

Guest editorial

Human oral microbiology is at a crossroads. Recent technological advances built upon a rich legacy of phylogenetic studies have brought us to the realization that nearly all oral bacterial diseases are polymicrobial in origin. The next steps will require the determination of the exact composition of the oral microbiota in states of health and disease, the physiologic interactions among members of this diverse community, and the host response to microbial challenge. Oral microbiologists are fortunate in that they can draw upon decades of studies defining the physiology and genetics of oral bacteria in monoculture, basic immunological markers of oral infection and inflammation, and rudimentary animal models that demonstrate Koch's postulates. The time has come to integrate these studies and translate this knowledge base for improved clinical success.

Recent advances in nucleic acid sequencing and computational technologies will allow us to determine the exact composition of the oral microbiota and central to these studies will be the identification and characterization of the uncultivated species. We are poised to settle the Great Plate Count Anomaly with respect to dental plaque but more importantly we can finally begin to determine the relevance of these unique species to oral health and disease. Likewise, the ultimate promise of oral metagenomics is to expand and refine culture-dependent phylogenies and provide diagnostic signatures that could benefit both the clinician monitoring disease progression and seeking new treatment options, and the basic researcher interested in microbial population dynamics; both of which are critical to our patients.

Understanding physiological interactions among microbes and their environment is the essence of microbial ecology and the oral microbiota is one of the best systems available for studying these interactions. Aside from ease of sampling, the oral cavity is the best characterized of all colonized body sites and provides several unique habitats, each with their local microbiotas and associated physiologies and pathologies. In these habitats you can find the entire universe of microbial interactions: coaggregation, biofilm formation, quorum-sensing, metabolic cooperativity, antibiosis, and horizontal gene exchange. However, a key question that remains is what defines a commensal and what defines a pathogen? In some situations they can be interchangeable.

The third point concerns host response and inflammation. Periodontal disease is an inflammatory process initiated by microbial challenge and sustained by an altered or deregulated host response. Much has been learned regarding the molecular mechanisms of pathogenesis by single organisms both *in vitro* and in animal models but key questions remain, specifically on the role of pathogenic consortia during the initiation and progression phases of oral disease and the role of the innate immune system in maintaining health and modulation of disease states.

We are now in possession of some of the most powerful research tools ever known to science and it is no longer a question of whether we can adequately investigate a particular problem but how do we ask the right questions and design the right experiments. Microbiology has accelerated into a new era of discovery. It is a great time to be a molecular oral microbiologist!

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