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Gingivitis, dental caries and tooth loss: risk factors for cardiovascular diseases or indicators of elevated health risks

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Abstract

Objectives: The results of earlier studies connecting dental diseases to cardiovascular diseases are inconsistent. Our aim in this cross-sectional study was to investigate whether there are associations of dental diseases and diagnosed angina pectoris among the 1966 Northern Finland Birth Cohort.

Materials and Methods: A postal questionnaire was sent to all cohort members in 1997–1998. The number of replies totalled 8690. Angina pectoris was determined by asking whether the respondent had been diagnosed with angina pectoris. Gingivitis, dental caries and tooth loss were determined on the basis of self-reported gingival bleeding, presence of dental caries and six or more missing teeth.

Results: We found overall associations of gingivitis (odds ratio (OR) 1.52, confidence interval (CI) 1.04–2.22), dental caries (OR 1.50, CI 1.04–2.18) and tooth loss (OR 1.53, CI 0.69–3.42) with the presence of angina pectoris. The associations were modified by gender and socioeconomic status. In addition, gingivitis, dental caries and tooth loss were also associated with several cardiovascular risk factors.

Conclusion: There were associations of self-reported gingivitis, dental caries and tooth loss with angina pectoris. However, the associations between dental diseases and cardiovascular risk factors suggest that the associations may be because of confounding.

Key words: cardiovascular diseases; dental diseases; young adults

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Most research on the relation between dental diseases and cardiovascular diseases (CVD) has focused on the possible relation of periodontitis with CVD. An association between periodontitis and CVD has been found in several studies (DeStefano et al. 1993, Morrison et al. 1999, Wu et al. 2000). Studies also exist that have shown an association between gingivitis and CVD (Lösche et al. 1998, Bazile et al. 2002, Buhlin et al. 2002b), but there are, likewise, studies that have given negative or statistically insignificant findings (DeStefano et al. 1993, Hujoel et al. 2000, 2002a, Wu et al. 2000). An association between dental caries and cardiovascular risk factors has also been found (Johansson et al. 1994, Larsson et al. 1995).

The nature of the relation of periodontitis and gingivitis to CVD is not known. The results of observational studies are inconsistent and there are no intervention studies demonstrating that periodontal treatment would cause reduction in CVD morbidity. One reason for the inconsistent findings could be that there is no causal relation between periodontal diseases and CVD, or that the association is weak compared with established strong risks such as smoking, unhealthy diet and insufficient physical exercise. Inconsistency in results may also stem from different outcome variables, differences in the measurement of the variables, a different study design, different types of study population and different covariates.

It has previously been suggested that positive findings may be – at least partly – because of confounding related to health awareness (Hujoel 2002) and health behaviour, especially smoking (Scott et al. 2001, Hujoel et al. 2002b, Spiekerman et al. 2003). In a situation where strong

risk factors and a possible weak risk factor exist, it is important to measure confounders accurately, as confounders that have been misclassified or left uncontrolled may cause quantitative or qualitative errors in such a situation.

Research has shown that those with periodontal diseases have higher levels of inflammatory markers than those who are periodontally healthy (Noack et al. 2001, Ebersole et al. 2002). This suggests that periodontal diseases could be an aetiological factor in the pathogenesis of atherosclerosis. Moreover, it has been found in several intervention studies that periodontal treatment may decrease the levels of inflammatory markers (Mattila et al. 2002, D'Aiuto et al. 2004, 2005, Montebugnoli et al. 2005). However, at present, there is no evidence on the effect of periodontal treatment on cardiovascular morbidity and mortality, meaning that definite evidence is lacking.

Angina pectoris is a diagnosis made in a patient who has chest pain of cardiac origin. It can occur either at rest or during physical activity, depending on the severity of the underlying disease, which, in most cases is a coronary hearth disease. Our aim in this cross-sectional study was to investigate whether gingivitis, dental caries and tooth loss are associated with diagnosed angina pectoris among young adults.

Material and Methods

The Northern Finland Birth Cohort covered 96% of all births in 1966 ($n = 12{,}058$) in the provinces of Lapland and Oulu (Rantakallio 1988). The present study is based on a postal questionnaire sent to members of the cohort between 1997 and 1998, when the cohort reached the age of 31 ($n = 11{,}541$). In the postal questionnaire, respondents were asked about their general health, oral health and health habits. The number of replies totalled 8690, giving a response rate of 75.3%. Those who lived in Northern Finland and in the Helsinki region ($n = 8463$) were invited to a health examination where laboratory tests were taken. The participation rate was 71% ($n = 6033$).

The subjects participated in a 31-year follow-up of this cohort after providing informed consent. A study protocol for the 31-year follow-up of the cohort was reviewed and approved by the Ethics Committee of the Faculty of Medicine, Oulu University.

