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# The Oral Microbiome in Health and Disease: Microbial Ecology, Dysbiosis, and Clinical Implications for Dental Practice

**Zaman Kareem Hanan**[1,*], **Adnan Abed Azeez Alazraqi**[2], **Aliaa Kareem Hanan**[3], **Ögret. Üyesi Aysel Sari**[4]

[1] *College of Medicine, University of Thi-Qar, Thi-Qar 64001, Iraq*

[2] *College of Dentistry, University of Thi-Qar, Thi-Qar 64001, Iraq*

[3] *Collage of Agriculture and Marshes, University of Thi-Qar, Thi-Qar, Iraq*

[4] *Department of Chemistry/Biochemistry, Faculty of Science, Fîrat University, Elazığ, Turkey*

[*] *Corresponding Email:* zaman.k_bio@utq.edu.iq

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**ABSTRACT**

The oral cavity harbors one of the most complex microbial ecosystems in the human body, comprising more than 700 bacterial species alongside fungi, viruses, and archaea. This review provides a comprehensive examination of the composition, ecological dynamics, and clinical significance of the oral microbiome, with particular emphasis on implications for dental practice. We describe site-specific microbial communities and biofilm succession, analyze the role of keystone pathogens in driving dental caries, gingivitis, periodontitis, peri-implantitis, and oral candidiasis, and examine the Socransky periodontal complex classification system. The bidirectional oral-systemic disease axis is explored, linking periodontal dysbiosis to cardiovascular disease, type 2 diabetes, colorectal cancer, Alzheimer's disease, and adverse pregnancy outcomes. A four-step evidence-based clinical management framework integrating diagnostic risk assessment, mechanical debridement, targeted antimicrobial and biological adjuncts, and long-term preventive maintenance is presented. Microbiome-based diagnostics and emerging precision dentistry approaches are also discussed. Understanding oral microbial ecology is indispensable for contemporary, evidence-based dental practice.

***Keywords:*** Dental Caries, Dental Plaque, Dysbiosis, Microbial Ecology, Oral Microbiome, Oral-Systemic Axis, Peri-Implantitis, Periodontal Disease, Precision Dentistry, Probiotics

## 1 Introduction

THE human oral cavity supports a remarkably diverse and stable microbial community the oral microbiome comprising more than 700 prokaryotic taxa across 185 genera catalogued in the Human Oral Microbiome Database (HOMD) [1, 2]. From a dental perspective, this community occupies a paradoxical position: in health it constitutes a protective ecological consortium resisting pathogen colonization and modulating mucosal immunity; in disease, its dysregulation underpins the two most prevalent chronic human infections dental caries and periodontal disease [3, 4, 19].

The contemporary understanding of these diseases has shifted decisively from a single-pathogen model toward an ecological framework in which community-level composition and function drive pathogenesis [4]. Advances in 16S rRNA sequencing and shotgun metagenomics have revealed that the oral microbiome is highly site-specific,

individually variable, and dynamically responsive to diet, oral hygiene, medications, and systemic health [5, 6]. This review synthesizes current microbiological evidence with clinical dental data to provide an integrated resource for dental clinicians, periodontologists, and oral medicine specialists.

## 2 Composition of the Oral Microbiome

### *2.1 Bacterial Diversity*

Six phyla consistently predominate across oral sites: Firmicutes, Bacteroidetes, Proteobacteria, Actinobacteria, Spirochaetes, and Fusobacteria [1, 2]. At genus level, Streptococcus, Veillonella, Prevotella, Fusobacterium, Haemophilus, and Actinomyces are frequently abundant. Streptococcus the dominant oral genus encompasses protective commensals (S. sanguinis, S. gordonii) and the cariogenic pathobiont S. mutans, exemplifying the context-dependent nature of oral microbial function [7]. Table 1 summarizes site-specific microbial profiles across key oral niches.

