The Role of Oral Microbiome in Preventive and Personalized Dentistry: A Comprehensive Review

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***Abstract*—The oral microbiome is a complex, dynamic ecosystem comprising over 700 microbial species that maintain a symbiotic relationship with the host through immune modulation, metabolic regulation, and ecological competition against pathogens. Disruption of this balance dysbiosis drives the pathogenesis of dental caries, periodontal disease, and peri-implantitis, and is increasingly implicated in systemic disease. This review examines the composition and developmental ecology of the oral microbiome, the mechanisms underpinning host-microbial homeostasis, and the pathways through which ecological imbalance leads to clinical disease. It further explores the emerging paradigm of personalised dentistry, encompassing salivary diagnostics, biomarker-guided risk stratification, and the CAMBRA model, alongside microbiome-informed preventive strategies including fluoride therapy, alkali-generating agents, probiotics, antimicrobial peptides, and targeted microbiome modulation. The findings collectively support a fundamental reorientation of dental practice from pathogen elimination toward ecological restoration advocating for individualised, evidence-based care as the standard for long-term oral health management.**

***Index Terms*—oral microbiome, dysbiosis, personalised dentistry, salivary diagnostics, ecological plaque hypothesis, caries risk assessment.**

I. INTRODUCTION

The oral microbiome, comprising bacteria, fungi, viruses, archaea, and protozoa, is the second-largest microbial community in the human body and an indispensable participant in host physiology.[1] Far from passive colonisers, these microorganisms actively contribute to immune regulation, metabolic function, mucosal integrity, and ecological defence against pathogens.[2]

For much of the twentieth century, dental diseases such as caries and periodontal disease were attributed to specific pathogenic species, directing clinical practice toward targeted pathogen elimination through antibiotics, antiseptics, and surgical debridement. This paradigm has since been supplanted by the ecological plaque hypothesis, which recognises that disease arises not from exogenous infection but from a disruption of the resident microbial community, a state termed dysbiosis.[3] Environmental stressors, including a diet high in fermentable carbohydrates, impaired salivary flow, smoking, and systemic illness, selectively favour pathogenic species, shifting the biofilm toward virulence and persistent inflammation. Critically, this ecological disruption is modifiable, and therein lies the central clinical opportunity. [4]

This reorientation of microflora has profound implications for clinical practice, demanding a transition from reactive, procedure-based treatment

toward proactive, risk-informed management that accounts for each patient's unique microbiological, immunological, genetic, and behavioural profile. The emerging paradigm of personalised dentistry seeks to identify disease risk before clinical signs emerge, stratify patients according to biological vulnerability, and tailor interventions accordingly. Tools such as the Caries Management by Risk Assessment (CAMBRA) protocol and salivary diagnostics exemplify how microbiome science can be translated into actionable, individualised care. Preventive strategies, including fluoride, alkali-generating agents, probiotics, antimicrobial peptides, and responsible antibiotic stewardship, increasingly target ecological restoration rather than indiscriminate microbial elimination.

II. THE ORAL MICROBIOME: COMPOSITION AND ECOLOGY

The term "microbiome" refers to the group of microorganisms that exist in our bodies. An organism's genetic material is called its genome. It consists of all of the DNA's genes. The genome of all the bacteria that live in the oral cavity is called the oral microbiome. After the gut, it is the second-largest microbial colony in humans. [1] Bacteria can invade the hard and soft tissues of teeth as well as the oral mucosa.[5] Microorganisms can thrive in a rich habitat found in the teeth, tongue, cheeks, gingival sulcus, tonsils, hard palate, and soft palate.[2] The surfaces of the oral cavity are covered in plenty of microorganisms called "bacterial biofilm."[6]

Development of Oral Microbiome

The mouth cavity of an infant can often be sterile despite the considerable risk of contamination. When the mouth is regularly inoculated with germs beginning with the first feeding, the process of obtaining resident oral microflora begins.[7]

Saliva is the main way that the disease is spread, although it can also be passively transferred from the mother by bacteria in milk, water, and the surrounding environment. [7-9] Colonization begins at birth or shortly thereafter. The first invaders shortly after birth are the pioneer species, like Streptococcus salivarius. Aerobes, such as Streptococcus, Lactobacillus, Actinomyces, Neisseria, and Veillonella, have taken over the mouth cavity by the first year. These organisms can settle on the nonshedding surfaces once tooth eruption starts. More surfaces are made available for colonization once each tooth erupts. Gingival fissures develop to allow periodontal bacteria to colonize. For distinct microbial colonies to form, plaque deposition is observed at various locations on the tooth, including smooth surfaces, pits, and fissures. This process leads to the development of high species diversity and microbial succession. When all teeth are lost due to aging, the flora resembles that of a young person before tooth eruption. [10]

