Journal of Dentistry & Oral Disorders

Open Access

Austin Publishing Group

Editorial

Could Only Bacteria Induce Periodontitis?

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Received: November 15, 2017; **Accepted:** November 28, 2017; **Published:** December 05, 2017

Editorial

This question is a challenge. Periodontal disease results from the association of the bacteria with many other predisposing risk factors. Although, bacteria are a critical etiologic factor that are needed to develop a periodontal disease, bacteria alone are insufficient to induce a periodontal disease; a susceptible host is also required, and the host's susceptibility as local and/or general predisposing risk factors are important determinants of the disease status The study of experimental gingivitis by the with-drawl of all forms of oral hygiene performed by Löe, et al. [1] was a landmark in the research of the bacterial etiology of gingival margin inflammation. These workers showed the pathogenic potential of normal oral commensal bacteria to induce gingivitis. Nevertheless, epidemiologic studies have clearly demonstrated that not all forms of gingivitis progress to periodontitis. Consequently, plaque-induced gingivitis is considered necessary, but insufficient cause of periodontitis. In the experimental gingivitis study, virtually all participants developed some degree of gingival inflammation in response to plaque accumulation for 11 to 21 days. However, gingivitis susceptibility varied considerably among the various individuals who harbored the similar bacterial complex. These data demonstrates that susceptibility to periodontal disease differs among diverse individuals and differs in various sites in the same individual, and sometimes differs among surfaces of a tooth. This fact may be due the multifactorial etiology of periodontal disease that requires an association among bacteria and diverse risk factors to induce the development of gingivitis. This study raises some questions: if all individuals of the study developed gingivitis after 11 to 21 days, why it was established that tooth brushing should be 3 times a day. If all individuals of the study developed gingivitis after 11 to 21 days, how long it would take for individuals to develop periodontitis. The answer is to induce experimental periodontitis in animals, but not asking the animals to stop brushing their teeth but introducing a predisposing risk factor to help bacteria to induce periodontitis as ligature physically tied on the tooth or inducing diabetes for example. Thereby, are we preventing the periodontal disease correctly, giving so much emphasis to the bacterial control? Which is easier to control bacteria or predisposing risk factors? The majority of all forms of periodontal diseases are considered as microorganisms induced dependent, which promotes a defensive inflammatory host's response against the bacteria and noxious materials from the bacterial plaque. The inflammatory process inactivates the bacteria, but produces the liberation of bacterial and neutrophils products such as enzymes, which induce periodontal tissue destruction by lytic activities. Could the sequels determined by defensive inflammatory host's response against bacteria and noxious materials from the bacterial plaque be considered as a disease, only because this defensive process as a side affect it produced a periodontal destruction? The majorities of infections induced by bacteria are normally controlled by the defensive inflammatory host's response sometimes aided by antibiotic therapy and usually heal without any complications. In periodontal disease the inflammatory process may prevent the penetration of bacteria into the periodontal tissues preventing septicemia, however, due to the particularities involved in the healing of the periodontal tissues, the sequels such as the formation of periodontal pockets remain propitiating a favorable environment for the growth of periodontopathogenic bacteria. In the early stages of periodontal wound healing, the wound stabilization and nutrition may be a critical variable to achieve regeneration after periodontal disease destruction. All healing wounds proceed through into three phases during the process of healing, following injury: inflammation, fibroblastic-granulation, and matrix formation and remodeling. In the sequencing of these events, during the early phase of healing, a fibrin clot is formed. In any wounds, this fibrin clot bridges the space between two vascular wound margins and serves as a base that epithelial cells migrate across to cover the wound, inducing protection to the underlying connective tissue as healing progresses. Periodontal disease produce wounds which should follow a similar healing pattern, but there are some significant differences in this specific wound healing environment that may affect the outcome of the healing. When periodontal wounds are healing, one of the wound margins is a vascularized connective tissue and the other is an avascular and rigid periodontitis-affected, contaminated and altered root surfasse which are separated from each other by the space of the periodontal pocket highly contaminated by the growth of exogenous anaerobic bacteria. This detail prevents or induces a contaminated fibrin clot formation which may not promote a normal healing. Despite causing periodontal tissue destruction, all types of periodontal disease are multifactorial diseases, which progress through successive destructive short acute phases, always interposed, by reparative long chronic phases. The indication of the periodontal disease reparative phase is possible to find in untreated periodontal pocket, as cementum and the gingival-attached connective tissue zone, separating the apical end of the ulcerated periodontal pocket epithelium from the underlying destructed alveolar bone which always presents a repaired cortical bone at a range of levels protecting the cancellous bone. In untreated periodontal disease, the gingival-attached connective tissue zone should be destructed, but they arise most often in untreated periodontal pockets. After each successive destructive acute phase, variations in the quantity and quality of the etiological agents and the predisposing risk factors would arise. However, periodontal diseases always cease at the destructive phase, initiating the reparative phase. This fact demonstrates that periodontal disease may be controlled by the host defense system, even considering that the etiological agents and predisposing risk factors during the periodontal disease progression, acquired better quality and quantity. However these

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increased predisposing risk factors associated with anaerobic exogenous bacteria which contaminated periodontal pocket, may not be able to maintain the periodontal disease destructive active phase all the time. Probably, the periodontal disease initiates and progress when at a given time, a specific temporary fragility, assists involved etiological agents in initiating the destructive acute phase of the periodontal disease. Then, are we treating the periodontal disease correctly? In the majority of periodontal surgical procedures, the treatment is applied when the reparative phase is ongoing, promoting formation of cortical bone inside the periodontal defect and repair of the cementum and gingival attached connective tissue zone, which separates the underlying destructed alveolar bone from the apical end of the ulcerated periodontal pocket epithelium. These repaired structures are responsible for separating infected periodontal pockets from the intra-marrow alveolar bone/periodontal ligament and seem to be the main barrier against progression of the destructive periodontal disease [2]. Because of this reparative process, in order to augment healing of periodontal defects, several bone graft surgical procedures require repaired cortical bone and gingival attached connective tissue mechanical debridement, to induce an intra-marrow and periodontal ligament mesenchymal stem cells penetration into periodontal bone defects. These facts lead us for more questions questions: Only by controlling bacteria would we be able to prevent periodontal disease? Bacteria alone could trigger periodontal disease? What is the best time to treat periodontal disease? What would be the best method to diagnose the active phase of periodontal disease to treat it at lower levels? We are researching focusing on these questions trying to find answers to better understand the periodontal disease [2-5].

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