Trends in Medicine

Editorial

Could only bacteria induce periodontitis?

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This question is a challenge. Many researches accept that periodontitis are considered as microorganisms-induced dependent. The majority if not all consider that to prevent periodontitis is necessary an efficient biofilm control. However, periodontitis is a disease dependent of the multifactorial etiologic agents, were, bacteria play an essential role but is improbable that bacteria alone are sufficient to induce periodontitis. We cannot find a blunt research, showing that only seeding periodontopathogenic bacteria in a heathy periodontium may cause experimental periodontitis. Most periodontitis induces local destruction, essentially due to the involvement between the opportunist bacteria and the various and inherent local predisposing risk factors. The local predisposing risk factor may provoke a mechanical vulnerability in the periodontal tissues around a tooth, and/or may assist in bacterial retention, development and organization, in order to originate periodontal tissue destruction. Generalized periodontal disease may arise, when the systemic predisposing risk factor interferes with the host's defence mechanism against the opportunist bacteria, once the host factor, operates in all periodontal tissues. The predisposing risk factor may statistically increase the occurrence of a disease, but it does not cause the disease. Periodontitis promote junctional epithelium ulceration, apical displacement of the gingival attachment, alveolar bone loss and periodontal pocket development, an increase in the depth of gingival sulcus, thereby, creating a favourable anaerobic environment to be infected, as a result of the recurrent contamination by the several species or the combination of the species as exogenous anaerobic and facultative bacteria which are considered as periodontophatogenic bacteria. These periodontophatogenic bacteria could iniciate periodontistis? This is improbable, periodontopathogenic bacteria need of a favorable anaerobic environment as periodontal pocket to grow and multiply. Since most periodontitis induces local destruction, is interesting that we can find only periodontopathogenic bacteria at destructed periodontal tissue presenting periodontal pocket and endogenous bacteria at neighbouring dental sites. Why does this happen? Periodontopathogenic bacteria cannot contaminate adjacent health areas? Maybe to iniciate periodontitis is necessary the association between endogenous bacteria and predisposing risk factors. Then if bacteria cannot iniciate periodontitis alone, why the management to prevent periodontitis is bacterial control? It would not be more logical also to diagnose and eliminate or/and establish a control in all predisposing risk factor? It is impossible to eliminate all bacteria from the mouth, but it is possible to establish effective control over most predisposing risk factors. Moreover the study of experimental gingivitis by the with-drawl of all forms of oral hygiene performed by Löe, *et al.*, 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 harboured 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 toothbrushing 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, not asking the animals to stop brushing their teeth but introducing a predisposing risk factor to help bacteria to induce periodontistis as ligature physically tied on the tooth 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? Maybe both.

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