### *2.2 Non-Bacterial Microorganisms*

The oral mycobiome is dominated by Candida albicans, a commensal in 45–65% of healthy adults that undergoes pathogenic hyphal transition under immunosuppression, antibiotic dysbiosis, or denture wear [8]. High-risk oral HPV strains (HPV-16, HPV-18) are causally associated with oropharyngeal squamous cell carcinoma, making viral oral ecology relevant to dental oncology screening [9]. Methanogenic archaea (Methanobrevibacter oralis), enriched in deep periodontal pockets, sustain anaerobic metabolism through interspecies hydrogen transfer a factor in periodontitis pathophysiology [10].

## 3 Ecological Principles Governing the Oral Microbiome

### *3.1 Biofilm Succession on Tooth Surfaces*

Dental plaque the etiological substrate of caries and periodontal disease develops through a defined ecological succession illustrated in Figure 1. Pioneer aerotolerant species (S. gordonii, S. sanguinis, Actinomyces naeslundii) adhere to the salivary pellicle via adhesin-receptor interactions. Fusobacterium nucleatum serves as a critical bridging organism, co-aggregating with early colonizers and creating an anaerobic microenvironment permissive for late-colonizing periodontal pathogens [11, 12].

### *3.2 Quorum Sensing and Metabolic Ecology*

The competence-stimulating peptide (CSP) quorum sensing system in S. mutans coordinates acid tolerance response (ATR) and bacteriocin (mutacin) production core virulence traits in cariogenesis [13, 14]. Metabolic cross-feeding between acidogenic S. mutans and lactate-consuming Veillonella partially buffers plaque pH, modulating caries risk [15]. Arginine-metabolizing commensals raise plaque pH via the arginine deiminase system (ADS), an ecological mechanism exploited by arginine-containing dentifrices for caries prevention [16].

## 4 Periodontal Microbial Complexes

The Socransky complex classification derived from checkerboard DNA-DNA hybridization of subgingival plaque samples remains the foundational framework for periodontal microbial ecology [17].

**Table 1.** Major oral niches: dominant microbial genera, oxygen requirements, and dental clinical relevance.

| Oral niche | Dominant genera | Oxygen status | Clinical relevance in dentistry |
|---|---|---|---|
| Supragingival plaque | Streptococcus, Actinomyces, Rothia | Aerobic / Facultative anaerobic | Dental caries; early gingivitis |
| Subgingival plaque | Porphyromonas, Tannerella, Treponema, Fusobacterium | Obligate anaerobic | Chronic & aggressive periodontitis; alveolar bone loss |
| Saliva | Streptococcus, Veillonella, Prevotella, Neisseria | Aerobic / Facultative | Biomarker platform; caries risk assessment; xerostomia evaluation |
| Tongue dorsum | S. salivarius, Veillonella, Prevotella | Facultative anaerobic | Halitosis; volatile sulfur compound (VSC) production |
| Buccal mucosa / Hard palate | Streptococcus, Gemella, Haemophilus | Aerobic | Denture stomatitis; oral candidiasis in immunocompromised |
| Peri-implant sulcus | P. gingivalis, Staphylococcus spp., enteric rods | Anaerobic (deep sulcus) | Peri-implantitis; implant failure risk |