Outcome variables

Self-reported angina pectoris diagnosed by a doctor was used as an outcome variable. This was determined on the basis of the following question: ''Have you ever had angina pectoris diagnosed by a doctor?''

Explanatory variables and potential confounding factors

Gingivitis was determined on the basis of the following question: ''In your opinion, do your gums bleed when you brush your teeth?'' (No/Yes). Self-reported caries was determined on the basis of the following question: ''In your opinion, do you have caries in your teeth at the moment?'' (No/Yes). In the questionnaire, the respondents were also asked how many missing teeth they had (0, 1–5, 6–10, more than 10 but not all, all). Respondents were classified into two categories according to the number of missing teeth (0–5 teeth missing *versus* six or more teeth missing).

Elevated blood pressure was determined in a manner similar to the one described above. ''Have you ever had elevated blood pressure diagnosed by a doctor?'' The body mass index (BMI) (under 25 *versus* 25 or more) was based on self-reported measurements of weight and height.

Blood samples were taken after an overnight fast. Serum total cholesterol, serum high-density lipoprotein (HDL) cholesterol, serum triglycerides and C-reactive protein (CRP) were determined by using standard enzymatic methods. Categorizations of serum cholesterol, HDL cholesterol and triglycerides were based on the scientific statement of American Heart Association (AHA) and American College of Cardiology (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Executive Summary of the third Report of the National Cholesterol Education Program (NCEP) 2001, Smith et al. 2001). Categorization of CRP were based on the statement of AHA and Centers for Disease Control and Prevention (Pearson et al. 2003). Leucocytes were categorized according to approximate tertiles.

The amount of tobacco smoked (packyears) was calculated for those who smoked regularly almost every day. The smokers were classified into three categories on the basis of packyears (0–5, 6–10, 11 or more). Non-frequent smokers and non-smokers each formed a category of their own.

Physical exercise was determined by the frequency of physical exercise: more than three times a week *versus* less. Consumption of vegetables was determined similarly: at least three times a week *versus* less.

The socioeconomic status (SES) of the respondents was measured by means of the respondents' educational level and income. Education was classified into four categories: education in university or in higher educational institutions, vocational education, comprehensive school only and other. Income was measured by gross income in Finnish marks per adult member of a family. Incomes were classified into four categories (0–49,999; 50,000–99,999; 1,00,000–1,99,999; and 2,00,000 and above).

Psychological characteristics were measured using coping strategies (active and passive coping), optimism and life satisfaction. There are two basic dimensions to the ways of coping: active coping (problem-focused coping), where a person who has adopted an active coping approach tries to change a situation into a more favourable one by taking action themselves, and passive coping (emotion-focused coping), where a person applying a passive coping strategy avoids stressful situations and emotions related to it. Active coping and passive coping were measured using the Ways of Coping Checklist (Lazarus & Folkman 1984). Test scores were classified into four categories based on distribution.

Optimism was measured using the revised version of the Life Orientation Test (LOT-r) (Scheier et al. 1994). The test assesses individual differences in generalized outcome expectancies, positive expectancies being associated with optimism and negative ones with pessimism. Respondents were asked to rate how well they agreed with six items across a five-point Likert's-type scale. Test scores were classified into four categories based on distribution.

Life satisfaction was measured by means of a single question: ''Are you satisfied with your life in general?'' The response alternatives were: (1) very satisfied, (2) quite satisfied, (3) quite dissatisfied, (4) very dissatisfied and (5) don't know. The responses of the ''quite dissatisfied'' and ''very dissatisfied'' were classed together as there were very few of them.

Statistical methods

We used generalized linear models in the analyses. We chose a binomial distribution with a logit link function (logistic regression). The data were analysed using the SAS GENMOD procedure, version 8.02. The results are presented in the form of odds ratios (ORs) with 95% confidence intervals (CIs).

Results

There were differences in several of the characteristics between those who reported gingivitis and those who reported no gingivitis, between those who reported a tooth loss of six or more teeth and those who reported less than six missing teeth and between those who reported dental caries *versus* no caries (Table 1). In fact, those who reported dental diseases and symptoms were overrepresented in almost all risk factor or risk indicator categories related to psychosocial well-being, general health, dental health and health habits.