Composition of Oral Microbiome

The human microbiome is made up of a core microbiome and a variable microbiome. Each person has a unique variable microbiome, yet everyone has the same core microbiome. The main species found in various body locations under normal circumstances make up the core microbiome. The variable microbiome is unique to each individual and has developed in response to distinct lifestyle and genotypic factors.[11]

Bacterial Members

With 392 taxa having at least one reference genome and the total number of genomes in the oral cavity reaching 1,500, the oral cavity is one of the most researched microbiomes to date.[12] It contains over 700 different species of prokaryotes. These species are found in 12 phyla and 185 genera. [6] Firmicutes, Fusobacteria, Proteobacteria, Actinobacteria, Bacteroidetes, Chlamydiae, Chloroflexi, Spirochaetes, SR1, Synergistetes, Saccharibacteria (TM7), and Gracilibacteria (GN02) are the twelve phyla. [13]

The main genera of bacteria present in a healthy mouth cavity include the following:[7]

Gram-positive

Cocci: Abiotrophia, Pepto streptococcus, Streptococcus, Stomatococcus

Rods: Actinomyces, Bifidobacterium, Corynebacterium, Eubacterium, Lactobacillus, Propionibacterium, Pseudoramibacter, Rothia

Gram-negative

Cocci: Moraxella, Neisseria, Veillonella

Rods: Campylobacter, Capnocytophaga, Desulfobacter, Desulfovibrio, Eikenella, Fusobacterium, Hemophilus, Leptotrichia, Prevotella, Selemonas, Simonsiella, Treponema, Wolinella

Non-Bacterial Members

The oral cavity is home to plenty of microorganisms, including viruses, fungi, and protozoa. The two most prevalent protozoa in the oral cavity are Entamoeba gingivalis and Trichomonas tenax, which are primarily saprophytic. The most common type of fungus found in the oral cavity is Candida species. Ghannoum et al. identified 85 fungal species through culture-independent research on twenty healthy hosts. Candida, Cladosporium, Aureobasidium, Saccharomycetales, Aspergillus, Fusarium, and Cryptococcus were the primary species found.[14]

III. THE MICROBIOME SYMBIOSIS AND HOMEOSTASIS

Microbiome Symbiosis

In the oral cavity, microbiome symbiosis is defined as a state of dynamic equilibrium where the resident microbiota and the host immune system maintain a mutually beneficial relationship. [15]

This symbiotic state is an active process of homeostasis rather than just a passive coexistence, according to the "immunomicrobial pathogenesis" model [15]. Commensal microorganisms in a healthy environment send vital signals that adjust the host's innate and adaptive immune responses and stop potentially harmful species (pathobionts) from proliferating.[15]

Immune Modulation and Host Defence

At the dentogingival junction, physiological host-microbial interactions stop excessive inflammation from destroying periodontal connective tissues. [15] In chronic gingivitis, dysbiotic biofilms persistently activate innate immune receptors, which leads to epithelial hyperplasia. [15]

The production of interleukin-1β and tumour necrosis factor-α, two key mediators of periodontal tissue damage, is stimulated by microbial lipopolysaccharides.[16] In periodontal lesions, chronic cytokine release increases matrix metalloproteinase activity, which leads to collagen degradation.[15]

Inflammatory mucosal disorders of the oral cavity may be more likely if mucosal immune tolerance is disrupted. Xerostomic patients are more vulnerable to opportunistic infections like oral candidiasis due to decreased salivary immune factors.[17] Rapid loss of periodontal attachment is linked to heightened immune responses to subgingival biofilms.[15]

Metabolic Functions and pH Regulation in Hard and Soft Tissue Pathology

Subsurface enamel demineralization, a sign of early caries, is initiated by persistent plaque acidification below the critical pH of 5.[18] This state of altered homeostasis is further exacerbated in periodontal pockets, where proteolytic metabolism produces toxic metabolites that compromise the integrity of epithelial cells.[16] As repeated acid challenges accelerate the progression from enamel lesions to dentinal involvement, the ecological shift becomes more pronounced.[18]Anaerobes produce short-chain fatty acids that influence host immune responses in inflammatory periodontal tissues.[16] Reduced buffering capacity in hyposalivatory states increases the risk of mucosal atrophy and rampant caries.[19] Biofilm metabolic dysregulation causes halitosis by producing volatile sulfur compounds.[16]

