreira et al. observed no such correlations[33, 73]. These discrepancies may arise from differences in study populations, implant sites, duration of microscrew use, and microbial detection methods such as culture-based approaches, sequencing, or morphological observation. There remains a lack of

ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

standardized, multicenter, large-sample studies with unified methodologies[74], which largely limits the generalizability of current conclusions.

In terms of preventive and therapeutic strategies, although surface-modified coatings and antimicrobial materials have shown promise in in vitro and animal studies, their clinical translation remains challenging[75, 76]. The long-term stability and safety of antimicrobial coatings in the complex oral environment have yet to be validated. Postoperative plaque control measures, such as chlorhexidine mouthwash, may reduce plaque in the short term, but long-term use carries the risk of inducing dysbiosis and antimicrobial resistance[77, 78]. Of note, current research has largely focused on strategies aimed at killing pathogenic bacteria, whereas ecoregulatory strategies based on promoting commensal bacteria such as nitrate-reducing bacteria remain underexplored and may represent a more promising direction for future research[79].

In summary, future studies should focus on the following directions. First, longitudinal cohort studies should be conducted to clarify the temporal relationship between microbial changes and disease progression. Second, methodological approaches should be standardized to enhance comparability across studies. Third, metagenomic and metabolomic techniques should be employed to dissect pathogenic mechanisms at the functional level of the microbiome. Fourth, microbiome-based intervention strategies, including probiotics, prebiotics, or phage therapy, should be explored to facilitate a paradigm shift from killing to regulation. Only through a deep understanding of the dynamic interplay between microscrew implants and the oral microbiome can the transition from passive anti-infection to active ecological regulation be achieved.

References

[1] Zhang G N, Liu Y, Li W Y, Fan T T, Wang J, Zhao Z J. Correlation between social psychology and personality characteristics and treatment options for adult patients with skeletal malocclusion[J]. West China Journal of Stomatology, 2020, 38(3): 312-317. (in Chinese)

[2] Mizrahi, E., The use of Miniscrews in Orthodontics: A Review of Selected Clinical Applications. Primary Dental Journal, 2016. 5(4): p. 20-27.

[3] Rosier, B.T., P.D. Marsh, and A. Mira, Resilience of the Oral Microbiota in Health: Mechanisms That Prevent Dysbiosis. J Dent Res, 2018. 97(4): p. 371-380.

[4] Balenseifen, J.W. and J.V. Madonia, Study of dental plaque in orthodontic patients. J Dent Res, 1970. 49(2): p. 320-4.

[5] Feldmann, I. and L. Bondemark, Orthodontic anchorage: a systematic review. Angle Orthod, 2006. 76(3): p. 493-501.

[6] Li L Q. Clinical application of micro-implant anchorage in orthodontics[J]. Chinese Community Doctors (Medical Professional), 2011, 13(30): 45. (in Chinese)

[7] Ma Y S, Han B, Liu Y, Bai Y F, Zhang X H. In vitro study of multifunctional orthodontic adhesive for preventing enamel demineralization[J]. Beijing Journal of Stomatology, 2019, 27(6): 306-312. (in Chinese)

[8] Park, H.S., S.H. Jeong, and O.W. Kwon, Factors affecting the clinical success of screw implants used as orthodontic anchorage. Am J Orthod Dentofacial Orthop, 2006. 130(1): p. 18-25.

[9] Jin A P, Hu X C, Huang Y Y. Application of micro-implant anchorage in orthodontics[J]. Chinese Journal of Aesthetic Medicine, 2011, 20(6): 968-969. (in Chinese)

[10] Zhou S S, Sun Y, Wu G P, Song D, Li Y. Alkali-producing metabolism of plaque biofilm and dental caries[J]. International Journal of Stomatology, 2016, 43(5): 573-577. (in Chinese)

[11] Ghannoum, M.A., et al., Characterization of the oral fungal microbiome (mycobiome) in healthy individuals. PLoS Pathog, 2010. 6(1): p. e1000713.

[12] Pride, D.T., et al., Evidence of a robust resident bacteriophage population revealed through analysis of the human salivary virome. Isme j, 2012. 6(5): p. 915-26.

[13] Matarazzo, F., et al., Diversity and quantitative analysis of Archaea in aggressive periodontitis and periodontally healthy subjects. J Clin Periodontol, 2011. 38(7): p. 621-7.

[14] Liu, X., et al., Analysis of subgingival microbiome of periodontal disease and rheumatoid arthritis in Chinese: A case-control study. Saudi J Biol Sci, 2020. 27(7): p. 1835-1842.