The associations of explanatory factors and potential confounders with angina pectoris are presented in Table 2, from which it can be seen that

Table 1. Descriptive characteristics of the study population

	Gingival bleeding ($n = 2016$) (%)	No gingival bleeding ($n = 6385$) (%)	p-value*
Health habits			
Smoking, never-smokers (%)	35.0	37.2	0.0878
Physical exercise at least four times a week (%)	42.5	50.1	<0.0001
Alcohol use more than three to four drinks at a time (%)	51.2	41.4	<0.0001
Tooth brushing at least twice a day (%)	39.8	59.1	<0.0001
Dental check-ups once in 2 years (%)	59.8	71.8	<0.0001
Use of sugar in coffee or tea (%)	51.6	56.6	0.0001
Use of vegetables at least three times a week (%)	47.8	59.8	<0.0001
Dental health			
Self-reported caries (%)	49.5	30.5	<0.0001
Tooth loss six or more teeth (%)	3.1	2.2	0.0204
Oral pain (%)	25.2	13.6	<0.0001
General health			
Body mass index 25 or above (%)	43.1	34.4	<0.0001
Diagnosed angina pectoris (%)	2.6	1.4	0.0003
Diagnosed elevated blood pressure (%)	14.1	12.1	0.0149
Inflammatory markers (means)†			
Total cholesterol (mmol/l)	5.14	5.06	0.0063
High-density lipoprotein cholesterol (mmol/l)	1.54	1.56	0.0600
Triglycerides (mmol/l)	1.26	1.17	<0.0001
C-reactive protein (mg/dl)	2.20	1.97	0.0481
Leucocytes (10e9/l)	6.01	5.92	0.0754
Psychological well being			
Life satisfaction			
Proportion of unsatisfied persons (%)	12.6	8.2	<0.0001
Proportion of very satisfied persons (%)	18.1	22.9	<0.0001
Optimism			
Proportion of very optimistic persons (%)	18.4	26.3	<0.0001
Socioeconomic factors			
Income: subjects in two highest income brackets (%)	39.7	45.7	<0.0001
Education: at least college-level education (%)	38.5	47.6	<0.0001
Gender: proportion of males (%)	*53.9*	*45.6*	*<0.0001*
Marital status: married or co-habiting (%)	70.5	73.6	0.0063

*p-values derived from the χ^2 test for categorical variables and from the t-test for difference of means.
†Means are based on a subsample of 5,690.

	Tooth loss (six or more teeth missing) ($n = 201$) (%)	No tooth loss (zero to five teeth missing) ($n = 8208$) (%)	p-value*
Health habits			
Smoking, never-smokers (%)	21.8	37.1	<0.0001
Physical exercise at least four times a week (%)	33.8	48.6	<0.0001
Alcohol use more than three to four drinks at a time (%)	59.8	43.5	<0.0001
Tooth brushing at least twice a day (%)	31.6	54.8	<0.0001
Dental check-ups once in 2 years (%)	54.1	70.8	<0.0001
Use of sugar in coffee or tea (%)	57.8	55.4	0.5239
Use of vegetables at least three times a week (%)	37.0	57.3	<0.0001
Dental health			
Self-reported caries (%)	51.5	34.9	<0.0001
Gingival bleeding (%)	31.0	23.8	0.0204
Oral pain (%)	20.8	16.3	0.0999

Table 1. (Contd.)

	Tooth loss (six or more teeth missing) ($n = 201$) (%)	No tooth loss (zero to five teeth missing) ($n = 8208$) (%)	p-value*
General health			
Body mass index 25 or over (%)	42.1	36.5	0.1091
Diagnosed angina pectoris (%)	4.5	1.7	0.0023
Diagnosed elevated blood pressure (%)	20.5	12.3	0.0005
Inflammatory markers (means)†			
Total cholesterol (mmol/l)	5.16	5.08	0.3291
High density lipoprotein-cholesterol (mmol/l)	1.49	1.56	0.0491
Triglycerides (mmol/I)	1.34	1.19	0.0218
C-reactive protein (mg/dl)	2.01	2.01	0.9898
Leucocytes (10e9/l)	6.20	5.93	0.0768
Psychological well being			
Life satisfaction			
Proportion of unsatisfied persons (%)	15.5	9.0	0.0018
Proportion of very satisfied persons (%)	18.5	21.9	0.2553
Optimism			
Proportion of very optimistic persons (%)	17.0	24.7	0.0141
Socioeconomic factors			
Income: subjects in two highest income brackets (%)	30.9	44.5	<0.0001
Education: at least college-level education (%)	25.0	45.8	<0.0001
Gender: proportion of males (%)	*62.3*	*47.3*	*<0.0001*
Marital status: married or co-habiting (%)	65.2	73.0	0.0137

*p-values derived from the χ^2 test for categorical variables and from the t-test for difference of means.
†Means are based on a subsample of 5,690.