Ecological Plaque Hypothesis in Oral Disease Pathogenesis

Both periodontal disease and dental caries are caused by an ecological imbalance in dental plaque. [3] Regular consumption of fermentable carbohydrates causes a cariogenic change in the composition of biofilms. [18] Increases in gingival crevicular fluid brought on by inflammation encourage the growth of proteolytic anaerobic species linked to periodontitis. [15]

Dysbiosis increases the expression of virulence, which includes immune evasion and protease synthesis. [4]

In order to maintain persistent periodontal inflammation and bone loss, keystone pathogens manipulate host immunity.[16] Alveolar bone resorption and progressive destruction of connective tissue occur when ecological balance is not restored.[15] Preventing pathological transformation of the oral microbiome still requires addressing environmental risk factors.[3]

IV. DYSBIOSIS: FROM ORAL HEALTH TO ORAL DISEASE

1. Dysbiosis: Definition and Mechanisms

Through a harmonious symbiotic relationship, the oral microbiota and the host coexist in a dynamic

equilibrium that preserves health. A disturbance of this equilibrium, known as dysbiosis, is symbolized by changes in the variety, composition, and relative abundance of microbial species. [20, 21] Oral disorders are not brought on by external infections, but rather by changes in the local microbiota, where typically harmless microorganisms become more prevalent and potentially harmful.[22]

After maturation, microbial communities in health are generally stable; nevertheless, physiological changes like aging, hormone changes, or environmental factors can upset this equilibrium.[22] In addition to microbial overgrowth, dysbiosis results in modifications to the structure and function of biofilms, creating an environment that promotes the development of disease.[20] The shift from a diversified microbial population to one dominated by fewer pathogenic species is a crucial characteristic.[21]

2. Risk Factors: Modifiable and Non-Modifiable

Both controllable and non-modifiable variables contribute to the development of dysbiosis. Poor dental hygiene, dietary habits, especially frequent sugar consumption, smoking, gum inflammation, and changes in saliva flow or makeup are all modifiable factors.[20, 22, 23] Pathogenic bacteria grow due to these factors changing the environment, which leads to lower pH, more food availability, and less oxygen.[22] Non-modifiable factors include age-related changes, hormonal changes, and genetic predisposition.[22] Additionally, systemic diseases like diabetes can alter the body's immune response, making dysbiosis more likely.[21, 22] The individual risk and progression of the disease depend on how these factors interact.

3. Dysbiosis and Dental Caries

Dysbiosis from environmental changes, especially frequent exposure to dietary carbohydrates, leads to dental caries.[22] Oral bacteria ferment glucose to produce organic acids. These acids lower the pH for a long time.[22, 23] This acidic environment disrupts the balance of the biofilm, promoting acid-loving and acid-tolerant bacteria.[22] Usually, salivary buffering and available minerals help with remineralization to balance out demineralization.[22] However, when acid production is higher than the ability to neutralize it, there is a net loss of minerals. This results in enamel demineralization and the formation of caries.[22] Therefore, caries is not just due to specific infections; it reflects a shift in microbial ecology.[20]

4. Dysbiosis and Periodontal Disease

Complex interactions between the host immune response and the dysbiotic microbiota mediate the transition from gingivitis to periodontitis. [21, 22] Gingival inflammation is caused by biofilm buildup, but tissue damage is caused by a dysregulated host response. [22]

Increased gingival crevicular fluid, bleeding, and decreased oxygen levels are all consequences of inflammation, which promote anaerobic and protein-dependent bacteria.[22] When the epithelium is micro-ulcerated, nutrients like iron are released, which promotes the proliferation of pathogens.[22] Altered microbial communities can influence host immune responses, resulting in persistent, non-resolving inflammation. [21, 22]

This creates a vicious cycle whereby microbial imbalance is encouraged by inflammation, and tissue damage and inflammation are further exacerbated by microbial alterations.[21] Disease progression is determined by individual susceptibility, which is controlled by systemic, environmental, and genetic factors.[22]