[15] Jiang, Q., et al., The Oral Microbiome in the Elderly With Dental Caries and Health. Front Cell Infect Microbiol, 2018. 8: p. 442.

[16] Chen, W.P., et al., Composition Analysis and Feature Selection of the Oral Microbiota Associated with Periodontal Disease. Biomed Res Int, 2018. 2018: p. 3130607.

[17] Long, J., et al., Association of oral microbiome with type 2 diabetes risk. J Periodontal Res, 2017. 52(3): p. 636-643.

ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

[18] Farrugia, C., G.P. Stafford, and C. Murdoch, Porphyromonas gingivalis Outer Membrane Vesicles Increase Vascular Permeability. J Dent Res, 2020. 99(13): p. 1494-1501.
[19] Han, Y.W. and X. Wang, Mobile microbiome: oral bacteria in extra-oral infections and inflammation. J Dent Res, 2013. 92(6): p. 485-91.
[20] Scarano, A., et al., Bacterial adhesion on commercially pure titanium and anatase-coated titanium healing screws: an in vivo human study. J Periodontol, 2010. 81(10): p. 1466-71.
[21] Nascimento, C.D., et al., Bacterial adhesion on the titanium and zirconia abutment surfaces. Clin Oral Implants Res, 2014. 25(3): p. 337-343.
[22] van Brakel, R., et al., Early bacterial colonization and soft tissue health around zirconia and titanium abutments: an in vivo study in man. Clin Oral Implants Res, 2011. 22(6): p. 571-7.
[23] Bollen, C.M., et al., The influence of abutment surface roughness on plaque accumulation and peri-implant mucositis. Clin Oral Implants Res, 1996. 7(3): p. 201-11.
[24] Almaguer-Flores, A., et al., Bacterial Adhesion on Polyelectrolyte Modified Microstructured Titanium Surfaces. MRS Online Proceedings Library, 2011. 1277(1): p. 1.
[25] Amarante, E.S., et al., Early dental plaque formation on toothbrushed titanium implant surfaces. Am J Dent, 2008. 21(5): p. 318-22.
[26] Fathy Abo-Elmahasen, M.M., et al., Do silver/hydroxyapatite and zinc oxide nano-coatings improve inflammation around titanium orthodontic mini-screws? In vitro study. Int Orthod, 2023. 21(1): p. 100711.
[27] Aboelmahasen, M.M.F., et al., Histomorphometric and CBCT comparison of osseointegration around orthodontic titanium miniscrews coated with different nanoparticles: An in-vivo animal study. Int Orthod, 2024. 22(1): p. 100823.
[28] Al-Hilaly, A.A. and A.F. Alhuwaizi, Assessment of coated orthodontic miniscrews with chlorhexidine hexametaphosphate antimicrobial nanoparticles: A randomized clinical trial. J World Fed Orthod, 2025. 14(2): p. 67-75.
[29] Nyvad, B. and M. Kilian, Microbiology of the early colonization of human enamel and root surfaces in vivo. Scand J Dent Res, 1987. 95(5): p. 369-80.
[30] de Freitas, A.O., et al., Microbial colonization in orthodontic mini-implants. Braz Dent J, 2012. 23(4): p. 422-7.
[31] Bachtiar, B.M., et al., Four-Week Evaluation of the Interaction Pattern Among Saccharibacteria, Nitrate-Reducing Bacteria, and Periodontopathogens in Orthodontic Miniscrew Implants. Dent J (Basel), 2025. 13(9).
[32] Mishra, G., et al., A pattern of microbiological colonization of orthodontic miniscrew implants. Am J Orthod Dentofacial Orthop, 2023. 164(4): p. 554-566.
[33] Apel, S., et al., Microflora associated with successful and failed orthodontic mini-implants. Clin Oral Implants Res, 2009. 20(11): p. 1186-90.
[34] Ferreira, N.O., et al., Bacterial biofilm on successful and failed orthodontic mini-implants--a scanning electron microscopy study. Microsc Res Tech, 2015. 78(12): p. 1112-6.
[35] Ghanbari, H.O., et al., Evaluation of periodontal condition in intruded molars using miniscrews. Journal of Dental Materials and Techniques, 2015. 4(4): p. 145-152.
[36] Song, Y.Y., J.Y. Cha, and C.J. Hwang, Mechanical characteristics of various orthodontic mini-screws in relation to artificial cortical bone thickness. Angle Orthod, 2007. 77(6): p. 979-85.
[37] Qian, Y., H.J. Zhou, and J.H. Wu, Clinical effects of micro-implant and traditional anchorage in orthodontic treatments. Shanghai Journal of Stomatology, 2017. 26(3): p. 339-342.
[38] Favero, R., et al., Maintaining Hygiene in Orthodontic Miniscrews: Patient Management and Protocols-A Literature Review. Dent J (Basel), 2024. 12(7).
[39] Cui, Z., P. Wang, and W. Gao, Microbial dysbiosis in periodontitis and peri-implantitis: pathogenesis, immune responses, and therapeutic. Front Cell Infect Microbiol, 2025. 15: p. 1517154.
[40] Thurnheer, T., N. Bostanci, and G.N. Belibasakis, Microbial dynamics during conversion from supragingival to subgingival biofilms in an in vitro model. Mol Oral Microbiol, 2016. 31(2): p. 125-35.
[41] Kinniment, S.L., et al., Development of a steady-state oral microbial biofilm community using the constant-depth film fermenter. Microbiology (Reading), 1996. 142 (Pt 3): p. 631-638.
[42] Shank, S.B., et al., Bone damage associated with orthodontic placement of miniscrew implants in an animal model. Am J Orthod Dentofacial Orthop, 2012. 141(4): p. 412-8.
[43] Bartold, P.M., et al., Influence of surface roughness and shape on microdamage of the osseous surface adjacent to titanium dental implants. Clin Oral Implants Res, 2011. 22(6): p. 613-8.
[44] Di Spirito, F., et al., Microbiota of Peri-Implant Healthy Tissues, Peri-Implant Mucositis, and Peri-Implantitis: A Comprehensive Review. Microorganisms, 2024. 12(6).
[45] Jia, P., et al., Microbiome of diseased and healthy implants-a comprehensive microbial data analysis. Front Cell Infect Microbiol, 2024. 14: p. 1445751.

ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

[46] Wen Y, Wang Y L. Research progress on plaque biofilm and peri-implantitis[J]. Journal of Prevention and Treatment for Stomatological Diseases, 2024, 32(9): 730-736. (in Chinese)
[47] Luo, R., et al., An examination of the LPS-TLR4 immune response through the analysis of molecular structures and protein-protein interactions. Cell Commun Signal, 2025. 23(1): p. 142.
[48] Korea, C.G., J.M. Ghigo, and C. Beloin, The sweet connection: Solving the riddle of multiple sugar-binding fimbrial adhesins in Escherichia coli: Multiple E. coli fimbriae form a versatile arsenal of sugar-binding lectins potentially involved in surface-colonisation and tissue tropism. Bioessays, 2011. 33(4): p. 300-11.
[49] Uitto, V.J., et al., A protease of Bacteroides gingivalis degrades cell surface and matrix glycoproteins of cultured gingival fibroblasts and induces secretion of collagenase and plasminogen activator. Infect Immun, 1989. 57(1): p. 213-8.
[50] Chang, M.C., et al., Effect of Butyrate on Collagen Expression, Cell Viability, Cell Cycle Progression and Related Proteins Expression of MG-63 Osteoblastic Cells. PLoS One, 2016. 11(11): p. e0165438.
[51] Chen H J, Wang D, Shen S. Mechanism of immune-inflammatory microenvironment in peri-implantitis[J]. Chinese Journal of Tissue Engineering Research, 2026, 30(8): 2054-2062. (in Chinese)
[52] Li, Y., et al., Immune dysregulation and macrophage polarization in peri-implantitis. Front Bioeng Biotechnol, 2024. 12: p. 1291880.
[53] Morandini, A.C., et al., Editorial: Microbial dysbiosis and immune dysregulation in periodontitis and peri-implantitis. Front Cell Infect Microbiol, 2025. 15: p. 1637248.
[54] Zou, W. and Z. Bar-Shavit, Dual modulation of osteoclast differentiation by lipopolysaccharide. J Bone Miner Res, 2002. 17(7): p. 1211-8.
[55] McGowan, N.W., et al., Cytokine-activated endothelium recruits osteoclast precursors. Endocrinology, 2001. 142(4): p. 1678-81.
[56] Sheikhi, M., A. Gustafsson, and C. Jarstrand, Cytokine, elastase and oxygen radical release by Fusobacterium nucleatum-activated leukocytes: a possible pathogenic factor in periodontitis. J Clin Periodontol, 2000. 27(10): p. 758-62.
[57] He, W., H. Zhu, and C. Liu, Profiles of inflammation factors and inflammatory pathways around the peri-miniscrew implant. Histol Histopathol, 2021. 36(9): p. 899-906.
[58] Hall, J.W. and Y. Ji, Sensing and Adapting to Anaerobic Conditions by Staphylococcus aureus. Adv Appl Microbiol, 2013. 84: p. 1-25.
[59] Higashi, D.L., et al., An inflammatory paradox: strategies inflammophilic oral pathobionts employ to exploit innate immunity via neutrophil manipulation. Front Oral Health, 2024. 5: p. 1413842.
[60] Mehrnia, N. and T.E. Van Dyke, Microbial dysbiosis and immune dysregulation in periodontitis and peri-implantitis. Front Cell Infect Microbiol, 2025. 15: p. 1678163.
[61] Gibbs, S., et al., Biology of soft tissue repair: gingival epithelium in wound healing and attachment to the tooth and abutment surface. Eur Cell Mater, 2019. 38: p. 63-78.