	Self-reported caries ($n = 2016$) (%)	No self-reported caries ($n = 6385$) (%)	p-value*
Health habits			
Smoking, never-smokers (%)	30.4	40.0	<0.0001
Physical exercise at least four times a week (%)	43.6	50.9	<0.0001
Alcohol use more than three to four drinks at a time (%)	51.8	39.5	<0.0001
Tooth brushing at least twice a day (%)	43.9	60.0	<0.0001
Dental check-ups once in 2 years (%)	52.6	77.6	<0.0001
Use of sugar in coffee or tea (%)	48.3	42.6	<0.0001
Use of vegetables at least three times a week (%)	48.3	61.6	<0.0001
Dental health			
Gingival bleeding (%)	33.9	18.6	<0.0001
Tooth loss six or more teeth (%)	3.5	1.8	<0.0001
Oral pain (%)	28.1	10.1	<0.0001
General health			
Body mass index 25 or over (%)	41.6	33.8	<0.0001
Diagnosed angina pectoris (%)	2.5	1.3	0.0001
Diagnosed elevated blood pressure (%)	13.1	12.3	0.2896
Inflammatory markers (means)†			
Total cholesterol (mmol/l)	5.11	5.06	0.1196
High-density lipoprotein cholesterol (mmol/l)	1.51	1.58	<0.0001
Triglycerides (mmol/l)	1.26	1.16	<0.0001
C-reactive protein (mg/dl)	2.08	1.99	0.3927
Leucocytes (109)/l)	6.13	5.84	<0.0001
Psychosocial well being			
Life satisfaction			
Proportion of unsatisfied persons (%)	11.6	7.9	<0.0001
Proportion of very satisfied persons (%)	18.4	23.6	<0.0001
Optimism			
Proportion of very optimistic persons (%)	20.1	26.8	<0.0001
Socioeconomic factors			
Income: subjects in two highest income brackets (%)	39.8	46.5	<0.0001
Education: at least college-level education (%)	37.1	49.8	<0.0001
Gender: proportion of males (%)	*55.9*	*43.2*	*<0.0001*
Marital status: married or co-habiting (%)	70.4	74.1	0.0003

*p-values derived from the χ^2 test for categorical variables and from the t-test for difference of means.
†Means are based on a subsample of 5690.

Table 2. Factors associated with the presence of angina pectoris

	Angina pectoris, *n* (yes/no)	OR (95% CI)*
Gender		
Female	47/4448	0.41 (0.30–0.59)
Male	102/4034	1.00
Elevated blood pressure		
Yes	41/1035	2.73 (1.89–3.93)
No	108/7439	1.00
Body mass index		
25 or more	40/2303	1.65 (1.19–2.31)
Under 25	55/3514	1.00
Tooth loss		
Six or more lost teeth	9/192	2.80 (1.40–5.57)
Less than six lost teeth	135/8053	1.00
Gingival bleeding		
Yes	53/1952	1.88 (1.33–2.64)
No	91/6286	1.00
Self-reported caries		
Yes	73/2894	1.88 (1.35–2.62)
No	72/5380	1.00
Total cholesterol		
High	43/2349	1.30 (0.86–1.97)
Low	48/3418	1.00
HDL cholesterol		
Low	7/332	1.36 (0.62–2.97)
High	84/5435	1.00
Triglycerides		
High	8/426	1.21 (0.58–2.51)
Low	83/5340	1.00
C-reactive protein		
High	25/954	2.39 (1.43–4.01)
Average	27/1348	1.83 (1.11–3.03)
Low	36/3288	1.00
Leucocytes (tertiles)		
Highest	49/2281	1.98 (1.16–3.37)
Intermediate	23/1685	1.26 (0.68–2.31)
Lowest	19/1748	1.00
Education		
At least college level	33/3814	0.34 (0.22–0.52)
Vocational school	63/2496	1.00
Comprehensive school	30/1020	1.17 (0.75–1.81)
Other/ studying	19/1060	0.71 (0.42–1.19)
Income bracket		
I (Highest)	6/426	0.57 (0.23–1.43)
II	35/3340	0.43 (0.25–0.73)
III	52/2758	0.76 (0.46–1.27)
IV (Lowest)	22/879	1.00
Smoking		
11 or more packyears	34/1081	2.89 (1.78–4.69)
6–10 packyears	20/833	2.20 (1.26–3.86)
0–5 packyears	43/2110	1.87 (1.18–2.96)
Non-frequent smokers	15/1189	1.16 (0.63–2.14)
Never-smokers	33/3030	1.00
Life satisfaction		
Highest	19/1832	0.36 (0.19–0.67)
Intermediate	100/5684	0.61 (0.38–0.98)
Lowest	22/765	1.00
Optimism (LOT scores)		
20–24 (highest)	18/2038	0.32 (0.18–0.54)
17–19	35/2418	0.52 (0.33–0.80)
14–6	41/2105	0.70 (0.46–1.07)
0–3 (lowest)	49/1755	1.00
Active coping		
I (highest)	14/1139	0.71 (0.36–1.40)
II	17/983	1.00 (0.53–1.90)
III	22/1457	0.88 (0.48–1.59)
IV (lowest)	22/1277	1.00
Passive coping		
I (highest)	26/1118	1.63 (0.86–3.10)
II	24/1734	0.97 (0.51–1.86)
III	10/1051	0.67 (0.30–1.49)
IV (lowest)	15/1052	1.00

*OR, unadjusted odds ratios; 95% CI, 95% confidence intervals.

traditional risk factors such as elevated blood pressure, high BMI and smoking are all positively associated with the presence of angina pectoris. Moreover, socioeconomic factors such as male gender, low income and low education and dental variables such as gingivitis, self-reported caries and tooth loss, and psychosocial factors such as satisfaction with life and optimism were associated with the presence of angina pectoris.

