Innate Immune Signaling and Porphyromonas gingivalis accelerated atherosclerosis.

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Abstract

Periodontal diseases are a group of diseases that lead to erosion of the hard and soft tissues of the periodontium, which, in severe cases, can result in tooth loss. Anecdotal clinical observations have suggested that poor oral health may be associated with poor systemic health; however, only recently have appropriate epidemiological studies been initiated, with defined clinical endpoints of periodontal disease, to address the association of periodontal disease with increased risk for cardiovascular and cerebrovascular disease. Although conflicting reports exist, these epidemiological studies support this connection. Paralleling these epidemiological studies, emerging basic scientific studies also support that infection may represent a risk factor for atherosclerosis. With P. gingivalis as a model pathogen, in vitro studies support that this organism can activate host innate immune responses associated with atherosclerosis, and in vivo studies demonstrate that this organism can accelerate atheroma deposition in animal models. In this review, we focus primarily on the basic scientific studies performed to date which support that infection with bacteria, most notably P. gingivalis, accelerates atherosclerosis. Furthermore, we attempt to bring together these studies to provide an up-to-date framework of emerging theories into the mechanisms underlying periodontal disease and increased risk for atherosclerosis, as well as identify intervention strategies to reduce the incidence of periodontal disease in humans, in an attempt to decrease risk for systemic complications of periodontal disease such as atherosclerotic cardiovascular disease.