Casey Hein, RDH, MBA

The Reality of the Risk Continuum

The science no longer supports a clinical crusade aimed exclusively at chasing down microbial plaque while disregarding other factors in the etiological cascade of events in periodontal disease.

The number of clinicians who identify and treat periodontal disease according to current etiological theory, commonly referred to as the Host-Bacterial Interaction Theory, may be in a very small minority.[1] As a result, many clinical outcomes of nonsurgical periodontal therapy fall short of optimal therapeutic endpoints and/or have difficulty sustaining periodontal stability during the maintenance phase of care.

What we know today about periodontal disease etiology is very different than what we thought in the past—"the clinical course of periodontitis in patients can vary greatly despite their harboring similar quantitative and qualitative levels of bacteria."[2] Current etiological theory suggests that in addition to acquired, environmental, and genetic risk factors, a multifactorial, highly complex interaction between the bacterial challenge and how a patient's immune system responds to the bacterial challenge (host response) is the key to unraveling the mystery of periodontal disease.[3]

Researchers have come to believe that it is this highly complex cascade of events that control the onset, severity, and rate of progression of the disease, and the patient's response to periodontal treatment.[3-8] According to consensus opinion, "ultimately, it is the host's reaction to the presence of bacteria that mediates tissue destruction."[9] So how does this information translate into daily clinical practice?

The goal of this series of articles is to provide an overview of how periodontal disease etiology theory has evolved over the last 5 decades and where the science has brought us today. My hope is that this might help readers identify outdated thinking and jump-start the process of redefining/refining clinical protocols and patient education to a level that better represents the findings of current research and the state of the science.

Universal Susceptibility Abandoned

Perhaps one of the most revealing discoveries related to the etiology of periodontal disease over the last 50 years is that not all people are equally susceptible. Although a gram-negative infection of the periodontal pocket is necessary for periodontal disease to occur, the presence of these virulent bacteria alone is not sufficient to initiate periodontal disease or escalate its progression.[3] Instead, it all comes down to how a patient's unique immune system responds to the infection.

The host response varies significantly between patients. These variations in host response are influenced by environmental, acquired (both locally and systemically), and genetic risk factors. Impaired, delayed, or exaggerated host responses are manifested in patients who present with a disproportionately small amount of plaque compared with the extent and severity of the periodontal destruction that has taken place in their mouths.[10]

These kinds of host responses are often associated with impaired wound-healing, white cell function, and ability to kill bacteria, and the suppression of local antibodies that hinder the host's ability to protect itself against periodontopathic organisms. Smoking, diabetes, and genetic predisposition to periodontal disease (commonly recognized as the IL-1 polymorphism) are all associated with immune system defects, which explains why they are considered risk factors for periodontal disease.[11,12]

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The American Academy of Periodontology states that there are "certain patients [who] possess nonmicrobial risk factors which are difficult to reduce or eliminate (ie, smoking and diabetes) or are beyond the clinician's ability to control (ie, genetic predisposition)..."[3] This explains why some patients with relatively plaque-free mouths develop periodontal disease and/or continue to lose attachment and bone, while other patients with a lot of plaque develop gingivitis that never crosses over that inflammatory threshold to chronic periodontitis.

Assessing periodontal disease risk is considered a cornerstone of comprehensive periodontal care.[13] Although periodontal disease risk assessment is beyond its infancy, its transfer to general practice settings can be likened to a recalcitrant toddler who refuses to learn to walk, and, therefore, continues to crawl.