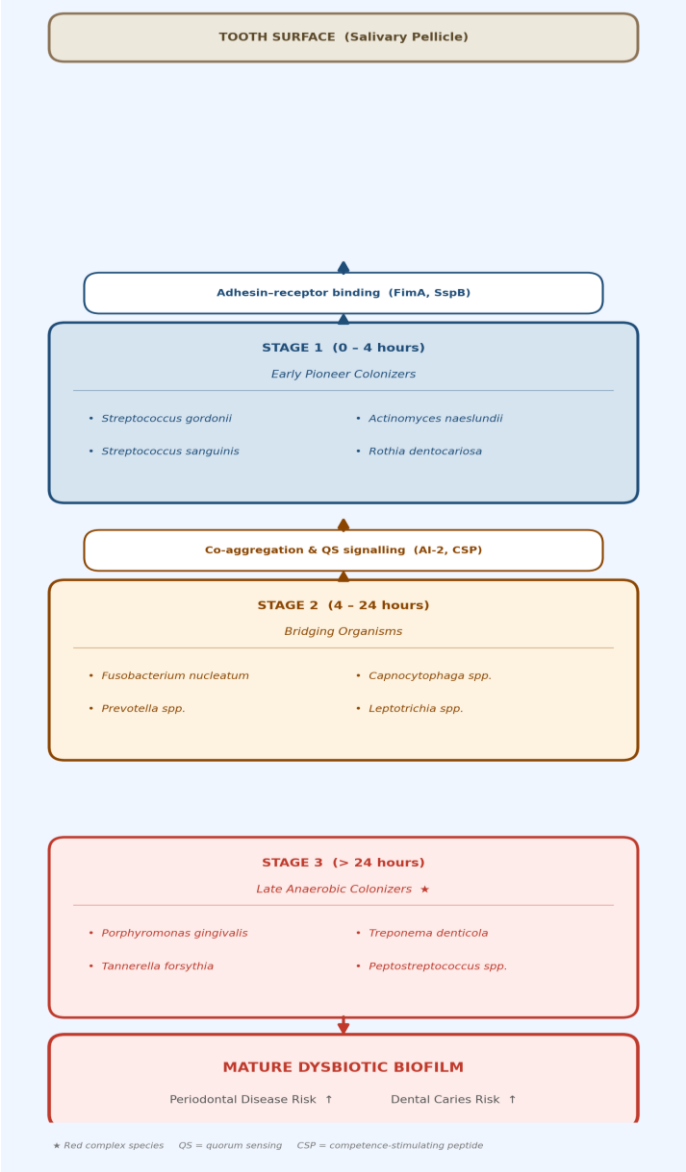


**Fig. 1.** Sequential ecological succession of the oral biofilm on the tooth surface. Pioneer aerotolerant colonizers adhere to the salivary pellicle, followed by bridging organisms, and ultimately late-stage obligate anaerobes associated with periodontal pathogenesis. QS = quorum sensing; CSP = competence-stimulating peptide.

Table 2 details all five complexes. The red complex (P. gingivalis, T. forsythia, T. denticola) most strongly correlates with severe chronic periodontitis; P. gingivalis acts as a keystone pathogen that remodels community structure disproportionate to its low abundance by disabling complement via C5aR-TLR2 crosstalk and degrading host antimicrobial peptides with gingipain proteases [18].

## 5 Dysbiosis and Oral Disease

Figure 2 illustrates the ecological tipping point model, showing the transition from eubiosis to dysbiosis driven by host and environmental factors, and the clinical pathways for microbiome restoration.

### *5.1 Dental Caries*

The ecological plaque hypothesis holds that caries results from a pH-driven community shift favoring acidogenic and aciduric organisms under frequent fermentable carbohydrate exposure [4]. Contemporary microbiome studies extend the cariogenic consortium beyond S. mutans/Lactobacillus to include Scardovia wiggsiae and Bifidobacterium spp. particularly in early childhood caries (ECC) [19, 20].

### *5.2 Periodontal Disease*

The polymicrobial synergy and dysbiosis (PSD) model establishes that P. gingivalis orchestrates dysbiosis through complement manipulation, suppressing protective IL-12 while driving destructive Th17-mediated alveolar bone resorption [18]. This supports site-specific local antimicrobial delivery doxycycline gel, CHX chip, minocycline microspheres over broad-spectrum systemic antibiotics as first-line adjuncts to SRP.

