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Role of the Oral Core Microbiome in Oral Microbial Communities and Its Impact on Oral Health

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Abstract: The core microbiome within oral microbial communities plays a fundamental role in maintaining oral health. Recent studies have demonstrated that these core taxa exhibit remarkable stability in healthy individuals, with their composition being largely resistant to external factors such as season and gender. Early colonization by core microbiota not only contributes to the establishment of a healthy oral environment but also exerts synergistic functional effects that are crucial for effectively defending against the onset of oral diseases. However, the emergence of ecological dysbiosis disrupts this equilibrium, thereby leading to oral health problems. This review aims to explore the composition and functional roles of the core oral microbiome, assess the impact of dysbiosis on oral health, and summarize current research progress and future directions, with the goal of providing new perspectives for oral microbiome research.

1. Introduction

The oral microbial community comprises microorganisms inhabiting the oral environment, including bacteria, fungi, viruses, and other microbes. These microorganisms maintain oral health by forming complex ecosystems through interspecific interactions, influencing host immune responses, metabolism, and oral milieu stability [1]. Dysbiosis---an imbalance in the oral microbial community---is closely associated with various oral diseases, including dental caries, periodontal disease, and oral cancer [2]. Investigating oral microbiota composition and its impact on oral health carries substantial clinical significance.

The core microbiome is defined as microorganisms ubiquitously present in a given habitat that critically contribute to its ecological equilibrium. In the oral cavity, the core microbiome encompasses bacterial species commonly found in healthy individuals, which sustain the stability and functionality of the overall microbial community [3]. These core taxa exhibit notable compositional stability in healthy hosts, largely unaffected by seasonal changes or gender. Early colonization facilitates a healthy oral ecosystem and provides synergistic functional benefits key to resisting oral disease initiation. Nevertheless, dysbiosis disrupts this homeostasis, predisposing the host to oral pathologies.

Genera such as Streptococcus and Actinomyces occupy central positions within the oral microbial community; these taxa are integral to oral ecology and host immune modulation. Understanding the composition and function of the core microbiome is essential for preserving oral

health [16]. Elucidating the core microbiome can aid in identifying both pathogens and beneficial commensals, offering novel avenues for prevention and treatment [5]. Disruption of the core microbiome may contribute to disease onset, while targeted modulation of its composition holds promise for improving oral health outcomes. In-depth research into the core microbiome's role in oral microbial ecological balance enhances mechanistic understanding of oral health maintenance and provides a theoretical foundation for future clinical translation.

2. Main Body

2.1. Compositional Characteristics of the Core Microbiome

The core microbiome comprises multiple genera[17][36], among which Streptococcus and Neisseria play pivotal roles in maintaining ecological balance and disease development. Other genera, such as Fusobacterium and Prevotella, also modulate the oral microenvironment. Gemella, another core member, exhibits distinct site specialization across oral habitats, preferentially colonizing the buccal mucosa, tongue dorsum, throat, tonsils, and dental plaque.

2.1.1. Role of the Genus Streptococcus

Streptococcus is a genus of Gram-positive cocci, classified into multiple subspecies and species based on hemolytic activity, antigenicity, and biochemical reactions. Streptococci are categorized into alpha-hemolytic (e.g., S. mitis and S. pneumoniae), beta-hemolytic (e.g., S. pyogenes and S. agalactiae), and gamma-hemolytic (e.g., S. bovis) types.

As a major component of the oral microbial community, Streptococcus encompasses both pathogenic and non-pathogenic strains. S. mutans is the primary cariogenic pathogen, promoting caries through acid production that demineralizes tooth structures. Conversely, commensal streptococci such as S. sanguinis and S. gordonii inhibit pathogenic bacteria and contribute to protective biofilm formation, thereby supporting ecological homeostasis. The diversity and abundance of this genus are closely linked to oral health status, and its imbalance may trigger oral diseases.