5. Dysbiosis and Peri-Implantitis

Peri-implantitis is caused by dysbiosis and shares pathogenic processes with periodontitis. [20, 22] While ecological changes promote the growth of anaerobic and pathogenic bacteria, biofilm formation around implant surfaces causes inflammation in surrounding tissues.[22]

A key factor in the development of disease is the interplay between host immune responses and microbial populations. [21] Peri-implant dysbiosis is further exacerbated by systemic disorders, smoking, poor oral hygiene, and implant-related parameters. [20, 22]

6. Oral Bacterial Proteins in Dysbiosis

Periodontal pathogens produce important virulence proteins that alter the balance of cytokines and affect gingival epithelial cells' (GECs') tendency to undergo apoptosis. The oral microbiome's signaling pathways, metabolism, cell interactions and oncogene regulation are all altered by co-infection with F. alocis and P. gingivalis. Osteoclast activation brought on by

dysbiosis increases alveolar bone resorption and tissue degradation via matrix metalloproteinases and inflammatory mediators. Changes in cytokine homeostasis are the driving force behind this process. Furthermore, apoptosis is mediated by suppression of MEK1/2 signaling and the extrinsic caspase-3 pathway. GECs generate proinflammatory cytokines like TNF-α, IL-1β, and IL-6, which are essential for causing apoptosis and maintaining inflammation.[24]

V. PERSONALIZED DENTISTRY: PRINCIPLES AND APPLICATIONS

Instead of using the same protocols for every patient, personalized dentistry is an emerging method that customizes preventive and therapeutic techniques based on each person's biological, behavioral, and environmental factors.[25] Early illness intervention, risk reduction, and tailored treatment strategies that enhance long-term results are the objectives.[26]

Conceptual Framework: From Population-Based to Individual Care

The majority of treatment decisions in traditional dentistry have been based on population-based disease models, which are informed by generalized epidemiological data.[27] Public health initiatives have greatly decreased the frequency of dental caries, but they fall short in addressing inter-individual differences in disease susceptibility.[28]

This paradigm is altered by personalized dentistry, which includes:[29]

- Genetic susceptibility
- Makeup of the oral microbiota
- Immunological response of the host
- Behavioral and lifestyle aspects
- Exposure to the environment

This methodology is consistent with the ideas of precision medicine, where clinical and multi-omic datasets are used to construct risk prediction models.[30] Personalized decision-making is further improved by the combination of digital health records and artificial intelligence.[31]

Biomarker Identification for Early Dysbiosis Detection

It is increasingly recognised that microbial dysbiosis, rather than the activity of a particular pathogenic microbe, is the cause of oral disorders such as dental caries and periodontal diseases. The term "dysbiosis" describes a disturbance of the oral microbiota's ecological balance, which leads to changed host-microbe interactions and heightened vulnerability to disease.

Biomarkers found in oral biofluids, including saliva and gingival crevicular fluid (GCF), which represent microbial activity and host inflammatory responses, can be used to diagnose dysbiosis early.

Microbial biomarkers:

Early enamel demineralization and an increased risk of caries are linked to elevated levels of cariogenic bacteria such Lactobacillus species and Streptococcus mutans. Similarly, a shift toward pathogenic anaerobic bacteria, which may be identified by microbiome profiling tools, is a characteristic of periodontal dysbiosis.

Inflammatory biomarkers:

In gingival crevicular fluid and saliva, inflammatory mediators including interleukin-1β (IL-1β) and tumour necrosis factor-α (TNF-α) are raised in periodontal pathology, indicating the host immune response to periodontal infection.In periodontal disease, these cytokines play a role in alveolar bone resorption, gingival inflammation, and connective tissue degradation.

Tissue breakdown biomarkers:

In the early stages of periodontitis, matrix metalloproteinases, especially MMP-8 and MMP-9, are significant biomarkers found in saliva and gingival crevicular fluid that show collagen degradation and periodontal connective tissue breakdown.

Functional biomarkers in saliva:

Changes in salivary flow, pH, and buffering capacity affect the oral microbial ecology and can be markers of microbial imbalance and tooth caries. It is now possible to identify microbial and host-derived biomarkers in oral biofluids before clinical symptoms become apparent, thanks to developments in next-generation sequencing, microbiome analysis, and proteomic technology. As a result, the examination of saliva and gingival crevicular fluid as diagnostic biofluids offers a non-invasive method for the early identification of dysbiosis and enables customised

treatment plans and preventive measures in oral disorders. [32-38]

Salivary Diagnostics as a Non-Invasive Screening Tool

Saliva's non-invasiveness, affordability, and convenience of collection have made it a valuable diagnostic biofluid. Both oral and systemic health are reflected in its DNA, RNA, proteins, enzymes, antibodies, and microbial components.

