Evidence-Based Dentistry (2000) 2, 59–60

www.nature.com/ebd

editorial

Cause célébre: oral health and heart disease

Richard Niederman[1,2] and Kaumudi Joshipura[3,4]

[1]Office of Evidence-Based Dentistry Harvard School of Dental Medicine, Boston, USA; [2]Office of Evidence-Based Dentistry, Forsyth Institute, Boston, USA; [3]Oral Health Policy and Epidemiology, Harvard School of Dental Medicine, Boston, USA; and [4]Department of Epidemiology, Harvard School of Public Health, Boston, USA

The recent series of articles in the *BMJ* and *JAMA* that examine the putative relationship between oral health and cardiovascular disease are impressive reminders of the value of high levels of evidence and evidence-based health care. They are also practical reminders that we floss, but we die anyway.

The recent series of articles in the *BMJ*[1–3] and *JAMA*,[4] all re-examining the putative relationship between systemic infections and coronary heart disease, are heart-stopping. The reports indicate that infections (*Chlamydia* infections[1–3] and periodontal disease[4]) may not, as previously thought, increase the risk of coronary heart disease. This raises concerns about widely distributed reviews implying that periodontal infection is a risk factor for heart disease (and by analogy, pre-term low birthweight, diabetes and respiratory disease[5–7]) and about the treatment of this issue by the lay press.[8–10]

As reported elsewhere in this issue of *Evidence-Based Dentistry*, establishing a causal relationship is not a trivial matter, generally requiring the fulfilment of a number of lines of evidence initially proposed by Bradford Hill. Clinically, a hierarchy of levels of clinical evidence has been defined (Centre for Evidence-based Dentistry: cebm . jr2 . ox . ac .uk/docs/levels.html). Conceptually, 'high' levels of evidence are prospective and randomised or balanced, whereas lower levels of evidence are retrospective or cross-sectional.

To determine the quality level and number of publications evaluating the causal relationship between periodontal disease and cardiovascular disease, we carried out a bibliometric analysis of the literature cited on MEDLINE from 1989 when Mattila *et al*[11] first reported a relationship between periodontal disease and cardiovascular disease. A search strategy was developed and implemented (Table 1) using the Internet Ovid interface (Gateway.ovid.com). As indicated, the search identified 321 articles (step 6) addressing oral health and cardiovascular disease. Of these, approximately one-third (119) addressed humans, and were published in English between 1989 and 2000 (step 8). Examination by hand of the titles and abstracts indicated that there were 14 original publications and 17 review articles that directly addressed the potential relationship between oral health and coronary heart disease (Table 2). The remainder examined other issues.

These results are interesting from several perspectives. First, there were more review articles than original research articles. Second, of the original articles, six provided high levels and eight provided low levels of evidence (Centre for Evidence-based Dentistry: cebm.jr2.ox.ac.uk / docs / levels.html). Third, one wonders whether the excitement evolved to a cause célébre prior to the demonstration of causal relationship.

More concrete evidence comes from the meta-analyses of Danesh[12], which

Table 1 Search strategy and results for 1989–2000 (October 2000, week 4)

Step	Search term	No. of articles
1	Explode periodontal diseases	38,150
2	Explode dental caries	22,393
3	Oral health	4453
4	1 or 2 or 3	60,678
5	Explode heart diseases/	442,707
6	4 and 5	321
7	Limit 6 to the years 1989–2000	139
8	Limit 7 to (human and English)	119

Table 2 Number of Publications and Level of Evidence

Type of study	Number of publications	Level of evidence
Primary		
Meta-analysis	1	High
Cohort	5	High
Case-control	4	Low
Cross-sectional	4	Low
Secondary		
Review	17	Low

found a small but significant association between periodontal disease and coronary heart disease, but from which the author concluded the relationship can be attributed to inadequate control of social class *in the absence of a causal relationship*. (A structured abstract and clinical commentary of the study appears in this issue of *Evidence-Based Dentistry*.) The Danesh[14] finding is consistent with the much earlier analysis of Joshipura *et al*[15] who found no relationship between periodontal disease and coronary heart disease. This was in a homogeneous population of 40 000 health professionals, which inherently controlled for social class, income and related behavioural factors.

