Diagnostic and Prognostic Tests for Oral Diseases: Practical Applications

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Abstract: Dental caries and periodontitis are initiated by specific bacteria and modified by host and environmental factors. Individuals have great differences in their rates of disease progression, but a small set of risk factors, such as smoking and diabetes, can distinguish patients at high risk for more severe disease. The application of information about factors that influence disease can be used to improve disease prevention and management. Knowledge of when specific information may be valuable should lead to the optimal management of individual patients. The use of diagnostic and prognostic tests and their application to the assessment and management of dental caries and periodontitis are the focus of this review.

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Disclaimer: Dr. Kornman is a full-time employee and shareholder of Interleukin Genetics, Inc., which develops and markets genetic susceptibility tests for common diseases, including periodontitis, osteoporosis, coronary artery disease, Alzheimer’s disease, and complications of diabetes. Interleukin Genetics has patents issued and pending on cytokine genetic markers, including polymorphisms in the genes for IL-1 and TNF, and the susceptibility to various diseases and the use of those genetic factors for preventive and therapeutic purposes.

Key words: periodontal disease, dental caries, risk factors, diagnostic tests, prognostic tests, interleukin-1, genetics

The ultimate goal of dental care is to assist in the lifelong maintenance of a dentition that is functional, comfortable, and esthetic. In addition to public health and education efforts, much of dental care is focused on effective and efficient preventive and therapeutic management of the major clinical diseases: dental caries and periodontitis. Both of these diseases are initiated by specific bacteria, and the diseases are modified by host and environmental factors. Although the disease-associated oral microbial ecologies are complex, substantial data indicate that the primary bacterial strains associated with dental caries are *Streptococcus mutans* and *Streptococcus sobrinus* and that *Porphyromonas gingivalis, Bacteroides forsythus, Actinobacillus actinomycetemcomitans*, and certain spirochetes are associated with chronic periodontitis in adults.

During the 1970s and 1980s, extensive animal and human clinical studies demonstrated that regular control of tooth-adherent bacterial plaque prevented both dental caries and periodontal disease. During this period, periodontitis was considered to be a simple chronic disease with a single dominant causative factor: bacterial plaque. Periodontitis in adults was considered to be a disease that always followed gingivitis. If gingivitis was not treated, it progressed slowly and affected most individuals equally.

Years of research have shown that specific bacteria in the plaque are essential for initiation and progression of periodontitis. In addition, the severity of periodontitis, rate of its progression, and response to therapy are determined by an individual’s biological responses to bacterial challenge and an individual’s risk factors. The risk of dental disease differs among individuals.

There are several clinical implications of these new concepts. One implication is that many cases of untreated gingivitis do not progress to periodontitis. We now know that individuals have great differences in their rates of disease progression and a small set of risk factors can distinguish patients at high risk for more severe disease and less treatment responsiveness. The complex combination of factors that influence clinical disease presentation provides several practical opportunities for diagnostic and prognostic tests. In addition, technological advances have introduced new opportunities for refining the clinical assessment of dental diseases.

This review will consider the current knowledge of the biology of dental caries and chronic periodontitis in the development of new diagnostic and prognostic tests. The use of these tests and their application to the diagnosis, prognosis, and management of these major dental diseases will be discussed.
Overall Clinical Objectives of Diagnostic Tests and Relevant Terminology

Diagnostic tests may serve multiple clinical objectives that benefit the individual patient. The clinician may use tests to: a) identify predisposing risk factors to modify risk and prevent disease; b) identify early disease-associated biochemical or physical changes prior to clinical signs of disease to halt the changes and reverse damage prior to loss of function; and c) determine which specific type of disease is involved to guide selection of the most effective therapy.

The goal of dental care is to provide effective and efficient preventive and therapeutic management of dental caries and periodontitis. Achievement of this goal requires targeted approaches to patient management and patient-specific diagnostics. The most efficient and best patient-centered outcomes are through prevention targeted at an individual based on his or her specific needs. This will prevent disease at a level that is meaningful to the patient.