2.1.1.1. Colonization and Interaction Mechanisms

As early colonizers, Streptococcus species use surface adhesins to bind to host surfaces and other microorganisms, promoting multispecies biofilm development and stability [11]. Oral polymicrobial community assembly and function are governed by complex interspecies interactions [51]. He et al. [6] demonstrated that Fusobacterium nucleatum adheres to streptococci such as S. sanguinis, enabling it to resist H_2O_2-mediated inhibition and integrate into multispecies communities. Jakubovics et al. [8] reported that streptococci employ adhesins, signaling peptides, bacteriocins, and metabolites like H_2O_2 and lactate to establish social networks within biofilms, maintaining community stability and regulating interspecies relationships. Kolenbrander [11] emphasized that interactions between streptococci and early colonizers like Actinomyces initiate biofilm formation, while fusobacteria act as "bridging organisms" promoting syntrophy between facultative and strict anaerobes.

2.1.1.2. Role of Hydrogen Peroxide

H_2O_2, a key streptococcal metabolite, confers a competitive advantage and acts as a signaling molecule regulating community structure. Zhu and Kreth [7] showed that S. sanguinis and S. gordonii inhibit competitors like S. mutans via H_2O_2 production and induce extracellular DNA

release, enhancing biofilm stability. Li et al. [13] found that celastrol activates pyruvate oxidase (SpxB), increasing H_2O_2 generation and suppressing S. mutans biofilm formation, lactic acid production, and insoluble glucan synthesis. Huang et al. [12] reported that mutanocyclin (MUC) promotes growth of health-associated streptococci and upregulates H_2O_2-related genes, enhancing their antagonism against S. mutans. Thus, H_2O_2 plays a critical role in both ecological balance and anticaries strategies.

2.1.1.3. Diversity and Caries Severity

Streptococcal community diversity is inversely associated with caries occurrence [4][14]. Children with severe early childhood caries (S-ECC) exhibited significantly lower streptococcal species richness ($N = 1.25 \pm 4.14$) and Shannon diversity index ($H' = 1.41 \pm 0.29$) compared to caries-free children (CF group: $N = 14.92 \pm 2.84$, $H' = 1.64 \pm 0.18$, $P < 0.05$), with higher detection of S. sanguinis and S. gordonii in the healthy group [14]. Low pH promotes S. mutans and lactobacilli while inhibiting acid-sensitive species like F. nucleatum and S. gordonii [10]. S. mutans, through acidogenicity and acid tolerance, fosters a cariogenic microenvironment [15]. Thus, reduced streptococcal diversity and cariogenic overgrowth are microbial signatures of caries.

2.1.1.4. Metabolites and Natural Products in Caries Prevention

Precise modulation of oral microbiota using metabolites or natural products is a growing research focus. d-Galactose inhibits S. mutans biofilm while promoting commensal streptococci [9]. Mutanocyclin suppresses S. mutans and enhances H_2O_2 production in health-associated streptococci [12], while celastrol augments H_2O_2 via SpxB activation, suppressing cariogenic activity [13]. These agents modulate microbial ecology through multi-target mechanisms. However, most studies use in vitro models that cannot fully replicate the oral environment. Causal relationships require validation through longitudinal clinical studies [14]. Future work should integrate multi-omics approaches to unravel microbe–host crosstalk and develop targeted ecological modulation strategies [15]. Comprehensive clinical safety and efficacy evaluations of natural products like celastrol remain urgently needed [13].

2.1.2. Functions of the Genus Neisseria

Neisseria is a significant component of the oral microbiome, playing critical roles in both oral health and disease pathogenesis. Its abundance and diversity differ markedly between healthy individuals and those with oral diseases: Neisseria abundance is generally higher in healthy subjects but significantly reduced in patients with dental caries or periodontitis [26]. Advances in genomics and high-throughput sequencing have elucidated the ecological characteristics of Neisseria and its disease associations, offering novel insights for oral health maintenance and disease prevention.

2.1.2.1. Inhibition of Pathogen Colonization

Neisseria species suppress pathogenic bacterial colonization through competitive exclusion and antimicrobial metabolite production. Certain Neisseria species produce antimicrobial substances that directly inhibit pathogens such as Porphyromonas gingivalis, thereby maintaining oral microbial homeostasis and reducing the risk of caries and periodontitis. Reduced Neisseria abundance is frequently associated with oral diseases, underscoring its protective role [18]. Additionally, Neisseria metabolites modulate oral pH and redox status, influencing other microorganisms. For instance, Neisseria generates short-chain fatty acids and nitrogen oxides that inhibit pathogens while promoting beneficial bacteria [18]. Its metabolic functions are essential for

maintaining oral microbial equilibrium, as its abundance is higher in healthy individuals but decreased in those with oral diseases.