**Table 2.** Socransky microbial complexes of subgingival plaque: composition, virulence mechanisms, and clinical significance.

| Complex | Key species | Virulence mechanisms | Clinical association |
|---|---|---|---|
| Red | P. gingivalis, T. forsythia, T. denticola | Gingipains; FimA fimbriae; C5aR complement evasion; TLR antagonism | Severe chronic periodontitis; alveolar bone loss |
| Orange | F. nucleatum, P. intermedia, P. nigrescens, S. constellatus | Bridging colonization; protease production; anaerobic niche creation | Moderate periodontitis; colorectal cancer association |
| Yellow | S. mitis, S. oralis, S. sanguinis | $H_2O_2$ production; competitive exclusion of pathogens | Protective commensals; rare: infective endocarditis |
| Purple | Veillonella parvula, Actinomyces odontolyticus | Early biofilm scaffolding; lactate cross-feeding | Pioneer colonizers; caries pH modulation |
| Green | Capnocytophaga spp., A. actinomycetemcomitans | Leukotoxin (LtxA); cytolethal distending toxin | Aggressive periodontitis; systemic infection in immunocompromised |

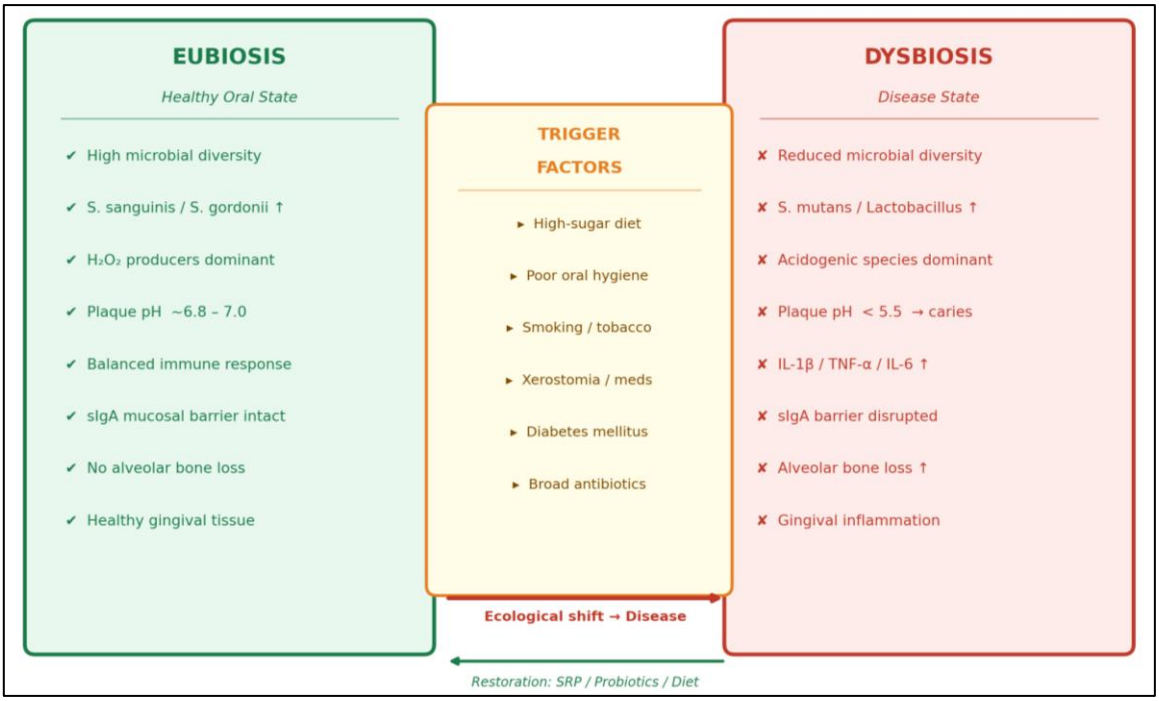


**Fig. 2.** The ecological tipping point model illustrating the transition from a healthy eubiotic oral microbiome to a dysbiotic disease state driven by environmental and host-related trigger factors. Restoration of eubiosis through scaling and root planing (SRP), probiotics, and dietary modification is the goal of microbiome-targeted dental therapy.