The term “diagnosis” is used in medicine to describe the determination of the nature of a disease. A “diagnostic” refers to tools, procedures, or technologies that are used in that determination. A diagnostic test or technology is used to identify elements of an existing disease process. Diagnostic tests differentiate whether or not a person has a specific disease at the time.

The accuracy of a diagnostic test in detecting a specific disease element is calculated using certain criteria. These criteria are based on whether the disease element does or does not exist at the time of the diagnosis. The accuracy of the diagnostic test is calculated when the specific disease element is present and when it is not present. Various diagnostic test performance criteria have been established, including sensitivity, specificity, and predictive values. The application of performance criteria to the clinical use of medical diagnostic tests has been extensively described.

A “prognosis” is the prediction of the future course and/or outcome of a disease or a disease treatment, given current information. Because there are no facts about the future, a prognosis is described in terms of probabilities. Prognostic tools or technologies are used to make the prediction. The strength of a specific prognostic test is often described in terms of the relative risk for a future event when the test parameter is present compared to when it is not present. Studies on the value of prognostic tests provide a confidence interval that gives the user some indication of how useful the test may be in a broad population.

“Risk markers” are biologic markers that either a) indicate disease or disease progression but are not causal or b) represent historical evidence of the disease. “Risk factors” are characteristics of the person or environment that, when present, directly result in an increased likelihood that a person will get a disease and, when absent, directly result in a decreased likelihood of disease. Because risk factors are involved in the cause of the oral diseases, modification of risk factors can reduce risk and, perhaps, decrease the disease.

Clinical Application

Our current knowledge of dental caries and periodontal disease has changed the way patients are evaluated and treated. When these diseases were believed to be a simple function of the amount of dental plaque and the same process in all patients, prevention involved only plaque control, and treatment consisted of repairing tissue damage. In this situation, patients were assessed based on existing disease, such as the severity and extent of existing caries, previous restorations, and the severity and extent of periodontal disease. Using this approach, the levels of existing and past disease were considered the best determinants of the treatment plan.

Now that we understand the causes and progression of dental caries and periodontitis, an evaluation and treatment plan can be individualized for each patient. This can be accomplished by performing an examination, making an initial clinical diagnosis, identifying the risk factors for future progression of the disease, establishing a prognosis, presenting treatment alternatives to the patient, obtaining informed consent, and then treating the patient. A major part of this process is the assessment of the current level of disease and the risk for future disease (Figure 1a). Past disease history is still a strong predictor of future disease, but only if the patient is in a later stage of the experience. For example, if a patient is forty-five years old and has had severe generalized periodontitis, there are data that indicate this patient is at increased risk for future disease. However, if the patient is thirty-seven years old with
early generalized periodontitis and has a few localized sites of moderate disease, the case could follow several different paths in the future. The risk for future disease is evaluated for factors such as home care to control bacteria, genetic susceptibility, smoking, and the presence of systemic diseases such as diabetes (Figure 1b).

Types of Diagnostic and Prognostic Information

Diagnoses are determined by analyzing the information obtained through the oral exam. The information routinely collected during this exam includes demographic data such as age and gender, medical history, history of current and past disease, radiographic findings, and clinical features or observations. The specific types of diagnostic and prognostic information relevant to dental caries and periodontal disease are described here.

Chronic periodontitis is usually not detectable until after age thirty-five, and it affects approximately 30 percent of adults in the United States and Europe. In contrast, approximately 20 percent of children between the ages of two and four years have dental caries, and by seventeen years almost 80 percent of young people have had a cavity. In addition, more than 66 percent of adults ages thirty-five to forty-four years have lost one permanent tooth to dental caries, and older adults suffer from root caries.