The results of the unstratified and stratified multivariate logistic regression analyses are presented in Table 3. They showed that gingivitis, dental caries and tooth loss were positively associated with the presence of angina pectoris after adjustments for several risk factors such as low education, low incomes, male gender, smoking, elevated blood pressure, high BMI and psychosocial factors such as optimism and satisfaction with life (Table 3).

Gingivitis as a confounder

Gingivitis could also be considered as a confounder as it has an imbalanced distribution in the categories of dental caries and on the condition that it is causally related to angina pectoris, or that it is an indicator of risk factors related to angina pectoris. When we made additional adjustment for gingivitis, the association between dental caries and the presence of angina pectoris weakened (OR 1.44, CI 0.99–2.09). Similarly, gingivitis could be considered as a confounder in the association between tooth loss and the presence of angina pectoris. In this case, further adjustment for gingivitis did not weaken the association (OR 1.55, CI 0.70–3.47).

Subgroup analyses

In the stratified analysis, the association between gingivitis and angina pectoris almost disappeared among men, whereas that among women was distinct (OR 3.48, CI 1.79–6.75). When the analysis was restricted to never-smokers, the results did not essentially differ from those of the total cohort (Table 3).

When the data were stratified according to education, the association was more pronounced among those with higher education. Actually, it was found that in the lowest educational group, there was an inverse association between gingivitis and the presence of angina pectoris. Because of this finding,

Table 3. Associations of self-reported gingivitis, dental caries and tooth loss with the presence of angina pectoris. Results of multivariate logistic regression analyses

	Gingivitis OR (95% CI)*	Caries OR (95% CI)*	Tooth loss OR (95% CI)*
In total cohort	1.52 (1.04–2.22)	1.50 (1.04–2.18)	1.53 (0.69–3.42)
Cohort, without the lowest educational group	1.90 (1.25–2.86)	1.74 (1.16–2.60)	1.80 (0.70–4.62)
Stratified analyses			
Gender			
Among men	1.05 (0.65–1.69)	1.45 (0.93–2.28)	1.54 (0.63–3.74)
Among women	3.48 (1.79–6.75)	1.58 (0.82–3.07)	1.05 (0.13–8.25)
Smoking habits			
Among smokers†	1.47 (0.96–2.26)	1.61 (1.06–2.45)	1.80 (0.80–4.07)
Among never-smokers	1.62 (0.70–3.74)	1.12 (0.48–2.61)	—
Socioeconomic status (SES)			
Among those of low SES‡	1.34 (0.86–2.09)	1.26 (0.82–1.94)	1.13 (0.44–2.91)
Among those of high SES§	2.09 (0.99–4.40)	2.52 (1.21–5.23)	6.19 (1.38–7.82)

*OR, adjusted odds ratios; 95% CI, 95% confidence intervals. Odds ratios are adjusted for gender, income, education, smoking, blood pressure, body mass index, optimism and life satisfaction.

†Current and past smokers.

‡Vocational education, comprehensive school only and other.

§Education in university or in higher educational institutions.

we performed additional analyses by excluding these individuals. The result of the analyses showed that the associations were stronger than those found in the total cohort (Table 3).

Discussion

We controlled for known risk factors such as low education, low incomes, male gender, obesity, elevated blood pressure and smoking. We also controlled for psychosocial resource factors such as dispositional optimism and satisfaction with life, which are related to a large range of health behaviour (Schwarzer 1994). After adjustment, it was found that gingivitis, dental caries and tooth loss were associated with the presence of angina pectoris. However, the association between tooth loss and the presence of angina pectoris was not statistically significant because of the small number of individuals who had six or more missing teeth. When we stratified the data, the strongest associations were found among women and among those of high SES, whereas the association among men and among those of low SES was weaker.

Possible biological mechanism

Among the cohort members, dental diseases were also associated with elevated levels of total cholesterol, low levels of HDL, high triglycerides levels and high leucocyte levels. The results showed that gingivitis and to some extent dental caries, but not tooth loss, were associated with CRP levels. The total cholesterol levels, and especially the ratio of total cholesterol and HDL, are known to be an important predictor of cardiovascular events (Libby et al. 2002). It has previously been found that patients with periodontitis have elevated levels of cholesterols (Saito et al. 2001), but it is not known whether this association is co-incidental because of risks in common or whether there is a causal relation. Lösche et al. (2005) found that periodontal treatment did not significantly change the plasma levels of different lipid fractions, whereas Pussinen et al. (2004) noted that periodontitis may affect CVD morbidity risks by reducing the antiatherogenic potency of HDL.

