

# Viruses in Periodontitis and Whole Mouth Disinfection

There is a vast amount of published literature on the causes of periodontitis site bacteria as the most likely cause of periodontitis. These studies also acknowledge the important contributing factors to this disease, including genetic pre-disposition, nutrition, occlusion, host resistance, immune over response and virulence and mix of bacterial species. Few of them acknowledge virus co-infection with periodontitis and fewer yet acknowledge their significance in the pathogenesis of the infection, however there is a growing body of evidence that they do play a significant role in the more aggressive periodontal infections. More studies are naming viruses as contributing causes in certain types of periodontitis, including more aggressive chronic periodontitis and especially refractory periodontitis. They are also describing the mechanism for how they are affecting the host tissues to accomplished attachment loss.

According to Dr. Jorgen Slots of the University of Southern California, viruses causing periodontitis are enveloped. Most aggressive periodontitis patients have one or more varieties of herpes virus, essentially 100%. Periodontal lesions are the main source of oral cytomegalovirus (CMV). When there is no periodontitis, then there is usually no CMV in the oral cavity. These viruses are found in macrophages and t cells and they are only found in periodontitis patients not in gingivitis patients. . These statements come from several published studies by Jorgen Slots. Many can be found in the California Dental Association Journal, June 2011.

Slots's finding on viruses is supported by a study on human T lymphotropic virus 1 in the Clinical Infectious Diseases Journal, 2010:50:e11-e18 entitled, "Association of Human T Lymphotropic Virus 1 Amplification of Periodontitis Severity with Altered Cytokine Expression in Response to a Standard Periodontopathogen Infection". In this study HTLV-1 seropositive individuals with chronic periodontitis (CP/HTLV-1) had significantly more bleeding, probing depth and attachment loss than did individuals with chronic periodontitis (CP) with seronegative HTLV-1 lab tests. The article goes on to site the cytokine alterations produced by the virus infection.

"The expression of tumor necrosis factor  $\alpha$  and interleukin (IL) 4 was found to be similar in the CP and CP/HTLV-1 groups, whereas IL-12 and IL-17 levels trended toward a higher expression in the CP/HTLV-1 group. A significant increase was seen in the levels of IL-1 $\beta$  and interferon  $\gamma$  in the CP/HTLV-1 group compared with the CP group, whereas expression of the regulatory T cell marker FOXP3 and IL-10 was significantly decreased in the lesions from the CP/HTLV-1 group."

The conclusion of the article was that HTLV-1 may play a critical role in the pathogenesis of periodontal disease through the deregulation of the local cytokine

network, resulting in an exacerbated response against a standard periodontopathogen infection.

Slotts said that the family of herpes viruses remain viable and dormant inside human immune cells such as t-cells, lymphocytes and b-cells. These include cytomegalovirus, Epstein Bar virus and herpes virus 1. The mechanism of pocket infection appears to be one in which these immune cells attack the bacteria infection but at the same time deposit their viruses into the pocket, establishing the viral infection. Once that happens the body recognizes the viral infection as more serious than the bacterial infection and switches from a humoral immune response to an innate cellular response. Essentially then, the body begins to ignore the bacteria to fight the virus. This results in a fulminating bacterial infection free to attack more aggressively.

Slotts went on to say that chlorhexidine is ineffective sub-gingivally because the molecules are too large and tie up protein. Slotts says that chlorhexidine and fluoride do not kill on contact. Slotts recommends sub-gingival irrigation with antibacterial rinses, however, they only go 0.1mm into the sulcus when a person merely swishes with any antimicrobial mouth rinse without other help. He says you must use an irrigator to get the antimicrobial down into the pockets. He then recommends combating the virus with Valtrex prescription.

We recommend Oracare as the rinse for this purpose. Although Dentist Select does not yet have clinical studies that prove sub-gingival antiviral activity in periodontitis, we do know from other industry studies that chlorine dioxide does kill viruses. We also know that chlorhexidine is not effective on viruses and is not approved for use sub-gingivally. Oracare can be used in the office and also at home by the patient for sub-gingival irrigation. Slotts also recommends you treat the tongue, a reservoir for bacteria and yeasts that can re-seed microbes to the teeth and gingiva. He says that tongue scraping is good to do but it does not do the whole job. He says you must use an antimicrobial rinse on the tongue to kill the microbes deep down in the villa. He recommends a whole mouth disinfection. This can and should be done at the dental office and also can be done at home by patients with proper instruction.

Slotts recommends hygienists irrigate sub-gingival before they scale. He also recommends sub-gingival irrigation with antimicrobial after scaling. Again, he recommends full mouth disinfection. He also places advanced periodontitis patients on two systemic antibacterial antibiotics and one antiviral antibiotic. 10 days later he recommends they do another round of sub-gingival antimicrobial irrigation and removal of calculus that becomes exposed during periodontal healing. We believe Slotts is correct in these recommendations and believe Oracare is a perfect product for the sub-gingival irrigation and whole mouth disinfection he advocates.