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# CUPID AND THE TOOTH FAIRY: THE LINK BETWEEN CARDIOVASCULAR DISEASE AND ORAL HEALTH

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The presence of a link between periodontal and cardiovascular disease is supported by epidemiological publications and studies of biological mechanism. Although still unproven, a causal relationship is attractive for several reasons. First, it fits with current thinking about atherosclerosis as an inflammatory disorder and the contribution from infective processes. Second, it suggests that current cardiovascular risk assessment could be improved by dental examination. Third, and of great potential clinical importance, prevention and treatment of periodontal disease could reduce cardiovascular disease (CVD).

Recent prospective cohort and case-control analyses by Beck and Loesche and their colleagues support this relationship. Le Beck analysed 1,147 men from the US Normative Aging and the Dental Longitudinal studies. Mean alveolar bone loss scores were significantly associated with total coronary heart disease (odds ratio 1.5), fatal coronary heart disease (1.9) and stroke (2.8). In a case-control study of 320 veterans who were either seen at a dental outpatient clinic (n = 206) or in a long-term nursing home (n = 114), Loesche found a statistically significant association between coronary heart disease and

several oral health parameters, such as the number of missing teeth, plaque benzoyl-DL-arginine-naphthylamide (BANA) test scores and salivary levels of *Streptococcus sanguis*.<sup>2</sup>

In epidemiological studies such as these, questions arise regarding potential confounders. Besides traditional risk factors such as age, cigarette smoking and diabetes mellitus that are common to both dental and CVD, both conditions are modified by factors such as level of education, income, stress and social isolation. Thus, the associations may primarily reflect the fact that people who are more health conscious are at lower risk of coronary heart disease and have better dental health. The decision to extract teeth, in addition to the condition of the teeth, is also influenced by financial considerations and access to health care, as well as attitudes about the value of oral health. Since these behaviours and attitudes are hard to measure, controlling for them is difficult and surrogates for healthy behaviour (for example, level of physical activity) should be considered. Beck and colleagues included level of education in their analytic models, along with age, body mass index, smoking status, blood pressure, family history of heart disease, cholesterol and alcohol consumption. Significant associations remained in the study by Beck as well as others that provided adjustment models, although questions remain as to how complete such adjustments are.

Diet is another possible explanation for the relationship between tooth loss and CVD. While a poor diet rich in sugar may lead to both periodontal disease and CVD, tooth

Vol. 10 No. 5 47

loss can then lead to harmful changes in diet, including reduced intake of fibre, fruit and vegetables and a higher sweet intake. Dietary data are difficult to report and were not included in the analyses by Beck and Loesche. Other unaccounted dietary factors such as cooking or processing could also impact on both periodontitis and CVD. Tooth loss may also lead to poor self-image and psychological stress that increases risk of CVD. Cigarette smoking is increasingly accepted as a risk factor for periodontitis and has been shown to affect different aspects of the host immune response. While the Beck and Loesche studies considered smoking history, their analyses may fail to fully address the harmful role of nicotine with regard to both dental and CVD. Unmeasured genetic factors may also predispose to both disease processes.

While a cause and effect relationship remains uncertain,<sup>3,4</sup> there are compelling biological links between infection and inflammation, and increased cytokine activity, monocyte activity, increased lipopolysaccharide activity and procoagulant changes.<sup>5</sup> Periodontal disease provides a strong and chronic stimulus to these inflammatory responses that are associated with atherosclerosis.

It is important that further prospective cohort and casecontrol studies are undertaken in both men and women that carefully take into account potential confounders. Clinical studies should be performed to determine whether improved periodontal care will reduce fibrinogen, C-reactive protein, other inflammatory factors and other intermediate markers for cardiovascular risk factors.<sup>6</sup> Although difficult to construct, randomised clinical trials will be needed to address the relationship between periodontal disease and CVD more completely. For many reasons, better self-care and professional care to minimise periodontal disease is recommended. When teeth have to be extracted, adequate prostheses and counselling to maintain a good diet are needed. If the possible link between dental disease and CVD can be more firmly established, it will be exciting to contemplate the opportunity to reduce the risk of CVD through improved dental care.

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48 Vol. 10 No. 5