Preparing Clinicians to Look Beyond the Pockets

Many clinicians know that chronic gram-negative infections of the periodontium may have a wide range of systemic effects. However, few clinicians have read the studies that discuss the strength of the evidence to support perio-systemic links or the theories of mechanism that explain the association of the disease links.[1]

Casey Hein, RDH, MBA

Casey is a periodontal therapist subcontractor in the Washington, DC/Baltimore area and founder of PointPerio, LLC, a clinical education consulting firm committed to coaching general dentists and hygienists in progressive periodontal therapeutics within a collaborative framework. She is a member of the American Dental Hygienists' Association. Casey lectures nationally on the role of the periodontal therapist in general practice and regularly contributes practice management articles to Dentist's Money Digest. *She welcomes comments and visitors to her Web site www.@pointperio.com.*

A thorough understanding of these multifactorial relationships is critical in responding to the call to action set forth in *Oral Health in America: A Report of the Surgeon General*, which states that "the mouth is the center of vital tissues and functions that are critical to total health and well being across the life span…the mouth is a mirror of health or disease, as a sentinel or early warning system, as an accessible model for the study of other tissues and organs, and as a potential source of pathology affecting other systems and organs."[14,15]

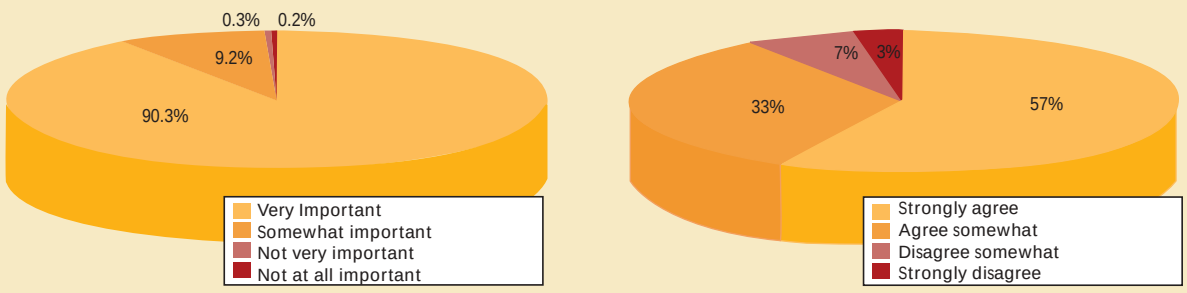
With each new research finding, pieces of the evidence puzzle seem to be validating that "the mouth is the center of vital tissues." A remarkable evidential finding came from the recently published epidemiological study known as INVEST (The Oral Infections and Vascular Disease Epidemiology Study), which explored the relationship of periodontal disease and tooth loss with subclinical atherosclerosis. The startling results of the study of 711 subjects with no history of stroke or myocardial infarction showed a significant association between tooth loss levels and carotid artery plaque prevalence. Among those with 0 to 9 missing teeth, 46% had carotid artery plaque; among those with 10 or more missing teeth, carotid artery plaque prevalence was 60% ($P<.05$), which is statistically significant. The conclusion of the study suggests, "that tooth loss is a marker of past periodontal disease in this population and is related to subclinical atherosclerosis, thereby providing a potential pathway for a relationship with clinical events."[16]

Sometime ago, scientific investigations documented how bacteria from the oral cavity can be released in the bloodstream to cause bacteremia.[17] Yet, more recent research related to endotoxins—substances produced by periodontal pathogens that suppress the activity of or kill polymorphonuclear leukocytes and lymphocytes involved in host defenses—demonstrated that it may be endotoxins that play a causative role in the development of systemic pathologies.[10,18] In this study, researchers investigated whether endotoxins could be released into the blood stream, producing endotoxemia as a result of the pumping effect created by the mechanical forces of normal mastication.[18]

The study found mastication induced an increase of endotoxemia in both healthy and periodontitis patients; the level of endotoxemia was significantly higher in the periodontist group.[18] The researchers concluded that "the oropharyngeal cavity can be a major source of chronic or even nearly permanent release of toxic bacterial components in the bloodstream under the influence of normal oral functions."[18] They also stated that "this could be the missing link" [to explain why] periodontal disease has been found by some authors to be statistically associated with the risk of occurrence of some systemic diseases such as cardiovascular diseases.[18]

We seem to be getting closer to solving the mystery of periodontal disease's association with

Figure 1—What is the importance of maintaining oral health? Consumer-patient opinions



"How important is prevention of 'gum disease' in maintaining overall health?"