CRP is a marker of inflammation and it has also been found to predict cardiovascular events (Koenig 2005). Previously, it has been found that those who suffer from gingivitis (Ebersole et al. 2002, Meurman et al. 2003), periodontitis (Noack et al. 2001, Ebersole et al. 2002) or tooth loss (Joshipura et al. 2004) have elevated levels of CRP. However, stress, insulin resistance, inflammatory diseases, smoking, diet, physical exercise and obesity, for example, are also associated with elevated CRP levels. Furthermore, atherosclerotic lesions cause additional synthesis and release of CRP (Labarrere & Zaloga 2004). This indicates that only a part of elevated CRP, if any, can be attributed to infection diseases such as periodontal diseases. This is supported by a study by Mendall et al. (2000), who found that after adjustment for non-circulating risk factors such as age, smoking, current and father's social class, obesity, height, forced expiratory volume blood pressure, total cholesterol levels and fibrinogen, the association between CRP and ischaemic heart disease became insignificant (Mendall et al. 2000).

Interestingly, Slade et al. (2000) found that that when there were one or more systemic risk factors involved, there were only marginal differences in CRP levels between patients with different degrees of severity of periodontal disease. In another study, it was noted that when BMI is high, periodontal disease has only a marginal, if any, effect on the levels of CRP (Slade et al. 2003). These findings are in agreement with the results of another epidemiological study, where it was found that the presence of periodontitis and gingivitis would not increase CHD risks among at-risk individuals (Hujoel et al. 2002a).

On the other hand, there are studies where periodontal treatment has been shown to reduce the inflammatory markers. D'Aiuto et al. (2004, 2005) found that periodontal treatment with patients with chronic severe periodontitis reduces the levels of interleukin-6 and CRP. Reduction of CRP levels because of periodontal treatment has also been reported by Mattila et al. (2002). Montebugnoli et al. (2005) reported that periodontal treatment reduced the levels of CRP and oxidized LDL among patients with diagnosed CVD. However, Pussinen et al. (2004) did not find any effect of periodontal treatment on CRP

levels. These findings indicate that the mechanism through which periodontal disease may cause CVD in not established. Moreover, to date, there is no evidence that periodontal treatment would have any effect on CVD morbidity. Future research can possibly show whether periodontal treatment has any effect on CVD morbidity, and whether such an effect is mediated through a mechanism related to cholesterol composition or CPR levels.

Confounding as an explanation

As shown in Table 1, several factors that cause elevated levels of CRP are associated with gingivitis, dental caries and tooth loss. Those who reported these diseases and symptoms undertook physical exercise less frequently, were more often heavy users of alcohol and ate vegetables less often. In addition, they were more often obese and were more likely to have high blood pressure. We have previously found an association between inferior dental behaviour and greater health risks such as high blood pressure, elevated levels of serum total cholesterol and serum triglycerides, low levels of HDL cholesterol, high BMI and abdominal obesity among these cohort members (Ylöstalo et al. 2003). We have also found an association between unhealthy health habits – including smoking, alcohol abuse, insufficient physical exercise and unhealthy diet – and dental health (Ylöstalo et al. 2003). The mentioned findings and a lack of comparability in subject characteristics as shown in Table 1 suggest that there may be a substantial amount of confounding in the association between dental diseases and the presence of angina pectoris. This means that the associations may be co-incidental because of risk factors in common, and dental diseases are merely indicators of risk factors for angina pectoris. Consequently, gingivitis, for example, is a confounder in the association between dental caries and angina pectoris. According to our expectations, the adjustment for gingivitis affected the association between dental caries and the presence of angina pectoris. On the other hand, the association between tooth loss and the presence of angina pectoris was not affected by adjustment for gingivitis. This may be explained by the possibility that both gingivitis and tooth loss represent, to some extent, periodontitis as an underlying disease. Moreover, as shown in Table 1, gingivitis seems, to a large extent, to represent risks that are common with tooth loss, with the exception that tooth loss seems to associate more strongly with behavioural health risks (especially smoking) than gingivitis. We must bear in mind the fact that the members of this cohort were about 30 years olds, which means that it is not plausible that periodontitis alone would lead to situations where extraction becomes the only treatment option. Periodontitis, however, together with large caries lesions or with the patient's negative attitude towards the preservation of teeth, for example, may lead to extraction as a form of treatment.