### *5.3 Peri-Implantitis and Oral Candidiasis*

Peri-implantitis affects approximately 22% of implants at 5 years [21]; its dysbiotic microbiome is enriched in Staphylococcus spp. and enteric rods compared to natural tooth periodontitis. C. albicans forms co-pathogenic biofilms with S. mutans exhibiting enhanced cariogenicity and antifungal resistance through β-glucan matrix integration [8]. Table 3 provides an evidence-based dental management summary for each oral disease.

## 6 The Oral-Systemic Disease Axis

Figure 3 illustrates the three principal mechanisms by which oral dysbiosis drives systemic pathology, and the bidirectional nature of the relationship.

### *6.1 Cardiovascular Disease and Diabetes*

Periodontal pathogens detected in atherosclerotic plaques promote endothelial inflammation via LPS-TLR4 activation and platelet aggregation [22]. Periodontal treatment reduces serum CRP and improves brachial endothelial function. The periodontal-diabetes relationship is bidirectional: SRP reduces HbA1c by ~0.4% over 3–6 months, comparable to adding a second-line antidiabetic drug [23].

**Table 3.** Oral diseases, microbial dysbiosis profiles, and evidence-based dental management strategies.

| Oral disease | Key dysbiotic organisms | Dysbiosis mechanism | Evidence-based dental management |
|---|---|---|---|
| Dental Caries | S. mutans, Lactobacillus spp., S. wiggsiae | Acidogenic pH-driven ecological shift | Fluoride therapy; fissure sealants; dietary counseling (sucrose reduction); arginine 1.5% dentifrices; CHX varnish |
| Gingivitis | F. nucleatum, P. intermedia, Treponema spp. | Reversible plaque-induced biofilm dysbiosis | Oral hygiene instruction; supragingival scaling; CHX / CPC mouthrinse (short-term) |
| Chronic Periodontitis | P. gingivalis, T. forsythia, T. denticola (red complex) | Keystone pathogen immune subversion (PSD model) | SRP ± systemic metronidazole + amoxicillin; surgical access flaps; supportive periodontal therapy (SPT) |
| Aggressive Periodontitis | A. actinomycetemcomitans, P. gingivalis | LtxA-mediated neutrophil killing | Early antibiotic adjuncts (azithromycin / metronidazole); full-mouth disinfection within 24 h |
| Oral Candidiasis | Candida albicans, C. tropicalis, C. glabrata | Fungal overgrowth under immunosuppression or antibiotic dysbiosis | Topical antifungals (nystatin, clotrimazole); denture hygiene; manage predisposing systemic factors |
| Peri-implantitis | P. gingivalis, T. forsythia, Staphylococcus spp. | Dysbiotic subgingival biofilm on implant surface | Mechanical debridement; Er:YAG laser decontamination; local antimicrobials (minocycline microspheres); implant surface modification |