2.1.2.2. Involvement in Biofilm Formation

Neisseria also participates in oral biofilm formation, enhancing biofilm stability and functionality through interactions with commensal bacteria. Biofilms formed by Neisseria with other oral bacteria, such as Streptococcus, increase antibiotic tolerance, making biofilm-embedded bacteria harder to eliminate. The biofilm-forming capacity of Neisseria is linked to its metabolite production, which promotes bacterial adhesion and growth, further augmenting biofilm complexity and stability.

2.1.2.3. Potential as a Biomarker

Neisseria abundance is reduced in acute pulpitis, chronic periodontitis [18], and oral cancer [20], and is lower in periodontitis patients than healthy controls.Multiple extrinsic factors modulate Neisseria abundance. Smoking (including e-cigarettes) reduces Neisseria while increasing Streptococcus and anaerobes [21][22], potentially via pH and redox alterations [23]. Antibiotics decrease Neisseria and may disrupt microbial balance [23]. Dietary factors also matter: high-sugar diets inhibit Neisseria [24], while nitrate and fiber intake enhance its abundance and improve oral microbiome health [25][26].Thus, Neisseria abundance monitoring may aid early diagnosis, and dietary modification—especially in early childhood—could promote oral health.

2.1.2.4. Role of High Genomic Variability

Neisseria possesses high genomic variability, enabling rapid adaptation via genomic rearrangements and horizontal gene transfer (HGT), enhancing competitive fitness. HGT facilitates antibiotic resistance acquisition via conjugation, transformation, and transduction [27]. Under antibiotic pressure, Neisseria can disseminate resistance genes through outer membrane vesicles, affecting both its own resistance and the commensal microbiota. Resistance gene abundance is higher in the oral environment than in other habitats, underscoring Neisseria's role in resistance spread. This variability confers a dual nature—ecological functions and pathogenic potential. Under normal conditions, Neisseria species exist as commensals, but under specific circumstances may transform into pathogens, with disruption of balance increasing disease risk [23].

Certain Neisseria species, such as N. mucosa and N. elongata, demonstrate probiotic potential by competing for epithelial colonization sites [28] and participating in nitrate reduction with cardiovascular benefits [29]. Neisseria abundance correlates negatively with early childhood caries, suggesting prevention potential [26]. However, whether these probiotic traits are directly associated with high genomic variability remains unclear.

2.1.3. Functions of the Genus Veillonella

Veillonella, an anaerobic Gram-negative coccus, is widely colonized in the oral cavity, gastrointestinal tract, and reproductive tract, and is generally considered a key component of the commensal microbiota. Changes in Veillonella abundance are associated with systemic diseases, including liver diseases, respiratory tract infections, and lung abscesses , suggesting its potential role in cross-domain microecological regulation.

Within the oral environment, Veillonella acts as an early colonizer and "bridging" species in interspecies interactions, promoting structural stability and functional integration of multispecies microbial communities [30][32]. Its ability to utilize lactate as a carbon source enables it to act as a

lactate scavenger, modulating local pH and influencing caries development [31]. Veillonella metabolites and cellular components may also modulate host immune responses, linking microbial community dynamics to oral mucosal health [33].

2.1.3.1. Morphology, Physiology, and Metabolism

Veillonella cells are spherical or short-chain, typically 0.3--0.5 μm in diameter. This strictly anaerobic bacterium lacks flagella, spores, and capsules. It utilizes short-chain organic acids (e.g., lactate) as energy sources. The genus comprises 16 known species, with eight (including V. atypica, V. parvula, and V. dispar) commonly found in the human oral microbiota [31]. Veillonella converts lactate into propionate and acetate via fermentation, participating in the ecological balance of the oral microbiome [31][30]. When S. mutans produces lactate, Veillonella converts it into short-chain fatty acids (SCFAs), reducing oral acidity and maintaining acid-base homeostasis [31]. Veillonella thus elevates oral pH and mitigates acidic erosion of tooth structures.