A diagnosis of periodontal disease is made after analyzing all of the information collected from a periodontal examination. This information includes 1) the presence or absence of clinical signs of inflammation such as bleeding upon probing, 2) probing depths, 3) extent and pattern of loss of clinical attachment and bone, 4) patient’s medical and dental histories, and 5) presence or absence of other signs or symptoms including pain, ulceration, and amount of observable plaque and calculus. Additional information on clinical assessment is beyond the scope of this review.

Periodontal disease is the result of a complex interplay among bacterial challenge, host response, and other modifying factors. Modifying factors can be divided into two categories: environmental and internal modifying factors. Environmental factors are habits and behaviors that can be changed, while...
internal factors are endogenous to the individual. Examples of environmental factors are smoking and irregular dental care. Examples of internal factors are genetic factors and systemic disease. This section will investigate some of these factors.

**Bacterial Challenge**

Periodontal disease is caused by bacteria within the dental plaque. If there are no bacteria, there is no disease.

The presence of plaque deposits constitutes a significant risk for periodontal disease. Plaque-induced periodontal disease is classified into seven categories: 1) gingivitis, 2) chronic periodontitis, 3) aggressive periodontitis, 4) periodontitis as a manifestation of systemic diseases, 5) necrotizing periodontal diseases, 6) abscesses of the periodontium, and 7) periodontitis associated with endodontic lesions. Studies have shown that clinical plaque levels may be misleading because some bacteria may not form large plaque deposits, but still may be destructive.

The presence of certain bacteria is known to cause periodontal disease. The failure to eliminate these bacteria is associated with disease progression and less favorable treatment responses. Common bacterial species associated with the majority of cases of chronic periodontitis are *Porphyromonas gingivalis*, *Bacteroides forsythus*, and *Actinobacillus actinomycetemcomitans* and spirochetes. In addition to these bacteria, there is evidence for a role for many other microorganisms that thrive in the disease-associated ecology, including *Campylobacter rectus*, *Eubacterium nodatum*, *Fusobacterium nucleatum*, *Prevotella intermedia*, *Peptostreptococcus micros*, *Streptococcus intermedius*, *Treponema denticola*, and numerous species that have not yet been cultured.

Most of these microorganisms are part of the normal flora of the oral cavity. However, a microbial analysis can be conducted to obtain information useful for patient management. Results of such testing may identify the nature of the patient’s periodontal infection and provide the clinician some guidance on the type of antimicrobial intervention to use. One group of patients, for which microbial analysis may be informative, is compliant patients who have been treated for periodontitis and continue to have clinical evidence of disease.

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**Figure 1b. The risk for future progression of chronic periodontitis in adults**
Host Response and Modifying Factors

Smoking. Individuals who smoke more than ten cigarettes each day have an increased risk of more severe periodontal disease, a less predictable response to initial therapy, and a more complicated therapeutic response. A study of adults aged sixty-five years reported 50 percent more periodontal attachment loss in smokers than in nonsmokers. In this study, the outcome of treatment for periodontal disease, including regenerative therapy, is significantly poorer in smokers than in nonsmokers. Progression of periodontal disease was five times greater in heavy smokers than in nonsmokers. These studies confirm that smoking is a powerful modifying risk factor and a prognostic risk factor for the onset and progression of periodontal disease.

Irregular Dental Habits. In addition to smoking, irregular dental habits are considered an important risk factor for periodontal disease. Patients who receive irregular dental care and do not comply with suggested oral hygiene and maintenance schedules experience more disease, worse healing, and more tooth loss compared to individuals who receive regular dental care and are compliant.

Diabetes. The influence of diabetes, both type 1 and type 2, on severity, progression, and treatment response of chronic periodontitis has been well established. In one cross-sectional study of adults, diabetes was the only systemic disease positively associated with attachment loss. Diabetes is known to influence multiple host defense and repair mechanisms that are involved in the integrity of periodontal tissues, including alterations in collagen metabolism and phagocytic cell function. Thus, the influence of diabetes on periodontal destruction appears to be due primarily to alterations in host response and repair mechanisms. This may explain why patients with diabetes that is not well controlled tend to have more severe periodontal disease than those with tighter control.