Previous studies have shown the coincidence between the effects of smoking, and the effects, possibly putative, of periodontal diseases (Jansson et al. 2002, Hujoel et al. 2002b, Molloy et al. 2004). In addition to smoking, it is possible that other forms of health behaviour or attitudes towards health maintenance cause confounding and can thus be an explanation for associations between dental diseases and lifestyle diseases in general. One of the most important determinants for health behaviour is SES. Previously, it has been found that people with a low SES use less health-care services and have unhealthier self-care habits than people with a high SES. Thus, it is not surprising that descriptive studies have shown that the prevalence and incidence of several chronic diseases including dental diseases varies according to SES.

The effect of SES is the strongest on periodontal diseases, followed by dental caries and oral cancer when these diseases are compared (Hobdell et al. 2003). Hobdell et al. (2003) estimated that SES alone accounts for 50% of variation in the prevalence of periodontitis between the ages of 35 and 44. This might explain why positive associations between periodontal diseases and systemic diseases closely related to SES, such as cardiovascular and cerebrovascular diseases, are common. This also implies that SES as an indicator of numerous indefinable characteristics may be a strong potential confounder. Besides, SES has properties such as not being directly interpreted categorically or numerically. This prevents accurate measurements of SES, which may lead to residual confounding.

Based on previous findings on the associations between dental diseases and other lifestyle diseases, and the association between dental diseases and SES, it is not surprising that an association exists between dental caries and the presence of angina pectoris in this cross-sectional data. The association is supposed to be mediated through risks in common such as diet. Larsson et al. (1995) concluded that dental caries could be an indicator for traditional risk factors for CVD, whereas the most plausible explanation for the finding was that dental caries is associated with diet, which in turn is associated with diet-related cardiovascular risks such as unfavourable lipid concentrations, high blood pressure and high BMI. The association between poor dental health and poor diet has also been found in studies by Johansson et al. (1994) and Hung et al. (2005).

Tuominen et al. (2003) concluded that positive associations between oral diseases and coronary health diseases are most likely explained by health behaviour. In their study, additional adjustment for other cardiovascular risk factors reduced the risk of dental diseases to CVD to non-significant. In addition to health behaviour, health awareness has been suggested to be an important confounder that may cause spurious associations (Hujoel 2002). In our data, psychosocial resource factors such as dispositional optimism and satisfaction with life were associated with the presence of angina pectoris. Adjustment for these made the association of gingivitis, dental caries and tooth loss with angina pectoris weaker, which was not an unexpected finding as optimism, for example, has previously been associated with better health behaviour (Schwarzer 1994).

Subgroup analyses

We analysed the association between gingivitis and the presence of angina pectoris among never-smokers and smokers separately. Contrary to our expectations, the estimates in the group of never-smokers did not essentially differ from those of the total cohort. It is possible, however, that the association could have been stronger among smokers if we had assessed gingivitis in clinical examination instead of assessing it on the basis of self-reported gingival bleeding as smokers are known to have less gingival bleeding and less bleeding on probing (Preber & Bergström 1985,

Haffajee & Socransky 2001). In the case of gingival bleeding, smoking fulfils the operational criteria for confounding. It is causally related to CVD and it is unequally distributed in the explanatory variable, and smoking is not an intermediate factor in the causal chain between dental diseases and CVD. The distribution in the explanatory variable may be affected by two opposite effects in the case of gingival bleeding: on the one hand, smoking reduces gingival bleeding, and on the other, smokers have less favourable tooth brushing habits, which tends to increase gingival bleeding. When we stratified the data according to smoking, the association between dental caries and angina pectoris among never-smokers weakened. The different pattern between gingivitis and dental caries is understandable as smoking is closely related to gingival bleeding, but not to dental caries. There were no never-smokers with angina pectoris among those who had lost six or more teeth, which made stratified analyses concerning tooth loss impossible.

Further subgroup analyses showed that the associations were strongest among women and individuals belonging to a high SES. This might be related to the fact that in these groups detection and reporting of dental diseases and symptom are better. Misclassification may be related to the level of respondents' health awareness, while knowledge about dental diseases is related to gender and educational level. In the case of gingivitis, detection of gingival bleeding depends on the smoking, which is common especially among those of low SES. For example, almost all men (18/21) and all women among those with only secondary education and who reported angina pectoris were smokers. In the stratified analysis, we found an inverse association between gingivitis and the presence of angina pectoris. When analyses were performed after excluding the lowest educational category, the associations were stronger than those in the total cohort.

When the data were stratified according to gender, we observed that gender modifies the association of tooth loss and gingivitis with the presence of angina pectoris. There was no clear association between gingivitis to angina pectoris among men, whereas it did exist among women. However, we are not aware of the existence of any biological mechanism that might explain why gender modifies the associations of tooth loss and gingivitis with angina pectoris.

In addition to differences in detection and reporting of dental symptoms and disease, it is possible that the association found among women or the highly educated also implies differences in the diagnosis of angina pectoris. This would indicate that angina pectoris remains under-diagnosed among those of low educational levels. This may be related to better health awareness among highly educated, but also to better opportunities to use medical services.