Veillonella also influences other bacteria through metabolite secretion, involving both symbiotic and competitive interactions. The symbiotic network formed by Veillonella in oral biofilms supports its own survival and provides adhesion sites for other pathogens, influencing oral health and disease progression [30].

2.1.3.2. Bridging Role and Opportunistic Potential

As a bridging species, Veillonella facilitates colonization of other bacteria, playing roles in periodontitis and dental caries [31][34]. Although generally considered a commensal, Veillonella may become opportunistic in immunocompromised individuals, causing bacteremia and lung abscesses. Increased Veillonella abundance correlates with disease severity in liver disease patients. Thus, its pathogenic potential warrants further investigation [31].

2.1.4. The Genus Actinomyces

2.1.4.1. Phylum and Taxonomy

Actinomyces (phylum Actinobacteria) includes A. israelii, A. naeslundii, and A. viscosus [35]. A. naeslundii promotes dental plaque formation and alters community stability [38]. A. israelii is associated with periodontitis and dental caries, suggesting a role in the health-disease transition [37]. Actinomyces cells are rod-shaped or branched Gram-positive bacteria; some strains form spores.

2.1.4.2. Main Roles and Characteristics

As a resident oral microbiota component, Actinomyces maintains microbial community stability and promotes biofilm formation. Its interactions with other bacteria contribute to oral homeostasis under healthy conditions, but it may become opportunistic when ecological balance is disrupted. A. naeslundii is common in healthy individuals, yet its role in plaque formation provides niches for subsequent pathogen colonization. Conversely, A. israelii may shift from commensal to pro-inflammatory factor in immunocompromised hosts or altered microenvironments, leading to oral tissue damage [37][38]. This dual role underscores its significance in oral health research.

2.1.4.3. Ecological Dysbiosis and Disease Associations

Oral microbial diversity and stability are essential for health. Dysbiosis—triggered by poor hygiene, imbalanced diet, or smoking—increases pathogenic bacteria like Fusobacterium and Porphyromonas while beneficial taxa such as Actinobacteria decline [38]. In periodontitis,

Actinomyces levels are elevated in saliva and gingival crevicular fluid, especially with bleeding, suggesting that Actinomyces metabolites modulate host immune responses and exacerbate local inflammation and tissue destruction [37][38]. Actinomyces abundance changes are also linked to diabetes and hematological malignancies, where Actinomyces species predominate in the oral microbiome [42], highlighting its bridging role in oral-systemic health.

Actinomyces metabolites exert dual immune effects. SCFAs (e.g., butyrate) promote anti-inflammatory responses via immune receptors, protecting the host. However, reduced Actinomyces abundance may disrupt immune balance, increasing infection and inflammation risk [38]. This supports targeting Actinomyces abundance for periodontal therapy. Oral microbiome alterations in craniofacial microsomia further underscore microbial sensitivity to diverse pathologies.

2.1.5. The Genus Rothia

2.1.5.1. Classification and Ecological Niche

Rothia (Actinobacteria) includes R. mucilaginosa and R. dentocariosa, with R. mucilaginosa most common in the oral cavity. Rothia utilizes multiple carbon sources and forms biofilms, participating in ecological networks that stabilize the oral microbiome. Its abundance is higher under healthy conditions but declines in periodontitis and dental caries. Rothia has been associated with cardiovascular health, and its fluctuations may influence systemic status [46][47]. Inhaled glucocorticoids may influence Rothia abundance and contribute to oral dysbiosis [41].

2.1.5.2. Biological Characteristics and Role in Oral Health

Rothia are Gram-positive bacteria with multi-layered peptidoglycan cell walls, conferring stress resistance. They utilize sugars, amino acids, and fatty acids, participating in nutrient metabolism and community balance through symbiosis, antagonism, and competition.