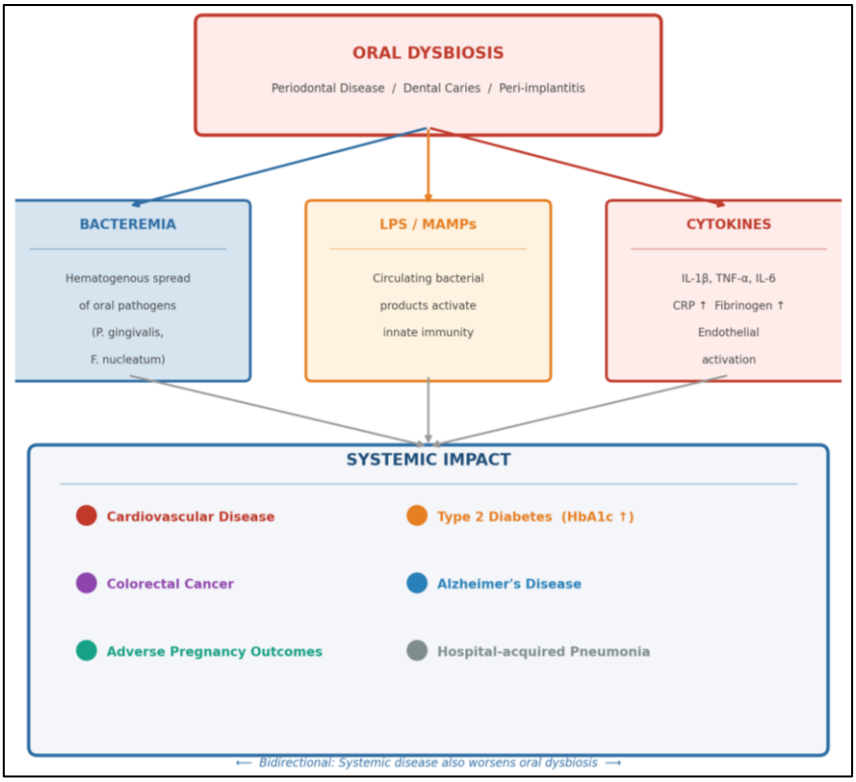


**Fig. 3.** The oral-systemic disease axis. Oral dysbiosis propagates systemic pathology through three principal pathways: hematogenous bacteremia, circulating lipopolysaccharides (LPS) and microbial-associated molecular patterns (MAMPs), and systemic pro-inflammatory cytokine dissemination. The relationship is bidirectional: systemic diseases such as diabetes mellitus and immunosuppression further exacerbate oral dysbiosis.

### 6.2 Cancer, Neurodegeneration, and Pregnancy

F. nucleatum FadA adhesin activates Wnt/β-catenin signaling in colorectal cancer tissue; salivary F. nucleatum quantification is a proposed non-invasive CRC biomarker [24]. P. gingivalis gingipains detected in Alzheimer's disease brain tissue are targets of clinical-stage inhibitors [25]. Periodontal treatment during the second trimester of pregnancy is recommended given hematogenous placental spread of oral anaerobes and risk of preterm birth [26]. Table 4 consolidates systemic associations and dental implications.

## 7 Host-Microbe Interactions and Mucosal Immunity

The oral mucosa maintains homeostatic inflammation through secretory IgA (sIgA), antimicrobial peptides (α-defensins, histatin-5, cathelicidin LL-37), and the complement cascade [27]. Commensal streptococci suppress TLR2 signaling to promote tolerance, while periodontal pathogens subvert this balance: P. gingivalis disables C5aR-TLR2-mediated IL-12 production, impairing Th1 adaptive immunity while allowing IL-6 and IL-1β-driven Th17 activation and osteoclastogenesis [18]. Histatin-5 exerts potent antifungal activity and is critically reduced in Sjögren's syndrome and post-irradiation xerostomia, explaining the dramatically elevated candidiasis and caries risk in these patient populations [7].

## 8 Microbiome-Targeted Dental Interventions

Figure 4 presents the four-step evidence-based clinical framework for managing oral microbiome dysbiosis in dental practice.

### 8.1 Mechanical Therapy and Chemical Adjuncts

Full-mouth SRP within 24 hours (FMD protocol) produces superior microbiological outcomes over quadrant-by-quadrant debridement by preventing re-infection from untreated sites [28]. CHX use should be limited to short therapeutic courses (2–4 weeks); prolonged use disrupts commensals and inhibits the oral nitrate-nitrite-NO pathway linked to blood pressure regulation [29]. Local antimicrobial delivery systems minimize systemic microbiome disruption compared to systemic antibiotics [30].