[62] Jin, C., et al., Substrate roughness induces the development of defective E-cadherin junctions in human gingival keratinocytes. J Periodontal Implant Sci, 2017. 47(2): p. 116-131.
[63] Surrency, S., et al., 3D Peri-Implant Epi-Mucosa-on-a-Chip Reveals Alterations in Epithelial Barrier Function Mediated by Host-Bacteria-Biomaterial Interactions. ACS Biomater Sci Eng, 2025. 11(12): p. 7134-7148.
[64] Udagawa, N., et al., Osteoclast differentiation by RANKL and OPG signaling pathways. J Bone Miner Metab, 2021. 39(1): p. 19-26.
[65] Kearns, A.E., S. Khosla, and P.J. Kostenuik, Receptor activator of nuclear factor kappaB ligand and osteoprotegerin regulation of bone remodeling in health and disease. Endocr Rev, 2008. 29(2): p. 155-92.
[66] Kong, Y.Y., et al., Activated T cells regulate bone loss and joint destruction in adjuvant arthritis through osteoprotegerin ligand. Nature, 1999. 402(6759): p. 304-9.
[67] Wada, T., et al., RANKL-RANK signaling in osteoclastogenesis and bone disease. Trends Mol Med, 2006. 12(1): p. 17-25.
[68] Schluessel, S., et al., Dental and Orthopaedic Implant Loosening: Overlap in Gene Expression Regulation. Front Immunol, 2022. 13: p. 820843.
[69] Hajishengallis, G. and T. Chavakis, Local and systemic mechanisms linking periodontal disease and inflammatory comorbidities. Nat Rev Immunol, 2021. 21(7): p. 426-440.
[70] Chen, G., et al., Peri-implant diseases triggered by oral microdysbiosis: pathogenesis and precision intervention strategies. Front Microbiol, 2025. 16: p. 1639095.

ISSN 2706-6819 Vol.8, Issue 2: 35-44, DOI: 10.25236/IJFM.2026.080204

[71] Talib, E.Q., et al., Microbial boundaries in peri-implantitis: a review of pathogen-related advances. Folia Med (Plovdiv), 2024. 66(6): p. 763-769.
[72] Vílchez, B., et al., Longitudinal Changes in the Peri-Implant Microbiome Following Implant Placement: A 3-Year Follow-Up Study. Clin Oral Implants Res, 2026.
[73] Ferreira, S.D., et al., Prevalence and risk variables for peri-implant disease in Brazilian subjects. J Clin Periodontol, 2006. 33(12): p. 929-35.
[74] Darby, I., Risk factors for periodontitis & peri-implantitis. Periodontol 2000, 2022. 90(1): p. 9-12.
[75] Gnanasekar, S., et al., Antibacterial MXenes: An emerging non-antibiotic paradigm for surface engineering of orthopedic and dental implants. Bioact Mater, 2025. 51: p. 150-176.
[76] Jadhav, L., V. Madiwal, and J.M. Rajwade, The dental implant surface: a review of the past, present and future. J Mater Sci Mater Med, 2026. 37(1): p. 21.
[77] Ren J Y, Zhou W J. Research progress on decreased susceptibility of oral bacteria to chlorhexidine and related mechanisms[J]. Journal of Oral Science Research, 2024, 40(8): 670-675. (in Chinese)
[78] Bartsch, S., et al., Chlorhexidine digluconate mouthwash alters the oral microbial composition and affects the prevalence of antimicrobial resistance genes. Front Microbiol, 2024. 15: p. 1429692.
[79] Joshi, A.A., et al., The Submucosal Microbiome Correlates with Peri-implantitis Severity. J Dent Res, 2026. 105(3): p. 313-322.