"Is there a link between 'gum disease' and other health problems?"

whole body consequences, but this is just the tip of the proverbial iceberg compared with what will be discovered within the next decade. Given the exponential rate of new information emerging from the research pipeline and the responsibility we have to meld this new research into clinical practice, the gap in transferring new science to private practice may be widening.[1]

Being able to connect the dots when assessing patients' risk for periodontal disease, incorporating those findings into treatment recommendations, and integrating credible evidence of periodontal-systemic risk is quickly emerging as a prerequisite skill set for practicing as a periodontal therapist in the 21st century. Between 2000 and 2010, the projection for the number of new jobs for dental hygienists is 54,000, which represents a 37% growth rate over new jobs from 1990 to 1999.[19] Although this growth rate appears to be over-the-top, it is my opinion that during this time, there will be a shake-out of expertise that differentiates dental hygienists based on the level of periodontal care they provide. I believe the demand for periodontal therapists is relatively high at this time and will increase in the future.

Accordingly, dental hygienists (aka, periodontal therapists) who are passionate enough to focus on perfecting clinical outcomes of the nonsurgical periodontal care they render will be self-directed learners, dedicated to updating their knowledge and skill sets. In this regard, whether the term *periodontal therapist* is officially recognized, I predict that by 2010 market demands and public health mandates will dictate the need for the periodontal therapist clinician. Patients will increasingly choose a dentist's office based on whether a periodontal therapist is employed there.

In addition, as the trend toward outcome assessments and evidence-based decision-making in dentistry continues, insurance companies may start looking for the periodontal therapist level of expertise when making decisions related to reimbursement for nonsurgical periodontal procedures rendered in general practice settings. Hopefully, this will translate into increased accountability for the clinical decisions we make and a newfound awareness for the technical capabilities necessary to achieve and sustain positive therapeutic endpoints, which will be welcome in the era of accountability.

> Meeting the needs of a growing and highly educated population will require constant vigilance to keep up with the science.

Today's consumer-patients have a preoccupation with ensuring whole-body wellness over repair of their bodies and they are becoming increasingly knowledgeable about possible periodontal systemic links. Figure 1 summarizes the findings of 2 very important oral health beliefs that indicate that most consumer-patients are aware that there is a link between periodontal disease and systemic consequences, and more than 99% recognize that prevention of periodontal disease is an important step in maintaining oral health.[20]

Meeting the needs of a growing and highly educated population will require constant vigilance to keep up with the science. Achieving and sustaining therapeutic endpoints will largely depend on how well clinicians understand the risk continuum, which means knowing how to connect the dots between risk that predisposes a patient for periodontal disease and, once initiated, periodontal disease as an exposure to systemic, whole-body consequences that are a result of gram-negative periodontal infection (Figure 2).

Given evidence that most currently practicing clinicians do not fully understand the multifactoral and complex relationships between gram-negative bacterial periodontal infection, the host response, and the effect that may have on systemic health, how do we prepare clinicians for rendering this level of care?[1]

Several questions emerge regarding how to ensure dental hygienists know current etiological theory of periodontal disease initiation and progression, know how to appropriately discuss the systemic implications of periodontal disease, and have the ability to turn that information into treatment recommendations.

1. How can we bridge the gap between what is known and what is practiced relative to periodontal disease risk and evidence that supports periodontal-systemic links?

Many clinicians who are unsure of the science may feel they are going out on a limb by discussing periodontal systemic associations.[1] On the other extreme are those clinicians who have started talking to patients about the potential of periodontal pathogens to translocate via intravascular "seeding" to other parts of the body, but these discussions can elicit a response of fear rather than a real understanding of the risk relationship.[1] Among this group of well-intentioned clinicians there may be some who have not studied the strength of the evidence necessary to support these claims. In these situations, the patient receives information on periodontal systemic links that can be very confusing and often contradicts consensus opinions formed through exhaustive meta-analysis, which eventually jeopardizes the credibility of both the clinician and the profession as a whole.

