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# *Porphyromonas gingivalis*
## A heartful oral pathogen?

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Periodontal diseases have long been associated with an increased, yet moderate, risk for the development of atherosclerosis and coronary heart disease.[1-3] The postulated mechanism underlying this association is the low grade systemic inflammatory response to pathogens engaged in chronic periodontal infection, such as *Porphyromonas gingivalis*, which may facilitate atheroma formation and thromboembolic events.[4] The vasculature may be affected either by direct interaction of the microbiota with the endothelial walls, or by the induction of acute phase response mediators, such as the C-reactive protein, or interleukin-6. The periodontal infection and its contribution to systemic inflammation cannot be held alone responsible for inducing cardiovascular disease, but can act as modifiers of existing risks, exposures, and disease outcomes.[5] For instance, recent epidemiological evidence suggests that periodontal disease may potentiate the risk for the subclinical development of atherosclerotic plaque calcification and coronary heart disease in diabetics.[6] Interestingly, periodontal therapy can, in long-term, reduce the systemic levels of inflammatory mediators, and improve endothelial function and flow-mediated dilation,[7-9] highlighting the association between periodontal and cardiovascular diseases.

*Porphyromonas gingivalis*, a major opportunistic periodontal pathogen[10] has been at the "heart" of the studies attempting to decipher this association. To understand better its role, several investigations have looked into its presence in atheromatous plaques or heart valves, primarily using polymerase chain reaction in specimens obtained during planned surgery. Atheromatous plaques from coronary arteries of patients with chronic periodontitis can harbor universal bacterial DNA, at 100% prevalence.[11] *P. gingivalis* DNA proved to be the most prevalent among periodontal pathogens, detected in as many as 50% of the cases.[11,12] A clear association was noticed between its presence in periodontal pockets and atheromatous tissues, but this was not the case for other oral bacteria.[11] The presence of *P. gingivalis* in atheroma samples demonstrates that this species can indeed find its way from the subgingival biofilm and onto the walls of coronary heart arteries, during the course of a transient bacteremia. Yet, it should be borne in mind that the assays used to detect its presence largely rely on the detection of its DNA, and do not provide tangible evidence of an active colonization of the coronary artery tissue.

When considering the heart valves, specimens from aortic or mitral valves were also analyzed for bacterial DNA, again by polymerase chain reaction, for various oral species. Most frequently found was *Streptococcus mutans*, followed by *Aggregatibacter actinomycetemcomitans*, with a positive correlation for dental plaque detection, particularly in the case of the former. However, *P. gingivalis* was only detected in approximately 10% of the specimens. The detection of these oral species on heart valves was attributed to bacteremic events.[13] Nevertheless, another study detected none of the eight periodontal pathogens tested, including *P. gingivalis*, in whole blood and stenotic aortic valves, indicating absence from blood stream and lack of colonization of the valves.[14] The study from Radwan-Oczko et al., featured in the present issue of *Virulence*, employed a patient population with calcific heart valve degeneration and mild periodontitis.[15] They also did not detect *P. gingivalis* in any aortic or mitral valve specimens tested, although this species was present in 50% of the periodontal pockets of the corresponding patients.

Collectively, the literature indicates that *P. gingivalis* can be present in atherosclerotic plaques, potentially finding its way there during transient bacteremia, but evidence of active colonization of the heart valves is still rather weak. Conventional bacterial culture assays should be used to verify this, while the indirect contribution of *P. gingivalis* to heart valve degeneration via induction of chronic systemic inflammatory responses should be further considered.

### Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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