Gingivitis, Periodontitis, Peri-Implantitis
(The Connection)

Periodontal pathogens have been linked with increased risk of systemic illness and complications in existing disease. Recently, several articles detailing these findings have been published, emphasizing the importance of the association between periodontitis and systemic health problems. In fact, a recent meta-analysis reports that oral infections have been shown to be associated statistically with mortality. By entering the bloodstream, periodontal pathogens have been shown to increase the risk of cardiovascular and pulmonary disease, and hinder glycemic control in diabetes.

There are least 30 bacterial species that cause and contribute to inflammatory periodontal disease including – *Eikenella corrodens, Fusobacterium nucleatum, Actinobacillus actinomycetemcomitans, Tannerella forsythensis, Prevotella intermedia, Porphyromonas gingivalis, Treponema denticola* – all of which exist in varying forms of heterogeneous biofilms, which are highly difficult to eradicate. With periodontal disease, millions of bacteria are in direct physical contact with ulcerated pockets of epithelial tissue, which provides an easy portal to the circulatory system. As a result of ensuing bacteremia, the body mobilizes inflammatory mediators such as cytokines, PMN, B-cells, and T-cells. Enzymes such as collagenase, gelastinase, elastase, and proteases are also produced; these enzymes cause the majority of damage to the periodontal tissue and bone. As part of the normal immune system response, the liver produces the C reactive protein (CRP), which also elevates the risk of heart attacks and strokes, in response to injury, inflammation or infection. Although periodontal disease is not the only contributor to elevated levels of CRP, statistically significant increase in CRP levels were observed in subjects with periodontal disease as compared with healthy controls.

Among the dental consumer population, dental implants are an increasingly popular option for replacing teeth and restoring function. Since implants became a common modality, assessments of their success have included improved appearance, restored ability to eat, rate of bone integration, and longevity of placement. By and large, the most appealing appearance possible drives the choice of dental implants for the patient. As opposed to many early designs, most dental implants now provide a natural-looking appearance. However, both patients and dentists should be aware of possible complications that could affect the patient's oral and systemic health, even when an excellent esthetic result is achieved.
In addition to optimizing esthetic results, concerns about infection of tissue surrounding the implant, or ***peri-implantitis***, other concerns have driven many of the developments in dental implant design and use. In fact, as with many areas of health care, a current paradigm shift moves consideration of dental implants from solely optimal repair, toward prevention of future health problems that may be implant related. Thus, treatment

of edentulous patients is increasingly moving from an approach focused primarily on esthetic and functional concerns, toward an approach including optimal health as a critical goal. Numerous published studies promote preventing the harboring of oral bacteria around implants as a key outcome, in addition to traditional measures of implantation success. Possible negative outcomes include failure of the implant to integrate with the bone, and infections that can lead to bone and soft tissue loss.

In the past decade, an abundance of articles indicates the importance of dental implants as a superior treatment modality for the replacement of teeth, relative to other restorative procedures. For the most part, implants are a widely available option and are being used with rapidly increasing frequency. Traditionally, implant success has been measured by three major criteria: esthetic result, restored function, and longevity of the implant. Failure, or implant loss, can be a result of several variables. Although individual reports vary widely and most are based on small patient numbers, these clinical variables include pre-existing periodontitis, peri-apical lesions, diabetes, or bone disease, as well as behavioral factors, such as smoking and alcohol consumption. Retrospective studies have given wide ranges of estimates for implant failure and progressive bone loss, traditionally for a 5-year period. More recently, long-term survival review studies of outcomes up to 14 years after placement have been reported clinical (probing depth and peri-implant mucositis) and radiographic assessments. However, marginal bone loss and other parameters initially prescribed for evaluation of implant success are repeatedly being re-evaluated. In particular, the assessments of bone and soft tissue response to implants range from the Albrektsson's pattern, a low-rate marginal loss, to the development of a continuously high rate of bone loss. Evidence of peri-implant infection, progressive bone loss and probing depth/bleeding upon probing is well documented in the literature. Peri-implant lesions are speculated as being relatively common, although prospective studies of multiple implant types are lacking insufficient numbers of patients and controlled conditions. Implant characteristics and surgical aspects have been evaluated considerably to provide a greater selection of implants, and increase the likelihood of success. Specifically, studies have evaluated implant length, diameter size, implant coating, and timing and location of implant placement and loading, with varying results. For example, one-stage placement protocols and short implant length have been associated with increased rate of failure (implant loss) over the standard 5-year evaluation period. Whether or not mechanical or biological factors are etiologic, ultimately the failure of implant osseointegration has been shown to be due to an inflammatory response to harboring of bacteria in the peri-implant region, in a fashion similar to periodontitis. It is this inflammation that causes not only failure of implant after placement, but may lead to health complications similar to those caused by periodontal disease.

Researchers have identified periodontal pathogens around and within the micro-gap of implant systems to be the same as seen in periodontal disease. These pathogens may have a direct systemic relationship in the areas of cardiology, orthopedics, general

health, pulmonary problems and pre-term birth as seen in periodontal disease. The implant system companies have not addressed this problem.

Studies have shown that periodontal pathogens surrounding dental implants contribute to peri-implant infections and can be the main cause of implant loss. However, this is not the only location where periodontal pathogens are sequestered. In vivo and in vitro studies have shown that dental implants with varying designs are capable of harboring bacteria within the micro-gap between the implant and the abutment. These periodontal pathogens are the same bacteria that cause periodontitis. The initial colonization of peri-implant pockets with periodontal bacteria has been shown to occur rapidly after implant placement. Even in the absence of clinically significant inflammation, detection of post-inflammatory molecules may indicate peri-implantitis. However, many patients may have significant infections and bone loss, with no symptoms, and may not pursue adequate follow-up care that would identify those conditions. Professional implant maintenance and diligent patient home care are important factors, but because the peri-implant surfaces are subgingival, patients and clinicians have limited control over hygienic measures to prevent infection. Even patients who are scrupulous in their hygienic care can suffer with implant infections if bacteria are harbored within and around implant components. **Because the micro-gaps at the implant abutment junction (IAJ) are non-cleansable, dental implants may post a risk to systemic health, if peri-implant infection leads to the same consequences as periodontitis.**

Sufficient evidence exists to conclude that both periodontitis and peri-implantitis involve the same bacteria, and the same inflammatory process that damages tissues, and also leads to bone loss. Because so many variables may be involved in the success or failure of implants, many variables have been explored in numerous studies and review articles over the years. These include optimizing the health of the bone at the implant site, and approaches to decrease or elimination bacteria on implant surfaces, evaluating different surface textures, **as well as elimination of the micro-gap of the implants to prevent peri-implantitis from the periodontal pathogens.**