Chapter 15

A Comprehensive Evaluation of the Virulence of Oral Flora

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ABSTRACT

The oral cavity is inhabited by more than 700 species of commensal bacteria. Several have been identified as pathogens of oral diseases, dental caries, and periodontal diseases. However, the lack of information concerning the interaction between pathogens and the remaining commensal bacteria has made it difficult to understand the precise etiologies of oral diseases. We need to identify the comprehensive species found in individual oral flora and compare these results with corresponding oral health conditions. In this chapter, the authors delineate the previous attempts to identify oral commensal bacterial flora and discuss the potential capability of modern molecular genetic technologies such as terminal restriction fragment length polymorphism, DNA microarray and pyrosequencing analyses utilizing bioinformatics.

INTRODUCTION

Oral Diseases and Microbes

Two major oral diseases, dental caries and periodontitis, occur at different rates among countries (Petersen, 2003). Dental caries is most widespread in several Asian and Latin American countries but is less common in most African countries. Although developed countries have shown a marked decrease in the prevalence of dental caries in children over the past decades, dental caries is still a major oral health problem in most industrialized countries, affecting 60–90% of schoolchildren and most adults. It is rare to find a person from a developed country who has not experienced dental caries. It is the most common chronic disease affecting children, including those in the United States, and is five times more common than the second most prevalent chronic disease, asthma (American Academy of Pediatrics, 2009). Periodontal diseases are also highly prevalent and affect up to 90% of the world’s population. Most
children exhibit signs of gingivitis (the mildest form of periodontal disease), redness, or swelling of gum tissue and bleeding from the gingival sulcus. Persons generally experience the initial stages of periodontal diseases as adults, and the likelihood of moderate or severe periodontitis, which has been reported in 13% of the United States population, increases with age (Albandar et al., 1997). Periodontitis may also be a main cause of tooth loss. Juvenile or early-onset aggressive periodontitis, which affects about 2% of youth, can lead to premature tooth loss (Albandar et al., 1997).

The etiologies of both oral diseases have been extensively pursued for more than 100 years. These diseases are believed to be infectious diseases caused by several specific oral bacterial species, although some features show signs of a multifactorial disease. W. D. Miller, an American dentist, visited Koch’s laboratory to seek the specific pathogens of oral diseases. However, he concluded in his publication “The Micro-organisms of the Human Mouth” (1890) that no single species of microorganism caused dental caries, but rather that the process (a chemo-parasitic process) was mediated by a microorganism capable of producing acid and digesting protein. His concept conformed to the modern interpretation of the dental caries etiology based on the mutual interaction of multi-bacterial species. However, many successors continued to chase the actual culprits among the acid-producing candidates in accordance with Koch’s postulate and Miller’s chemo-parasitic theory. Streptococcus mutans, which is presently being reclassified into seven detailed species (mutans, sobrinus, cricetus, rattus, ferus, macacae, and downei) known as the “mutans streptococci” group (Bratthall, 1970; Ota et al., 2006), was the favored candidate during the mid-1900s. Animal experiments clearly demonstrated the dental caries etiology of S. mutans (Loesche, 1986). Much epidemiological research confirmed the etiology based on the S. mutans story; however, the subjects in these studies were biased to infants with deciduous teeth or young subjects with permanent teeth immediately after tooth eruption. As far as we know, few epidemiological studies support that S. mutans causes dental caries in adults. Of interest is that a recent study with restricted adult subjects reported contrasting results to the S. mutans theory (Nishikawara et al., 2006). Hence, bacteriological etiology has not yet been established.

The bacteriological etiology of periodontal diseases is much more complicated than that of dental caries because of the complex modification of the former by multiple host immune-inflammatory responses. The host’s response should be protective, but hypo- and hyper-responsiveness can lead to the destruction of periodontal tissues. Studies have been dedicated to understanding the relationship between periodontal diseases and microflora using conventional modern molecular techniques. The putative anaerobic pathogens, such as Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola, were frequently detected in the subgingival sites with periodontitis. These bacterial species release proteolytic enzymes that can damage host tissue and produce various putative virulence factors. However, it is difficult to declare these particular bacterial species as the causative species or as being able to adapt to the anaerobic conditions of the deep pocket in the periodontal lesion. One of the reasons that it is difficult to ascertain the exact periodontal pathogen is because the field lacks an appropriate animal model that can establish the relationship between bacterial infection and periodontitis.

**Problems in Oral Microbiology**

Should we pursue the pathogen directly responsible for oral diseases without considering the more than 700 bacterial species inhabiting the oral cavity? There must be an interaction among or between pathogens, and possibly the surrounding non-virulent bacterial species, that can regulate the expression of virulence factors from pathogens. We may also have to consider the virulence of oral
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