### 8.2 Probiotics, Prebiotics, and Emerging Approaches

L. reuteri (DSM 17938/ATCC PTA 5289) and S. salivarius K12/M18 reduce gingival inflammation indices and plaque S. mutans counts in randomized controlled trials [31]. Arginine 1.5% dentifrices exploit the ADS ecological mechanism to prevent caries [16]. Mediterranean diet adherence correlates with reduced periodontal severity [32]. Specifically targeted antimicrobial peptides (STAMPs) selectively eliminating S. mutans [33] and bacteriophage therapy targeting P. gingivalis represent next-generation precision ecological interventions.

**Table 4.** Oral microbiome associations with systemic diseases: mechanisms and dental management implications.

| Systemic condition | Implicated oral organisms | Proposed mechanism | Dental management implication |
|---|---|---|---|
| Cardiovascular Disease | P. gingivalis, F. nucleatum, S. sanguinis | Bacteremia; platelet aggregation; endothelial inflammation via LPS | Periodontal therapy reduces serum CRP and improves endothelial function |
| Type 2 Diabetes | Red complex organisms, Prevotella spp. | Bidirectional: LPS impairs insulin signaling; hyperglycemia worsens dysbiosis | SRP reduces HbA1c ~0.4%; glycemic control essential for periodontal healing |
| Colorectal Cancer | Fusobacterium nucleatum, Peptostreptococcus micra | FadA adhesin activates Wnt/β-catenin; TIGIT immune checkpoint engagement | Salivary F. nucleatum levels proposed as non-invasive CRC screening biomarker |
| Alzheimer's Disease | P. gingivalis, T. denticola | Gingipain-mediated neuroinflammation; bacterial DNA detected in brain tissue | Regular periodontal care in elderly may reduce systemic inflammatory burden |
| Adverse Pregnancy Outcomes | F. nucleatum, Prevotella spp. | Hematogenous placental spread; $PGE_2$-induced preterm labor | Periodontal treatment in 2nd trimester recommended to reduce preterm birth risk |
| Hospital-acquired Pneumonia | S. aureus, Klebsiella spp. (via aspiration) | Oral bacteria aspirated to lower respiratory tract | CHX oral hygiene protocols reduce VAP incidence ~40% in mechanically ventilated ICU patients |


**Fig. 4.** A stepwise evidence-based clinical framework for dental management of oral microbiome dysbiosis. The four stages integrate diagnostic risk assessment, mechanical biofilm disruption, chemical and biological adjuncts, and long-term preventive maintenance. SRP = scaling and root planing; FMD = full-mouth disinfection; CHX = chlorhexidine; SPT = supportive periodontal therapy; CPP-ACP = casein phosphopeptide-amorphous calcium phosphate.

## 9 Microbiome-Based Diagnostics in Dentistry

Chair-side PCR salivary diagnostics (e.g., MyPerioPath, OralDNA Labs) identify specific periodontal pathogens and antibiotic susceptibility patterns, enabling personalized antibiotic selection. Salivary S. mutans and Lactobacillus quantification stratifies caries risk [34]. Machine learning integration of multi-omic oral datasets for predictive risk scoring of caries, periodontal disease progression, implant failure, and systemic disease represents the frontier of precision dentistry [35].

## 10 Conclusion

The oral microbiome is a sophisticated ecological system whose balance is fundamental to oral and systemic health. Dental caries, periodontal disease, peri-implantitis, and oral candidiasis are community-level ecological diseases arising from microbial dysbiosis rather than simple pathogen invasion. The bidirectional oral-systemic disease axis places dentistry at the interface of preventive medicine: periodontal management is a systemic health intervention with demonstrated benefits for glycemic control, cardiovascular risk, and pregnancy outcomes. Microbiome-targeted precision dentistry, guided by ecological principles and advanced diagnostics, constitutes the future of evidence-based clinical dental practice.

**Conflict of Interest:** The authors declare no conflict of interest.
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**Ethical consideration:** The study was approved by Thi-Qar University, Thi-Qar, Iraq.

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