Genetic Factors. Twin studies have demonstrated that a substantial part of the variance in clinical expression of chronic periodontitis among individuals is explainable by genetic factors.

Following the first report of specific gene variations associated with chronic periodontitis in 1997, approximately sixteen candidate genes/gene families have been studied as potential factors in the severity of periodontitis in adults. Of these candidate genes, significant association with disease has been reported in more than two studies for only two of the gene families, Fc-gamma receptor and interleukin-1. Much of the current data on Fc-gamma receptor gene variations (polymorphisms) and chronic periodontitis is in Japanese populations, but recent studies among Caucasians suggest that these gene variations may explain some of the variance in disease in multiple populations. The IL-1 gene cluster includes genes that encode for the IL-1 alpha, IL-1 beta, and IL-1 receptor antagonist. Approximately 30-43 percent of the Caucasian population tests positive for the specific combination of IL-1 gene variations associated with disease progression and more severe disease. The prevalence of the IL-1 risk genotype is lower in Chinese and other Asian populations (unpublished data). The IL-1 genotype has been shown to increase the risk for progression and severity of chronic periodontitis, as shown in Figure 2.

Biochemical Parameters. A number of enzymes, tissue breakdown products, and inflammatory mediators are released from host cells and tissues during the development and progression of periodontal infections. Some of these substances have been suggested as possible markers for the detection of progressing periodontal lesions.

- Host-derived enzymes. Matrix metalloproteinase-1 is a host-derived enzyme produced by fibroblasts and epithelial cells. Matrix metalloproteinase-1 is part of the family of collagenases that has been implicated in periodontal disease progression.
- Host-derived inflammatory mediators. Some of the inflammatory mediators in gingival crevicular fluid, including prostaglandin E2, interleukin-1a and interleukin-1, acute-phase proteins, and immunoglobulin types and subclasses, have been associated with disease severity and progression.
- Tissue breakdown products. One of the major features of periodontitis is the destruction of connective tissue and bone. As tissue is destroyed, substances are released from these tissues. These substances increase during the progression of periodontal disease. Biochemical markers that are characteristic of either collagen or bone have shown promise as measures of disease progression. Hydroxyproline, an amino acid characteristic of collagen, has been used as a marker for connective tissue destruction. Current data do not support the value of hydroxyproline assays in distinguishing between sites with gingivitis or periodontitis. Other products released when bone is actively remodeling are also being investigated.
These agents include pyridinoline, collagen telopeptides, osteocalcin, and osteonectin.6

The Diagnosis and Prognosis of Dental Caries

Dental caries is a bacterial disease1 that results in destruction of tooth structure by acid-forming bacteria in dental plaque in the presence of sugar.13 The factors that contribute to the diagnosis and prognosis of dental caries will be considered in this section.

The difficulty of reliably identifying carious lesions that are not yet overtly cavitated has greatly complicated the clinical application of innovative caries management strategies. Although the histopathology and morphology of different lesions have been well described,37,38 there are practical issues of how to clinically identify and classify early stage lesions. Current technologies display diagnostic thresholds that inherently limit practical clinical detection of many lesions until late stage cavitation.39,40

Risk factors for prediction of caries activity have been described by Featherstone37 and involve a balance between well-described pathological and protective factors. The pathological factors are primarily the levels of acidogenic bacteria, the frequency of fermentable carbohydrate ingestion, and the level of saliva flow. The protective factors include salivary proteins and antibacterial components, salivary composition of key minerals—for example, calcium and fluoride—and protective dietary components. Therefore, assessment of risk for future caries activity involves the following two factors. First, of course, it is not possible to implement clinical strategies to halt or reverse the early stage lesion prior to the need for a reparative dental restoration if one cannot reliably detect such lesions. In recent years, in addition to visual-tactile examination, bitewing radiographs, and fiber-optic transillumination, other technologies have been introduced in controlled clinical trials to assess early stage carious lesions. These include electrical conductance measurements,41-43 quantitative fluorescence analysis,44-47 multi-photon imaging,48 and ultrasound.48-50

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dental caries is primarily affected by the frequency of ingestion of fermentable carbohydrates such as sucrose, glucose, fructose, and cooked starch. The frequency of ingestion is important because repeated ingestion stimulates bacteria to produce new acid. Second, people at high risk for dental caries due to irregular dental care are minority children and economically underprivileged, elderly, chronically ill, and institutionalized people. Such people experience more dental caries and should be the focus of educational and preventive outreach programs.