Salivary diagnostics are used in personalized dentistry for the following purposes:

- Bacterial load analysis for caries risk assessment.
- Monitoring periodontal disease using inflammatory markers.
- Identification of biomarkers for oral cancer and systemic diseases like diabetes.
- Testing for genetic susceptibility.

Chairside decision-making and early intervention techniques are made possible by point-of-care salivary testing equipment. [39-44]

Risk Classification and Caries Management by Risk Assessment

A solely restorative approach to caries therapy has increasingly given way to preventive measures that are based on an individual's risk of developing the condition.

To classify people into low-, moderate-, or high-risk categories for the development of disease, Caries Risk Assessment (CRA) methods examine biological, behavioural, and protective factors.

Three main elements are assessed by the Caries Management by Risk Assessment (CAMBRA) model: Disease signs include obvious cavitated lesions, caries-related restorations already in place, and prior caries experience, all of which are excellent predictors of future disease activity.

High levels of cariogenic bacteria, frequent consumption of fermentable carbohydrates, decreased salivary flow, and poor oral hygiene habits that promote biofilm accumulation are major risk factors.

Protective factors: To preserve oral microbial balance and prevent demineralisation, protective factors include frequent fluoride exposure, sufficient salivary flow, the use of fluoride toothpaste, and antibacterial measures.

In addition to causing dental caries, the buildup of pathogenic bacteria and dental biofilm also plays a major part in the development of periodontal disorders, where dysbiotic plaque causes gingival inflammation and periodontal tissue loss.A thorough oral risk assessment is crucial since shared risk factors, including poor oral hygiene, a high bacterial load, and decreased salivary protection, may enhance vulnerability to both dental caries and periodontal disease.By regulating biofilm and altering behavioural risk factors, CRA-based treatment may also help lower periodontal inflammation in patients with elevated microbial load and plaque retention. Personalised preventive and therapeutic approaches are advised based on the individual risk category, such as:

Topical fluoride treatment to prevent cariogenic microorganisms and promote remineralisation. antimicrobial medications to lower the oral biofilm's harmful bacterial burden. Dietary guidance to minimise acidogenic problems and restrict consumption of fermentable carbohydrates. To protect vulnerable tooth surfaces and encourage mineral recovery, use sealants and remineralising agents. Minimal intervention dentistry, which prioritises early identification, preventive care, and tooth structure preservation over major restorative procedures, is also supported by risk-based management techniques. [45-48]

Clinical Applications of Personalized Dentistry

Customized dentistry assists:

- Personalized intervals for preventive recall.
- Specific antibacterial treatment.
- Susceptibility evaluation based on genotype.
- Periodontal treatment guided by biomarkers.

These tactics improve long-term oral health results and increase patient engagement. [49-53]

VI. PREVENTIVE STRATEGIES INFORMED BY THE ORAL MICROBIOME

1. Oral Microbiome-Informed Preventive Techniques

The oral microbiome is a dynamic and intricate ecosystem made up of bacteria, fungi, viruses, and archaea that cohabit in a state of balance called eubiosis. When this equilibrium moves toward dysbiosis, favoring acidogenic and inflammatory-associated microbes, disease results. Today, preventive dentistry understands that ecological

changes in the microbial community, rather than a particular pathogen, are the cause of oral disorders such as periodontal disease and caries. Therefore, rather than eradicating all microorganisms, modern preventive measures concentrate on preserving ecological stability.[54]

Risk-based evaluation, salivary diagnostics, microbial profiling, and customized care planning are all part of microbiome-informed prevention. Clinicians can apply focused therapies that maintain helpful microorganisms while reducing detrimental species by comprehending the interplay between host immunity, saliva, nutrition, and biofilm structure.[55]

2. Ecological Methods for Preventing Illness

According to the ecological plaque hypothesis, disease arises when pathogenic bacteria outcompete helpful species due to environmental changes like increased sugar consumption or decreased salivary flow.[56] Important ecological tactics consist of: keeping the pH of the mouth neutral to stop the growth of acidogenic bacteria; limiting fermentable carbohydrates; promoting salivary flow for antibacterial and natural buffering effects; promoting biofilm stability rather than complete biofilm eradication; and encouraging microbial variety to promote ecosystem resilience and prevent inflammatory or acid-producing bacteria from taking over.[57]

