Innate immune recognition of invasive bacteria accerlerates atherosclerosis in apolipoprotein E-deficient mice.

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Abstract

Background— Infectious diseases have emerged as potential risk factors for cardiovascular disease (CVD). Epidemiological studies support a connection between periodontal disease, a chronic inflammatory disease of the supporting tissues of the teeth, and CVD.

Methods and Results— To directly test the connection between periodontal disease and atherosclerosis, apoE–/– mice were orally challenged with the periodontal disease pathogen Porphyromonas gingivalis or an invasion-impaired P gingivalis fimbriae-deficient mutant (FimA–). Both wild-type P gingivalis and the FimA– mutant were detected in blood and aortic arch tissue of apoE–/– mice by PCR after challenge. ApoE–/– mice challenged with wild-type P gingivalis presented with increased atherosclerotic plaque and expressed the innate immune response markers Toll-like receptor (TLR)-2 and TLR-4 in aortic tissue. Despite detection of the FimA– mutant in the blood and in aortic arch tissue, apoE–/– mice challenged with the FimA– mutant did not present with periodontal disease, upregulation of TLRs, or accelerated atherosclerosis. Furthermore, we demonstrate that immunization to control P gingivalis-elicited periodontal disease concomitantly prevents P gingivalis-accelerated atherosclerosis.

Conclusions— We conclude that invasive P gingivalis accelerates atherosclerosis.