

Gingivitis: New insights into inflammation and periodontal diseases.



At the forefront of dentistry today is an increasing knowledge of the role of chronic inflammation and the changes it can cause in both the oral cavity and systemically. Now you can gain a new level of understanding with these informative articles to help you more effectively treat your patients with gingival inflammation. Originally appearing in the July 2004 Supplement to the *Compendium of Continuing Education in Dentistry* entitled, "Gingivitis: An Inflammatory Periodontal Disease" these articles are available to you online at www.ColgateProfessional.com or by calling your Colgate Representative.

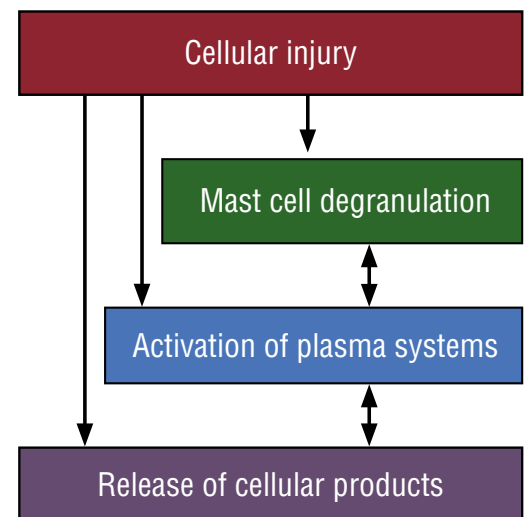
CE1 A Primer on Inflammation

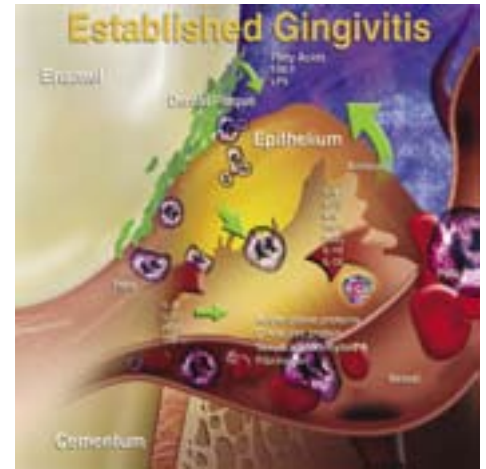
Angelo Mariotti, DDS, PhD

Inflammation is the localized, protective response of the body to injury or infection. The classic clinical signs that characterize inflammation are heat, redness, swelling, pain, and loss of function. During inflammation, cells and their secreted chemicals attempt to destroy, dilute, or wall off the injurious agent. A series of biochemical events cause the blood vessels to dilate and become more permeable, resulting in the activation of the complement,

clotting and kinin systems. The end result of inflammation is the return of function by the regeneration or repair of the affected tissue. In some instances, inflammation may continue for a prolonged period of time, producing untoward consequences for localized tissue as well as the entire body. The purpose of this article is to provide a basic and simplified understanding of how the inflammatory process functions in the human body.

ACUTE INFLAMMATORY PROCESS



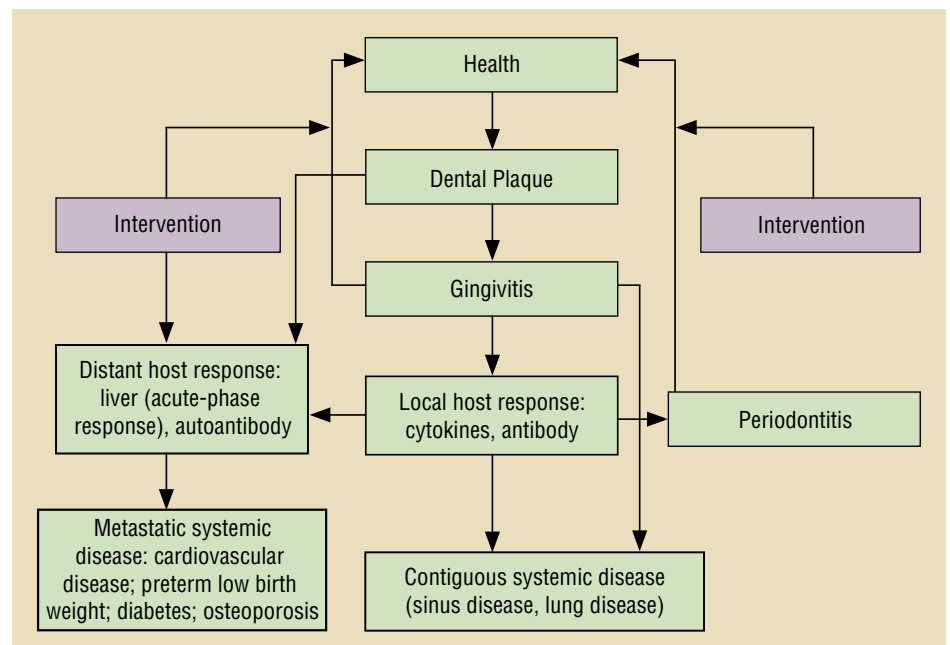


CE2 Periodontal Inflammation: From Gingivitis to Systemic Disease?