3. Changes in Diet, Lifestyle, and Oral Hygiene

The oral microbiota is significantly shaped by diet. Frequent consumption of fermentable carbohydrates encourages the demineralization of enamel and the generation of acid. Salivary buffering capacity is supported by cutting back on sugar consumption, promoting fiber-rich meals, and staying hydrated. Immune response and microbial composition are influenced by lifestyle factors like smoking, stress, and systemic diseases. Periodontal and general dental health are positively impacted by quitting smoking and improving general health.[58] Maintaining microbial equilibrium still depends on effective mechanical plaque management. Using interdental cleaning techniques and brushing twice a day with fluoride toothpaste minimize the growth of harmful biofilms while maintaining commensal bacteria.[59]

4. Alkali-Generating Agents and Fluoride

Fluoride inhibits bacterial metabolism, increases enamel remineralization, and decreases demineralization. It encourages the production of fluorapatite, which is more resistant to breakdown in acid. Additionally, fluoride reduces the generation of acid by blocking cariogenic bacteria's metabolic pathways.[60] Dental plaque's pH equilibrium is maintained by alkali-generating substances like arginine. Ammonia is produced by commensal bacteria through the breakdown of arginine, which neutralizes plaque acids and supports ecological balance. Fluoride and arginine work better together to prevent dental cavities.[61]

5. Probiotics and Prebiotics in Dental Health

When given in sufficient quantities, probiotics are live microorganisms that offer health advantages. Certain strains of Lactobacillus and Streptococcus salivarius have shown promise in dentistry for reducing cariogenic bacteria and regulating inflammatory reactions.[62]

Substrates that specifically foster beneficial bacteria are known as prebiotics. Probiotics and prebiotics work together to stabilize biofilms and lower the risk of disease.[63]

6. Targeted Microbiome Modulation and Antimicrobial Peptides

The innate immune system's antimicrobial peptides (AMPs) specifically target harmful microbes. They minimize damage to host tissues while rupturing microbial membranes.[64]

Precision techniques that decrease pathogenic bacteria while maintaining beneficial species are used in targeted microbiome modification. This strategy lowers the danger of resistance and ecological damage by using bacteriocins, narrow-spectrum antibiotics, and biologically inspired treatments.[16]

Periotrap is a targeted antimicrobial peptide–based therapeutic approach designed to selectively eliminate harmful periodontal bacteria while preserving beneficial oral microorganisms. It mainly acts against Porphyromonas gingivalis, a key pathogen involved in the development and progression of periodontal disease. The peptide is engineered to bind specifically to receptors on the surface of P. gingivalis, allowing selective attachment to the pathogen. After binding, the antimicrobial peptide disrupts the bacterial cell membrane, leading to destruction of the targeted bacteria without significantly affecting the normal oral microbiota. This targeted approach helps control

periodontal infection, reduce inflammation, and restore microbial balance in the oral cavity. [61,41]
Stannous fluoride, triclosan, zinc compounds, and other antimicrobial agents are present in some toothpastes that help in reducing periodontal pathogens like P. gingivalis. These ingredients can disrupt bacterial membranes, inhibit virulence factors and reduce plaque biofilm associated with periodontal disease. (eg, Sensodyne Sensitivity & Gum Toothpaste, Colgate Total Advanced Health Toothpaste).[71]

7. Preventing Indiscriminate Use of Antibiotics
Antimicrobial resistance is a result of improper antibiotic use, which also disturbs the oral and systemic microbiome. By eradicating helpful commensal bacteria, broad-spectrum antibiotics can promote the growth of opportunistic infections.
Strict adherence to clinical guidelines, the use of local antibacterial medicines when necessary, and evidence-based prescribing are all key components of preventive dentistry. Antibiotic stewardship that is reasonable protects microbial diversity and slows the emergence of resistance.

VII. CONCLUSION

In conclusion, the evolving understanding of the oral microbiome compels a fundamental reorientation of dental practice. Clinicians who engage with this science are better positioned to move beyond disease management toward genuine health promotion providing care that is evidence-based, ecologically informed, and individually tailored. The oral microbiome is not a clinical adversary; it is a biological community whose equilibrium is the cornerstone of oral health, and whose understanding is central to the future of dentistry.

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