Plaque deposits are an indicator of bacterial infection. The infection results in the loss of tooth material that begins on the outer surface of the tooth and progresses through the dentin to the pulp, ultimately destroying the entire tooth. The presence of plaque constitutes a significant risk for dental caries.

Dental caries is caused by specific, caries-promoting bacteria that produce acid by fermentation of carbohydrates. The major cariogenic bacteria are Streptococcus mutans and Streptococcus sobrinus and several of the lactobacillus species. These acid-producing bacteria are necessary for the progression of dental caries. Levels of these cariogenic bacteria in the mouth can be assessed by selective media culturing either in a microbiological laboratory or in the dental office.

All of the components of saliva neutralize the acids produced by cariogenic bacteria. For this reason, the production of saliva is important in maintaining oral health. Any agent or condition that reduces the amount of saliva increases the risk of dental caries. Medications such as anti-asthma therapy and those with xerostomic side effects as well as radiation therapy for cancer of the head and neck may result in reduced salivary secretions. Some systemic diseases or genetically induced conditions such as Sjogren’s syndrome cause salivary dysfunction.

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**Stages of Disease and Stage-Specific Diagnostic Information**

Periodontal disease and dental caries progress through a series of stages that are described in this section. Oral hygiene practices and risk factors contribute to the movement of patients from one disease stage to another. By controlling or managing environmental risk factors, it is possible to slow or even stop disease progression. It is important to note that, at each stage of disease, certain information may or may not be valuable to the management of the individual case.

**Health Stage**

The goal of oral health is to maintain this condition by practicing good oral hygiene and managing the risk factors that contribute to disease. The effective maintenance of periodontal health requires the control of bacterial plaque and habits that lead to disease, such as smoking. For dental caries, prevention or management includes reducing the intake of sweets and control of plaque. Smoking is one of the single most important factors that contribute to oral disease.

Clinical evaluation of many risk factors in the overtly healthy patient can be assessed without substantial cost. For example, a questionnaire addressing dental history, frequency of ingestion of fermentable carbohydrates, medical history, dental history, and smoking can easily be administered. Dental professionals will add tactile and visual inspection of the teeth and periodontium using explorers and probes. Dental radiographs may be used to evaluate the interproximal surfaces of the teeth and interproximal bone levels.

Risk algorithms can be used to systematize evaluations to improve predictability of care. Some simple clinical risk algorithms may be very accurate. However, algorithms that rely on severity of past disease to predict future disease have some limitations. The primary limitation is that past disease experience does not provide critical information on factors that may be used to prevent future disease. For example, specific patients may have developed past periodontal disease for different reasons. If those reasons are not specifically explored, future prevention efforts may be misdirected. This limitation was not well appreciated when it was believed that plaque level was the only determinant of future disease. In addition, past disease history may not be informative for young patients or those at an early stage of disease.

At this stage, patients should be followed with a routine oral examination and a periodontal examination. Practitioners should explore risk factors, including smoking status and history of diabetes that can be assessed with minimal to no additional use of resources.

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Subclinical Disease

The goal of management and treatment of subclinical disease is to identify pathological processes before clinical disease develops. This involves tracking patients to determine if there is a change in health status. By definition, the detection of subclinical disease requires some type of assessment technology that identifies biological or structural changes in critical tissues before signs or symptoms are evident clinically. The rationale for subclinical disease detection involves the assumption that intervention prior to clinical detection reduces morbidity and/or cost. This assumption may be valid for caries, for which antibacterial and demineralization reversal strategies and technologies may prevent irreversible destruction that requires restoration of tooth structure. This assumption for periodontal disease does not appear to be valid, since treatment of early clinical disease is the same as treatment of subclinical disease and the damage is either reversible or of no practical consequence.