In this regard, any reference to the potential of periodontal disease causing heart disease or periodontal-medical associations presently under investigation should be carefully worded to match the science. If we are going to talk about periodontal-systemic links with our patients, we better make sure we understand it well. Increased education related to periodontal-systemic medicine needs to be added to undergraduate curriculum and advanced level continuing education courses, focusing on how to incorporate periodontal-systemic medicine into daily practice.

2. Is there enough time in current 2-year and 4-year program curriculums to teach etiological theory to a level sufficient enough for dental hygienists to

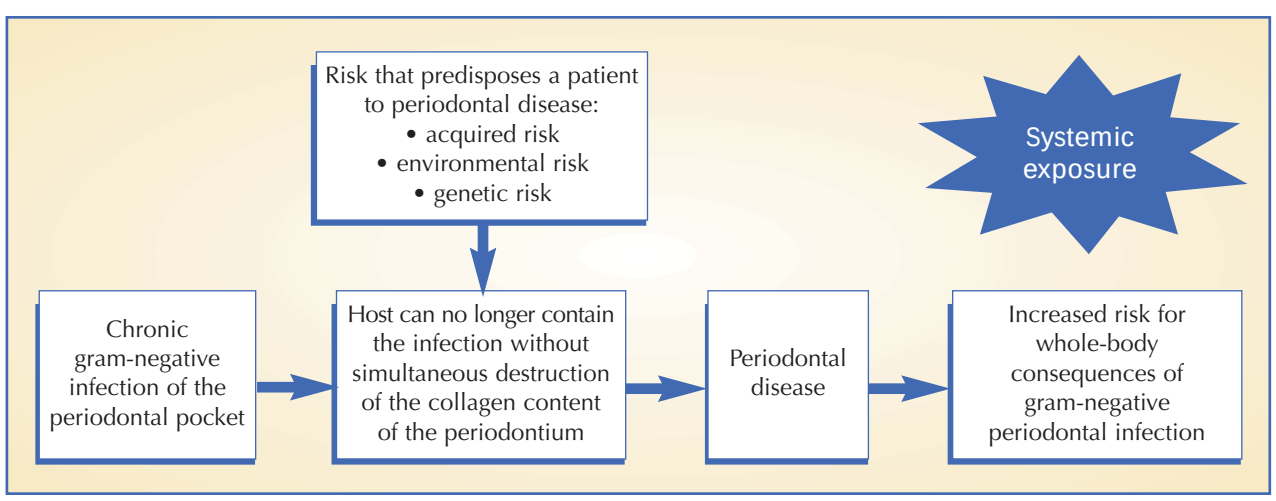
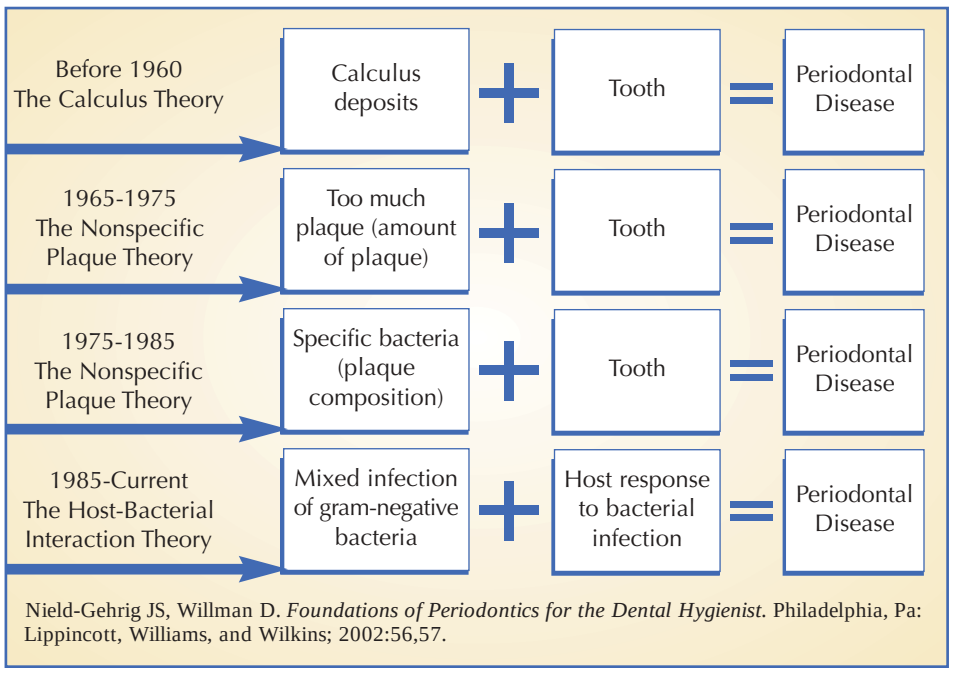