Frank A. Scannapieco, DMD, PhD

There has been a resurgence of interest in recent years in the systemic effects of oral infections such as periodontal diseases. The study of the various means by which periodontal infections and inflammation may influence a variety of systemic conditions is collectively referred to as periodontal medicine. The periodontium responds to tooth-borne biofilm (dental plaque) by the process of inflammation. Dental biofilms release a variety of biologically active products, such as bacterial lipopolysaccharides (endotoxins), chemotactic peptides, protein toxins, and organic acids. These molecules stimulate the host to produce a variety of responses, among them the production and release of potent agents known as cytokines. These include interleukin-1 beta, interleukin-8, prostaglandins, and tumor necrosis factor-alpha. There is a spectrum of periodontal response to these

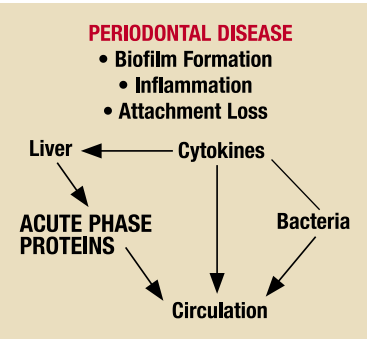
molecules, from mild gingivitis to severe destructive periodontitis. These and other host products and responses may influence a variety of important disease pathways, including atherosclerosis, mucosal inflammation, and premature parturition. The purpose of this article is to review the possible biological pathways by which periodontal diseases may influence these disease processes.



CE3 Cardiovascular Disease and Periodontal Diseases: Commonality and Causation

Sheilesh Dave, DDS; Eraldo L. Batista Jr, DDS, MSc; Thomas E. Van Dyke, DDS, PhD

Periodontal diseases have long been recognized as a public health problem. Awareness of the destructive nature of periodontal diseases and the importance of a tight control of bacterial plaque are basic concepts of periodontal treatment. In the past decade, there has been a conceptual shift from periodontal diseases as an oral problem to periodontitis having an impact on systemic health. Recent evidence suggests a strong relationship between periodontal inflammatory



disease and systemic diseases, such as cardiovascular disease. It is now generally accepted that inflammation plays an important role in atherosclerosis, and factors that systemically amplify inflammation are under close investigation. This article reviews some of the emerging concepts for the inflammatory mechanisms of periodontal diseases and atherosclerosis and examines the potential role of local inflammation in systemic inflammatory disease.

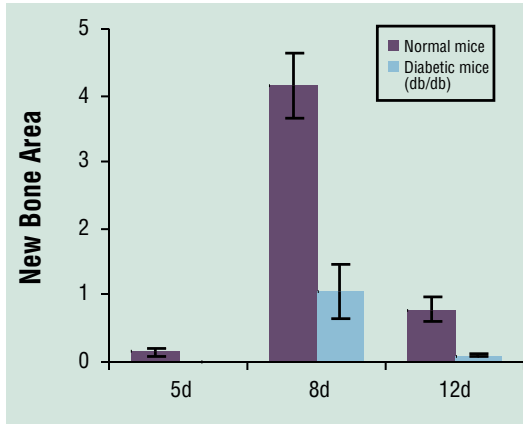
CE4 Diabetes Aggravates Periodontal Disease and Modifies the Response to an Oral Pathogen in Animal Models

Dana T. Graves, DDS, DMSc; Hesham Al-Mashat, DDS; Rongkun Liu, DDS, PhD

Bacterial plaque has been shown to initiate periodontal diseases. Most studies indicate that the host response, rather than the direct effect of bacteria, is responsible for much of the destruction associated with periodontitis. Bacteria or their products have an indirect role by stimulating inflammation, which is associated with the excessive production of inflammatory mediators, such as prostaglandins, or cytokines, such as tumor necrosis factor-alpha (TNF- α) and interleukin-1. These mediators, in turn, induce the production and activation of enzymes that destroy gingival connective tissue and stimulate the formation of osteoclasts to resorb bone. Based on results

in animal models and studies in humans showing that similar responses occur, the initial steps in the breakdown of connective tissue attachment to the tooth surface and bone resorption involve the production of inflammatory cytokines. Moreover, the risk and severity of periodontal diseases is affected by systemic factors, such as diabetes. Diabetes in particular seems to impair the ability to produce new bone formation after bone loss by preventing the formation of new bone that normally occurs after bone is resorbed, a process called coupling. In addition, the cytokines that stimulate loss of tissue, particularly TNF- α , may kill the cells that repair damaged connective