Although the value of procedures involved in detecting subclinical periodontal disease is not clear, much effort has been invested in this area. One of the reasons for this effort is that the same technologies may be useful for distinguishing progressive from stable clinical conditions. No strong technology candidates are available at this time for detection of subclinical disease. However, assays of biochemical markers of periodontal disease progression are in development. It is believed that such markers could be used clinically to determine disease progression. Enzymes currently under study are aspartate aminotransferase, matrix metalloproteinases, alkaline phosphatase, beta-glucuronidase, elastase, cathepsins, and dipeptidyl peptidases. The inflammatory mediators that might be associated with advancing periodontal disease are prostaglandin E2, acute phase proteins, and several proinflammatory cytokines. Tissue breakdown products include glycosaminoglycans, hydroxyproline, and several bone-associated proteins. To date, there are no completely validated diagnostic or prognostic tests that can help identify progressing cases of periodontitis. The most promising agents for the development of diagnostic tests are listed in Table 1.

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<th>Table 1. Possible markers for the progression of periodontitis</th>
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Early to Moderate Clinical Disease

The goals of effective management of early disease for both caries and periodontal disease are to appropriately identify and manage disease progression factors and to repair or reverse early tissue destruction. When less was known about the biology of both diseases, the approach to disease management focused on repair and control of factors that were common to all cases. For caries, the standard approaches to therapy involve cleanout of infected and damaged tooth structure, plaque control, and use of fluorides. The new knowledge of risk factors allows more attention to patient-specific identification of risk for caries progression to guide more appropriate therapy. The goals of early therapy for periodontal disease are to remove local irritants in order to stop the progression of attachment loss and to encourage the patient to comply with suggested oral hygiene and periodontal maintenance so that the stability achieved by active therapy can be maintained.11 We now know, however, that most moderate to severe periodontitis occurs in a limited segment of the population. For this reason, it is now practical to identify the early periodontitis cases that are likely to remain stable with treatment and those that are most likely to develop into severe generalized disease.

Assessments of caries and periodontitis patients in the early to moderate disease state involve the standard clinical evaluations for past disease experience, but also should include assessments of risk factors that contribute to disease activity and future progression. For caries, these include assessment of dietary intake of carbohydrates, cariogenic bacteria levels, and salivary flow.1,51-53 For periodontal disease, risk assessment should focus on the well-established risk factors for disease progression. These include total bacterial levels, diabetes, smoking, and genetic predisposition.54-55 Microbial testing may be important for individuals who continue to experience disease progression despite excellent compliance and well-executed conventional therapy or for young patients with highly destructive forms of periodontitis. Detection of specific bacteria can be performed by culture, microscopic examination, nucleic acid analyses, detection of specific bacterial antigens, and detection of bacterial enzymes.

Severe Generalized Disease

The goals of treating severe generalized periodontitis or extensive caries are to stop the progression of current disease, primarily by eliminating bacterial reservoirs; repair structural damage and restore function; and establish a disease prevention regimen based on the patient’s specific risk factors.

In patients who already have severe disease, risk factors are used to guide the selection of therapy and focus on specific prevention approaches. Regular monitoring of the patient’s risk factors allows the practitioner to efficiently manage the individual’s specific needs. Such approaches have been demonstrated to be effective in clinical practice.9,53,55

Conclusion

There are extensive data about the factors that determine the initiation and progression of dental caries and periodontal disease. Both diseases are caused by specific bacteria, but the rate of progression and severity are influenced by systemic factors and habits, such as smoking. The collection and application of certain information about factors that influence disease should be useful to improve disease prevention and management. Knowledge of when specific information may be valuable should lead to the optimal management of individual patients.

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