Rothia inhibits pathogens via antimicrobial substances and SCFAs that modulate immune responses. Its cell wall components may interfere with biofilm formation. Rothia abundance correlates positively with oral health—higher levels associate with lower caries and periodontal disease. It also regulates oral pH and promotes salivary secretion. Maintaining appropriate Rothia abundance is crucial for oral homeostasis and may represent a preventive strategy.

2.1.6. Impacts of Other Associated Microorganisms

Fusobacterium nucleatum is an important pathogenic bacterium associated with periodontitis and oral cancer [48], with Mendelian randomization studies substantiating the causal link between oral microbiota dysbiosis and oral cancer [44][45]. Prevotella plays a significant role in maintaining oral health and promoting immune responses [49][48].

2.1.6.1. Characteristics and Functions of Fusobacterium

Fusobacterium (F. nucleatum, F. polymorphum, F. periodonticum) is a key oral microbiota component, with abundance elevated in gingivitis, periodontal disease, and colorectal cancer [52]. F. nucleatum acts as a "bridging microorganism" facilitating pathogen colonization and biofilm formation in oral and intestinal niches, influencing immunity and metabolism [53]. It promotes biofilm maturation via coaggregation, enhancing antibiotic resistance and immune evasion [54][55]. Its synergy with P. gingivalis augments biofilm formation and pathogenicity [56]. Metabolically, Fusobacterium produces SCFAs, hydrogen sulfide, and amino acids that modulate gut microbes, influence immune responses, and promote tumorigenesis [19][52][72]. F. nucleatum metabolites

promote colorectal cancer cell proliferation and reshape the tumor microenvironment [53]. Fusobacterium thus transitions from commensal to pathogen under dysbiotic conditions [53].

2.1.6.2. Characteristics and Functions of Prevotella

Prevotella is a diverse anaerobic genus with over 50 species, distributed in the oral cavity and intestinal tract, forming complex symbiotic relationships. P. intermedia and P. melaninogenica are associated with periodontitis and dental caries while also occupying important positions in the healthy oral microbiome [57]. Prevotella metabolizes complex carbohydrates like plant polysaccharides [58]. It influences oral pH and regulates other microorganisms' growth ; interactions with Fusobacterium and Porphyromonas may drive disease progression during dysbiosis [59]. Elevated Prevotella levels increase periodontitis risk [60]. Prevotella produces SCFAs (acetate, butyrate) from dietary fiber, supporting barrier function and exerting anti-inflammatory effects, while its metabolites modulate immune responses and chronic disease risk [59]. Prevotella metabolism is linked to diabetes and obesity [60].

2.1.6.3. Interactions and Disease Associations

Fusobacterium and Prevotella exhibit a dynamic coexistence-competition relationship. F. nucleatum becomes prominent in periodontal disease , while Prevotella changes correlate with disease onset. Resource competition affects their survival and proliferation [61]. F. nucleatum enhances biofilm stability and antimicrobial resistance, facilitating synergistic colonization [48]; Prevotella participates in biofilm formation, and its co-action with F. nucleatum promotes colonization capacity, associated with more severe periodontitis and dental caries [62]. This interaction promotes P. gingivalis colonization, driving dysbiosis [48][62]. In chronic inflammation, both secrete pro-inflammatory cytokines and activate NF-κB and MAPK pathways [48]. Prevotella changes link to metabolic syndrome and autoimmune inflammation [63]. Clinical evidence shows elevated F. nucleatum and P. intermedia in periodontitis patients, correlating with bleeding and probing depth [48][64]. High F. nucleatum associates with cardiovascular disease and diabetes [48]. Both genera contribute to periodontal damage through biofilm formation, immune pathway activation (TLR--NF-κB/MAPK)[50], and tissue-destructive enzymes [48]. This interaction network mediates the transition from stability to dysbiosis, with abundance changes reflecting local and systemic inflammatory risk [48][64].