Figure 2—The risk continuum: connecting the dots between risk that predisposes a patient for periodontal disease and, once initiated, periodontal disease as an exposure to systemic, whole-body consequences that are a result of gram-negative periodontal infection.



Nield-Gehrig JS, Willman D. *Foundations of Periodontics for the Dental Hygienist.* Philadelphia, Pa: Lippincott, Williams, and Wilkins; 2002:56,57.

Figure 3—The evolution of the etiological theory of periodontal disease.[1]

thoroughly comprehend the complexity of the bacterial-host interaction in causing periodontal disease and the causative role and implication periodontal disease may have in compromising systemic health?

Undergraduate dental hygiene education offers an average of only 2,000 classroom hours, and requirements for core competencies impose significant limitations in terms adding to the curriculum.[21] Therefore, it is highly unlikely that current 2-year and 4-year programs will be able to sufficiently teach etiological theory without dramatically changing curriculum requirements and hours spent learning the curriculum.

A Perspective from the Top

I recently had an opportunity to interview John Gunsolley, DDS, the director of the Center for Clinical Studies and chairman of the Department of Periodontology at the Dental School, Baltimore College of Dental Surgery, University of Maryland, Baltimore, Maryland (J Gunsolley, oral communication, July 2004). Gunsolley, an expert in meta-analysis of periodontal research, has a healthy respect for dental hygienists who have a strong understanding of the science.

I asked Gunsolley, "Is there enough time in current didactic and clinical education for dental hygienists to learn what they need to learn, given the amount of new information we have about periodontal disease?"

"It depends on what kind of hygienist you want," he said. If we assume that patient populations are homogeneous (no differences in susceptibility and a one-size-fits-all treatment protocol), then perhaps it is not necessary for dental hygienists to develop critical thinking skills. What is happening in many general practice settings relative to soft-tissue management programs is "a little scary" because many dentists and dental hygienists do not recognize patients' different levels of susceptibility. "The research is very clear that we, as a profession, have not been very good at changing patients' behavior related to plaque control [and] that we have been relatively ineffective in getting patients to comply." For some patients though, "you can't blame the hygienist" for the inability to inspire good plaque control, "because these are patients who will lose attachment regardless of excellent oral hygiene" (J Gunsolley, oral communication, July 2004).

Gunsolley's comments echo the new paradigm shift in disease etiology—it is no longer all about plaque control. Whether a patient "loses ground" relative to periodontal stability depends more on the patient's host response to the bacterial challenge than on the bacteria itself.

Etiological Theory Revisited

Etiological theory surrounding periodontal disease has evolved dramatically since the 1960s (Figure 3). Unfortunately for the public we serve, the identification of periodontal disease and many recommendations made for periodontal care are still a function of what most clinicians learned in school and do not always reflect clinical decisions based on current etiological theory. As a result, clinician judgment is compromised and treatment recommendations are unsupported by science in many instances. The host response component of the disease cascade can no longer be overlooked and the clinicians who understand this part of the disease equation are in a minority.

In a future issue, we will take a more in-depth look at how these etiological theories differ and the clinical implications of those differences. In the meantime, before you tell another patient that all they have to do is floss everyday to ensure periodontal health, you might want to assess their risk for periodontal disease. COH

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