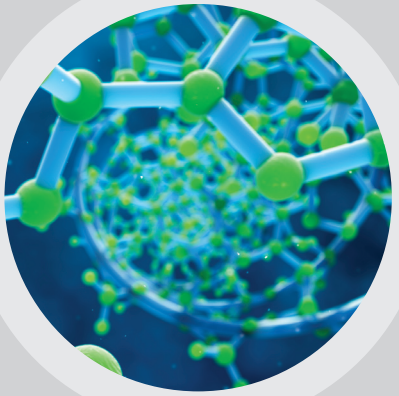
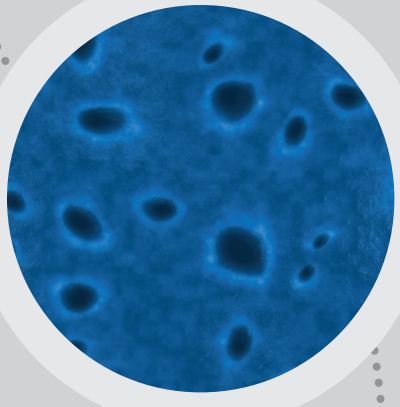
WHAT DO THEY HAVE IN COMMON?

Genetics

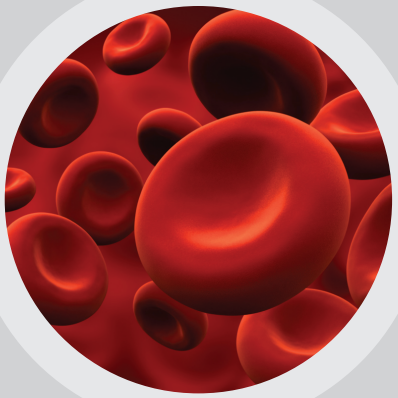


Periodontal Disease



Cardiovascular Disease

Diabetes



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In the 25 plus years I have been a hygienist, I am amazed at how much research and science has expanded our understanding of biofilms, inflammation, periodontal disease development, and how a patient's genes affect oral/systemic disease activity.

In the 1960's the non-specific plaque hypothesis supported the idea that it was quantity rather than the quality of plaque that caused disease. Calculus was viewed as the primary cause of the periodontal inflammation. Disease was controlled through removal only by scaling. Definitive "root planing" was the key in the 1970's, with the goal being the removal of contaminated surface cementum creating a smooth hard surface. By the 1980's periodontal inflammation was directly correlated to specific invasive microorganisms. Periodontal Debridement was the primary goal of instrumentation and no longer was scaling and root planing the sole modality of treatment. Perio debridement focused on the resolution of the inflammation and the disruption of bacterial colonies through microsonics. In the 1990's periodontitis activity was considered multifactorial and dependent on the interaction between biofilm communities and the host immune response. Finally in the 21st century, we now know that tissue response with zero bleeding is the new clinical end point of therapy that follows bacterial reduction and balance. Perio pathogens represent less than 5% of the 700 species of bacteria in the mouth, and we know that it is those specific groups of bacteria that aggregate on biofilms that cause periodontal disease.

The clinician must understand how to manage this biofilm based on the understanding of infection and inflammation as it relates to the medical model. Infectious pathogens may cause disease by two principle means. The first is the direct toxic effects of the bacteria and their endotoxins, which happens when bacteria are introduced into the body. The pathogenic bacteria intimately reside along the epithelium lining in the periodontal pocket and can rapidly enter the cells and migrate through the epithelium into the underlying connective tissue. This results in a bacteremia consisting of live bacteria and their endotoxins floating in our body. The second way the pathogen may cause disease is from the host inflammatory response. Although it is essential to mount an effective host response to invading pathogens, paradoxically the host inflammatory response may cause as much pathology as the initial bacteremia. The indirect pathway of pathogenesis results from the host inflammatory response by way of the immune system that comprises inflammatory mediators and cytokines.