DIABETES IMPACT ON CAPACITY TO FORM NEW BONE



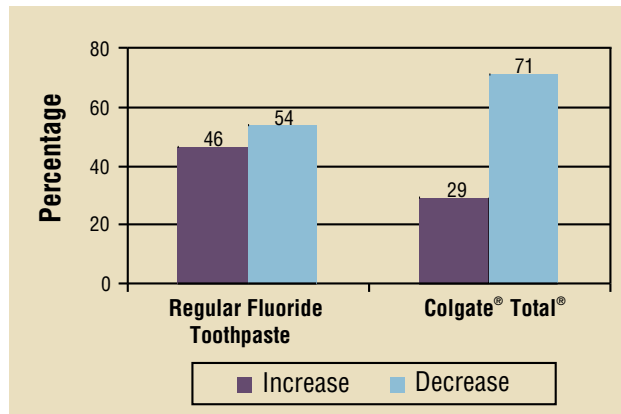
tissue or bone. In diabetes there may be more TNF- α produced, leading to an even more limited capacity to repair tissue. The diminished capacity to form new bone may make it more difficult for diabetics in particular to repair the loss of tissue that occurs in periodontal diseases.

Effectiveness of a Triclosan/Copolymer Dentifrice on Microbiological and Inflammatory Parameters

Tao Xu, DMD, PhD; Meenal Deshmukh, PhD; Virginia Monsul Barnes, DDS; Harsh M. Trivedi, MS; Diane Cummins, PhD

According to the US Surgeon General's report, "Oral Health in America," published in 2000, most adults in the United States show some degree of periodontal pathology, with severe periodontal diseases affecting about 14% of middle-aged adults. Periodontal diseases are polymicrobial-induced inflammatory diseases, and they vary from mild gingival inflammation to severe deterioration of the periodontium, i.e., loss of periodontal supportive tissues and, ultimately, tooth loss. New evidence shows that periodontal diseases may impact systemic health. For this reason, the maintenance of a healthy mouth is becoming increasingly important for the overall health of the body. This article summarizes laboratory research conducted during the development of a novel, multibenefit, oral-care technology based on triclosan — a broad-spectrum

COMPARISON OF EFFICACY PATTERN OF COLGATE® TOTAL® WITH A REGULAR FLUORIDE TOOTH PASTE ON ORAL PATHOGENS



antibacterial agent — and a polyvinylmethylether/maleic acid copolymer. This unique combination of agents is found in Colgate® Total®, a clinically proven efficacious dentifrice for control of dental plaque and gingivitis. Data are presented that demonstrate the unique antibacterial properties of this dentifrice: (1) a broad-spectrum antimicrobial profile; (2) the long-lasting retention of triclosan on hydroxyapatite and epithelial

cells; and (3) molecular evidence of antibacterial activity against specific pathogens in clinical dental plaque. In addition, data are presented that demonstrate the anti-inflammatory effects of triclosan on specific cytokines, the interruption of inflammatory pathways, and the inhibition of bone resorption. Overall, these data support the multibenefit clinical effects of Colgate® Total® and suggest a plurality of mechanisms of action.

Rationale for the Daily Use of a Dentifrice Containing Triclosan in the Maintenance of Oral Health

William DeVizio, DMD, Robin Davies, BDS, PhD

This paper provides an overview and summary of the data that demonstrate the effectiveness of a dentifrice containing triclosan/copolymer/fluoride in significantly reducing plaque, calculus, gingivitis, and the onset and progression of periodontitis. The caries-preventive benefit of the triclosan/copolymer/fluoride dentifrice was at least as good as that of a fluoride dentifrice. No evidence of bacterial resistance or the development of pathogenic or opportunistic bacteria was observed.



Recommend Colgate® Total® with confidence to your patients as

part of their normal

oral hygiene regimen. Recent studies indicate that the triclosan/copolymer formula found only in Colgate Total, delivers long-lasting antibacterial and antigingivitis effects for the prevention and control of gingival inflammation. Visit www.ColgateProfessional.com for the complete articles featured here or speak to your Colgate Representative for more information.