2.2. Core Microbiota: Early-Life Core Microbiota Shaping Long-Term Microbiome

Early-life core microbiota colonization critically shapes an individual's microbiome. Within hours after birth, Streptococcus and Lactobacillus appear in the oral cavity, establishing the foundation for community assembly [65]. As individuals age, oral microbiome diversity and abundance increase. Colonization is influenced by genetics, maternal health, feeding practices, and antibiotic use [66]. Maternal oral health, delivery mode, feeding habits, and antibiotic exposure affect infant oral microbiome [40][68][73] and fungal colonization [71]. Microorganisms from maternal saliva and breast milk transmit via direct contact or vertical transmission [69]. Hygiene, diet, and socioeconomic status also influence colonization. High similarity between maternal and infant oral microbiomes underscores the mother's importance [67]. Early-life core microbiota composition has profound implications for future oral and systemic health, particularly immune development and oral disease onset [68].

Early colonizers maintain ecological balance by occupying niches and inhibiting pathogen colonization through resource and space competition [67]. Veillonella metabolizes lactate, elevating oral pH and inhibiting pathogen growth in acidic environments. Through niche occupation and pH

regulation, early core microbiota establish a relatively stable oral microecology, reducing disease risk.

2.3. Research Methods and Challenges in Core Microbiota Studies

2.3.1. Value of Genomics and Metabolomics

Genomics and metabolomics have advanced core microbiota research by revealing genetic characteristics, adaptive mechanisms, and metabolite changes reflecting physiological status and ecological functions [67]. Whole-genome sequencing identifies oral-health-associated genes and microbial interactions; metabolomics monitors metabolite fluctuations, aiding understanding of microbial roles [69]. Integrating multi-omics (e.g., transcriptomics, metabolomics) provides comprehensive insights into microbe-host interactions [75]. These tools support microbiome-modulation strategies and personalized oral health management.

2.3.2. Challenges in Core Microbiota Research

Core microbiota research faces several challenges. Insufficient sample size reduces statistical power and conclusion reliability [72]. Individual variability—including genetics, diet, and lifestyle—influences microbiome composition, complicating generalizability. Limited interdisciplinary collaboration restricts research depth; integrating oral medicine, microbiology, genomics, and metabolomics expertise is essential for understanding core microbiota's role and translational applications [67]. Establishing multidisciplinary platforms will advance research and clinical translation.

2.4. Experimental Modulation Strategies for Core Microbiota

2.4.1. Effects of Probiotics and Dietary Modulation

Probiotics and dietary modulation regulate the core microbiota. In early life, breast milk probiotics promote beneficial bacteria growth while inhibiting pathogen colonization [66]. Dietary sugar intake is closely linked to oral microbiome composition: excessive sugar promotes cariogenic bacteria like S. mutans, whereas a healthy diet maintains microbial balance [70]. Adjusting dietary patterns to increase fiber and prebiotic intake promotes beneficial bacteria and improves oral health. Thus, combining probiotic supplementation with healthy dietary strategies represents an effective intervention for oral microbiome modulation.

2.4.2. Potential Applications of Chemical Agents and Local Interventions

Chemical agents and local interventions show potential for modulating oral core microbiota. Graphene nanomaterials inhibit oral pathogens while promoting beneficial bacteria [67]. Topical antimicrobials (chlorhexidine, fluoride) reduce dental plaque and gingivitis [74]. However, long-term effects require safety and efficacy evaluation. Integrating chemical agents with microbiome research offers novel directions for oral health. Orthodontic treatment also alters oral microbiome and metabolome composition [43].

3. Conclusion

Oral microbiome research has increasingly demonstrated the critical role of core microbiota in maintaining oral health. These communities stabilize the oral ecosystem, influence host immune responses, and affect nutrient absorption [39]. Dysbiosis—reduced bacterial diversity, pathogen

proliferation, and inflammatory activation—increases risks of caries and periodontitis and may link to systemic diseases, underscoring the importance of core microbiota maintenance.

Future research should continue exploring core microbiota functions and therapeutic applications. Genomics and metabolomics can elucidate microbial interactions and dysbiosis mechanisms, offering new prevention and treatment insights. Intervention strategies targeting dysbiosis—probiotics, dietary modulation, and other measures—warrant further investigation.

Findings from different studies must be integrated cautiously, as variations in sample selection, experimental design, and analytical methods may yield divergent results. Unified research standards and technical platforms will strengthen both basic research and clinical translation. Core microbiota are indispensable for oral health; maintaining their ecological stability is essential for disease prevention and overall well-being.

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