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Infection and inflammation are not the same thing. Infection is caused by bacteria and is the etiology of periodontal disease. The pathogenesis of periodontal disease is inflammation. Inflammation is the body's defense to the offending environment. Inflammation can be in defense of self against non-self as in a bacterial or viral assault. It can be in defense of self against self as in autoimmune diseases. Inflammation is also a consequence of the activity of our own immunity. Cells that make up our immune system produce proteins as a response and are manifested as the classic signs of inflammation: pain, swelling, redness, warmth.

The body responds to inflammation by sending out the scouts of the innate immune system, such as Beta-Defensin 1 (DFB1), CD14, and Toll-like Receptor 4 (TLR4). Defensins are an immediate response to pathogenic bacteria, fungi, and some viruses. CD14 is a receptor present on monocytes, macrophages, and neutrophils that recognize pathogenic bacterial cell wall lipopolysaccharides. TLR4 signals and connects the troops with the acquired immune response. The cells that are released by the innate immune system typically herald in the arrival of the cells of the acquired immune system. These proteins

include Tumor Necrosis Factor (TNFa), Interleukin 1 (IL1), Interleukin 6 (IL6), Interleukin 17A (IL17A), and Matrix Mellatoproteinase 3 (MMP3).

All of the above listed proteins have specific gene markers that predict a person's set point or baseline inflammatory response. These gene markers will also dictate whether the body can mount a defense against the offending environment, self or non-self. We now know that we should look at a patient with a comprehensive view in mind. We used to look at only one genetic marker because that was all we had. We tried to make it fit the Mendel's Law of genetics. A Mendelian trait was one that was controlled by a single gene in an inheritance pattern. A mutation of a gene would cause a disease that could be inherited such as sickle-cell anemia or cystic fibrosis. We now know that when assessing the genetic risk periodontitis as well as other systemic diseases such as cardiovascular disease or diabetes, research now points to the multi-gene approach. It is exciting that we now have a test that can analyze the genotypes of 10 independent gene markers that are a cross section of both the innate and acquired immune system. This will give the dental clinician a broader picture of that person's inflammatory risk profile. The genetic test can be used to relate the systemic inflammatory process to cardiovascular disease and diabetes. Inflammation has come a long way from something observed as normal response to non self to now where we see a little is good but a lot is not necessarily better. For example, short-term or acute inflammation allows our body to heal and protects the body as in a cut or scratch. When chronic inflammation happens, the inflammatory host response is out of proportion to the threat and goes against inappropriate targets, like in the case of autoimmune diseases and periodontal disease.

Ultimately, sustained disease remission is the goal of perio therapy.

So what can we do as clinicians to change chronic inflammation? There is very little we can do about our genetic predisposition. However, we can be aware and know our patient's risk as far as their genetic "wiring" for inflammation as it impacts their overall health. This will help support them in a customized treatment plan for perio and better lifestyle choices. The goal of genetic testing for inflammation is to identify those people who are at increased risk of inflammation by virtue of the fact that they subsequently will have chronic elevated levels of inflammation throughout their body. This sets the stage for future risk of systemic disease if their lifestyle choices are not in alignment with health. Genetics loads the gun, but lifestyle pulls the trigger. Their genetics can't be changed. It is the things that multiply that genetic predisposition further that can be modified. Together with a knowledgeable clinician, the patient has a lot more information as to the why their body is responding and he or she can be an integral part of the supportive solution. Ultimately, sustained disease remission is the goal of perio therapy. Specifically the clinician will be carefully looking at clinical signs of chronic inflammation, the elimination of the pathogen risk, as well as the overall genetic inflammatory response of the patient.

I hope that you attend the IAPA where I will be explaining the following in depth:

1. Understand the difference between infection and inflammation as it relates to systemic disease.
2. Discuss the difference between the innate immune system and the acquired immune system.
3. Discuss the genetic response of a patient and how it will affect a treatment plan as well as long-term maintenance.

Hygiene has certainly come a long way since I first started…it is exciting to utilize all this knowledge for the benefit of our patients!

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