Now, corroborating the initial 1996 findings of Joshipura *et al*[13] come the studies of Morrison *et al*. (1999)[14] and, ironically, Mattila *et al*,[15] whose seminal work partly stimulated the spasm of activity examining these relationships. (Again, a structured abstract and clinical commentary of Matilla *et al*[15] appears in this issue of *Evidence-Based Dentistry*). To these can now be added the publication from Hujoel *et al*.[4]

Are these findings disheartening, or an indication of a cause célébre? Perhaps both, or neither, depending upon one's viewpoint. The initial findings stimulated multiple reports in the lay press, increasing public awareness, clinical visits and grant funding – all presumably worthy outcomes. The current findings, however, may occlude the flow of goodwill. Will they be as widely publicised as the initial exciting findings? Or, more problematically, generate ridicule?

In any of these occurrences, the current findings are cogent reminders of the investment benefits of evidence-based health care. They remind us that demonstrating causality requires consistent, high-quality studies.

1. Danesh J, Whincup P, Walker M, Lennon L, Thomson A, Appleby P, *et al*. Low grade inflammation and coronary heart disease: prospective study and updated meta-analyses. BMJ 2000; 321:199–204.
2. Danesh J, Whincup P, Walker M, Lennon L, Thomson A, Appleby P, *et al*. *Chlamydia pneumoniae* IgG titres and coronary heart disease: prospective study and meta-analysis BMJ 2000; 321:208–213.
3. Wald NJ, Law MR, Morris JK, Zhou X, Wong Y, Ward ME. *Chlamydia pneumoniae* infection and mortality from ischaemic heart disease: large prospective study. BMJ 2000; 321:204–207.
4. Hujoel PP, Drangsholt M, Spiekerman C, DeRouen TA. Periodontal disease and coronary heart disease risk. JAMA 2000; 284:1406–1410.
5. Anonymous. Position paper. Periodontal disease as a potential risk factor for systemic diseases. J Periodontol 1998; 69:841–850.
6. Paquette DW, Madianos P, Offenbacher S, Beck JD, Williams RC. The concept of risk and emerging discipline of periodontal medicine. J Contemporary Dent Prac 1999; (www.thejcdp.com/issue001/index.htm).
7. Mealey BL. Influence of periodontal infections on systemic health. Periodontology 2000; 21:197–209.
8. Brody J. Gum disease in pregnancy linked to premature low-weight babies. NY Times 1996; October 9.
9. Grady D. Linking infection to heart disease. NY Times 1998; February 17.
10. Brody J. Flossing protects far more than the teeth and gums. NY Times 1999; January 19.
11. Mattila KJ, Nieminen MS, Valtonen VV, Rasi VP, Kesaniemi YA, Syrjala SL, et al. Association between dental health and acute myocardial infarction. BMJ 1989; 298(6676):779–781.
12. Danesh J. Coronary heart disease, *Helicobacter pylori*, dental disease, *Chlamydia pneumoniae*, and cytomegalovirus: meta-analyses of prospective studies. Am Heart J 1999; 138(5 pt2):S434–S437.
13. Joshipura KJ, Rimm EB, Douglass CW, Tirichopoulos D, Ascherio A, Willett WC. Poor oral health and coronary heart disease. J Dent Res 1996; 75:1631–1636.
14. Morrison HI, Ellision LF, Taylor GW. Periodontal disease and risk of fatal coronary heart and cerebrovascular diseases. J Cardiovasc Risk 1999; 6:7–11.
15. Matilla KJ, Asikainen S, Wolf J, Jousimies-Somer H, Valtonen V, Nieminen M. Age, dental infections, and coronary heart disease. J Dent Res 2000; 79:756–760.

Editor's note

Commentaries in EBD

The commentaries presented in the summary section of the journal are invited from individuals with specialised knowledge of the field from which the original study is drawn.

Commentaries aim to draw out the main features of the study whether these are positive or negative, and place them in to the overall context of that particular field. The commentators are also asked to address the practical implications for the dental practitioner.

The overall aim is to provide an overview of the best available dental research in an easily digestible form for the general dentist in a busy practice.

While we endeavour to be as fair and balanced as possible in the content of the summaries it is important to remember that they are written by individuals.

As individuals we all bring our own individual biases and interpretations to whatever we read and write. Because of this it is important to remember that appraising the original article oneself is the best way of integrating clinical evidence with external evidence on the way to deciding whether one should change one's approach to clinical care.