Limitations of the study

Most epidemiological studies on the association between periodontal diseases and CVD do not support the role of gingivitis as an aetiological factor for CVD (Armitage 2000, Genco et al. 2002). Rather, they suggest that severe periodontal disease could be causally associated with CVD, although the evidence is not definite. This means that gingivitis is by no means the most important or the most interesting variable from the point of view of a potential causal explanation between periodontal diseases and CVD. From this point of view, periodontitis would have been more interesting. Unfortunately, the data in this study were self-reported, so it was not possible to assess periodontitis. On the other hand, it is not possible to state conclusively what gingival bleeding represents in this study. It is possible that some of those who reported gingival bleeding actually have periodontitis and not gingivitis.

A major drawback is that the study is based on self-reported diseases and symptoms, which is, however, quite commonplace in large epidemiological studies. This means that dental diseases and symptoms as well as systemic diseases and symptoms are subjected to biases related to observation, reporting and level of information. Previous studies have shown that simple, detectable dental diseases or symptoms correspond quite well to clinical findings (Unell et al. 1997). An example of a well-defined finding is tooth loss. In our questionnaire, the respondents were asked how many missing teeth they had (0, 1–5, 6–10, more than 10 but not all, all). A high cut-point (0–5 *versus* six or more) was chosen so as to avoid misclassification because of extractions of third molars, minor traumas and extractions related to orthodontic treatment. In the case of the number of missing teeth, an epidemiological study carried out by the National Health Institute in 1998 showed a fairly high concordance between the total population and this cohort (Ylöstalo et al. 2004). Gingival bleeding is fairly easy to detect and can be observed and reported quite easily. However, concordance between self-reported gingival bleeding and clinically determined gingivitis has been found to be poor (Buhlin et al. 2002a). Self-reported dental caries is an example of a variable that was not well defined, is difficult to detect and is thus subject to errors. Reporting of dental caries is affected by biases related to observation and the level of information. Therefore, the results related to self-reported caries should be considered as purely indicative.

The outcome variable, angina pectoris, was also self-reported. A question was asked as to whether the respondents had angina pectoris diagnosed by a doctor. It is possible that there may have been bias related to reporting, as no clinical examination was carried out. However, an earlier study in Finland has shown that concordance between CVD assessed by questionnaire and medical records is substantial in the Finnish population (Haapanen et al. 1997). Moreover, the prevalence of angina pectoris in this age group of 31 year olds was 1.7%, which compares quite well with studies conducted in Sweden and USA. Prevalence in the age group of 35–44 year olds in Sweden was 2.8% for men and 1.5% for women, and 3.6% for men and 2.2% for women in USA (Glader & Stegmayr 1999, Zaher et al. 2004).

In addition, this study has several limitations related to the study design. One limitation is that it is difficult to make causal statements because of temporal ambiguity related to a cross-sectional study design, meaning that we do not even know whether gingivitis, dental caries and tooth loss precede angina pectoris. On the condition that these precede angina pectoris, we have to be aware that the time period from exposure to disease is at best short in relation to cardiovascular alterations.

The assessment of risk factors after a disease has been diagnosed may also affect responses, and may thus cause information bias. There are, however, facts that limit the effect of selection and information biases in these data.

Firstly, the subjects were members of a general birth cohort and were not selected among those with disease. Secondly, the outcome variables, such as dental diseases and systemic diseases and symptoms, are quite common in the total population and neutral in nature. The possibility of bias because of selective response cannot be totally excluded either, although the high response rate (75.3%) reduces the magnitude of the effect of selection bias.

Conclusion

Our aim in this paper was to investigate whether there are associations of self-reported gingivitis, dental caries and tooth loss with the presence of angina pectoris. The results show that they were all associated with the presence of angina pectoris. They were also associated with several CVD risk factors, suggesting confounding as an alternative explanation for causality. Moreover, we found an association between tooth loss and angina pectoris among young adults where periodontitis alone is not a plausible explanation for tooth loss. This stresses the importance of factors other than periodontal infection as a cause for CVD. A causal explanation is not a very feasible explanation also because an association exists between dental caries and the presence of angina pectoris. However, the possibility that both causal association and confounding may exist simultaneously cannot be totally excluded.

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Clinical Relevance

Scientific rationale: The results of earlier studies connecting dental diseases to CVD are inconsistent, which have been explained by the fact that confounders are not properly controlled in the analyses.

Principal findings: Dental diseases were associated with the presence of self-reported angina pectoris but also with traditional risk factors of CVD such as high blood pressure, unfavourable lipid levels, obesity and lack of physical exercise.

Practical implications: The presence of dental diseases indicates elevated health risks because of their associations with unhealthy health behaviour and with cardiovascular risk factors.

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