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EDITORIAL

The Microbiology of Dental Caries and Periodontitis: Current Insights and Clinical Implications

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Dental caries and periodontitis are among the most prevalent diseases worldwide and, as such, are the most commonly encountered conditions in dental practice. Both diseases are microbial in nature-a fact recognized as early as the late 19th century (1,2). Our understanding of their microbiology has dramatically evolved since then, accelerating significantly over the last two decades, albeit with limited clinical impact. This editorial provides an overview of recent developments in the microbiology of dental caries and periodontitis, highlighting their potential to revolutionize dental care.

Work done throughout the 20th century, which was dominated by culture-based techniques, led to the identification of key cariogenic and periodontal pathogens. These include *Streptococcus mutans* and *Lactobacilli* in dental caries, and *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola* (later referred to as the 'red complex') in periodontitis. These discoveries were incorporated into the ‘specific-plaque hypothesis,’ which proposed that both diseases are caused by specific infections (2). In 1994, Philip Marsh introduced his famous 'ecological plaque hypothesis,' proposing that dental diseases result from microbial community imbalances driven by ecological pressures that favor the overgrowth of putative pathogens (3)-a groundbreaking concept ahead of its time that closely aligns with our current view of the etiology of dental diseases.

The past two decades have witnessed significant conceptual breakthroughs that have transformed our understanding of the role of microbial communities in health and disease. These breakthroughs have been driven by technological advancements, particularly the advent of next-generation sequencing (NGS) technologies, which have enabled a more comprehensive and detailed characterization of microbial communities and their genetic content (the microbiome). The microbiome has become a major area of research, revealing that microbial community imbalances (microbial dysbiosis), particularly in the gut, play a key role in various diseases, including diabetes, cardiovascular disease, cancer, autoimmune disorders, obesity, inflammatory bowel disease (IBD), neurodegenerative diseases, and even mental health conditions such as depression and anxiety (4,5).

Applying these advancements to the study of oral microbial communities (now known as the oral microbiome) has identified complex microbial shifts and interactions involving dozens of species associated with dental caries or periodontitis. This provides a more detailed version of the ecological plaque hypothesis, with greater emphasis on microbial community imbalance (dysbiosis) than on specific putative pathogens (2). In the case of dental caries, studies have revealed that dysbiotic interactions can also involve non-bacterial species, such as *Candida albicans*, leading to the concept of interkingdom interactions in oral diseases (6). In periodontitis, research has revealed that there is a bidirectional loop between the microbiome and immune response, whereby early microbial dysbiosis triggers periodontal inflammation, which in turn, drives further dysbiosis (7). As such, periodontitis and dental caries are likely the best examples of dysbiosis-induced diseases.

Such progress in our understanding of the microbiology of dental caries and periodontitis has, however, seen little clinical translation, as the management of these two diseases continues to primarily rely on non-specific mechanical or chemical elimination of microbes at both the personal and professional care levels. Although effective, this standard-of-care approach carries with it a major drawback: collateral damage to commensal, potentially beneficial microbes. Earlier views of dental diseases as specific infections may have been difficult to translate into clinical practice, as targeting specific pathogens in dental plaque is challenging, and efforts to produce vaccines against putative pathogens has been met with limited success.

Fortunately, the current view of dental caries and periodontitis as dysbiosis-induced diseases seems to open novel avenues for prevention and treatment. One emerging concept is microbiome modulation, where agents such as prebiotics, probiotics, synbiotics, and natural products can be used to reverse dysbiosis and maintain a balanced, healthy microbiome (eubiosis or normobiosis), at least as an adjuvant to current management strategies. These agents can be applied through personal oral healthcare products or specialized professional treatments. An additional potential approach to manipulating the oral microbiome is microbiome transplantation, i.e., the transfer of a healthy microbiome from a donor to a patient. The National Institute of Dental and Craniofacial Research recently launched an initiative to stimulate research on these approaches (8).

Another evolving clinical application is the use of microbiome signatures as diagnostic aids to complement clinical and radiographic examinations, for example for patient risk stratification, tailoring treatment plans, predicting prognosis, and assessing treatment response (9). While microbiome data are technical and challenging to interpret, the application of artificial intelligence has enabled researchers to transform them into intuitive, clinically relevant indices of dysbiosis with high diagnostic accuracy, which can be used to personalize dental care based on a patient's level of dysbiosis. An example of this is the subgingival microbial dysbiosis index (SMDI) developed by our group for periodontitis (10).

Ongoing research into the oral microbiome is likely to deepen our understanding of the etiology of oral diseases and uncover novel approaches that could revolutionize the prevention and treatment of dental caries and periodontitis.

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