

Oral health of monozygotic twins with and without coronary heart disease: a pilot study

Farnaz Tabrizi, Kåre Buhlin, Anders Gustafsson and Björn Klinge

Department of Periodontology, Institute of Odontology, Karolinska Institutet, Huddinge, Sweden

Tabrizi F, Buhlin K, Gustafsson A, Klinge B. Oral health of monozygotic twins with and without coronary heart disease: a pilot study. *J Clin Periodontol* 2007; 34: 220–225. doi: 10.1111/j.1600-051X.2006.01041.x.

Abstract

Aim: The purpose of this study was to investigate the oral health in monozygotic twins where one twin had coronary heart disease (CHD) and the other twin had no clinical signs of the disease.

Methods: Ten monozygotic twin pairs (age 55–81 years, eight male, and two female pairs) were recruited from the Swedish twin register. The inclusion criterion for participation was discordance regarding the presence of CHD within every twin pair. All participants underwent a full dental clinical examination including a panoramic radiograph.

Results: Twins with CHD had 51.5% bleeding on probing compared with 21.1% without CHD ($p = 0.01$), and more pathological pockets (≥ 4 mm) were detected among those with CHD (20 ± 15 versus 8 ± 5), $p = 0.047$). Twins with CHD had a reduced horizontal bone level in comparison with the healthy group (73% versus 78%, $p = 0.03$). Logistic analyses using odds ratio (OR) showed that an increase of one periodontal pocket (≥ 4 mm) resulted in an increased risk for the actual twin of belonging to the CHD group (OR 1.17, $p = 0.03$).

Conclusions: This study indicates worsened periodontal conditions among twins with CHD compared with their siblings with no history of CHD. This strengthens the association between periodontal inflammation and the presence of atherosclerosis.

Key words: cardiovascular disease; oral health; periodontitis; risk factors; twins

Accepted for publication 7 November 2006

This study involves two of the most common diseases of the world: cardiovascular disease (CVD) and periodontitis. Today, both diseases are known to have an inflammatory origin (Page & Schroeder 1976, Ross 1999). Atherosclerosis is the most common underlying mechanism behind coronary heart disease (CHD) (Ross 1999). The periodontal pathogens and periodontal inflammation may trigger a systemic

inflammatory response, which favours an atherosclerotic process. Since 1989, when the first of several carefully designed Finnish studies investigated the association between CVD and oral health, the alleged association has been one of the main research areas in periodontology (Mattila et al. 1989, 1995, Paunio et al. 1993, Persson et al. 2003, Spahr et al. 2006). Periodontal disease as a possible risk factor for the development of atherosclerosis has been the subject of investigation, albeit with conflicting results (for a review, see Meurman et al. 2004). According to some researchers, the association depends on mutual risk factors, such as smoking, as this is a known risk factor for both CVD and periodontitis (Hujoel et al. 2002). However, there are also other factors that could cause a false association, and

these have been discussed in this context, such as the genetic influence, diabetes mellitus (Salvi et al. 1997, Beck et al. 1998, Patel & Kent 1998) and different socio-economic variables (for a review, see Klinge & Norlund 2005). The role of inheritance in periodontitis may be so great as to be around 50% (Michalowicz et al. 2000). Likewise the hereditary effect on CVD is substantial (Haskell 2003). Therefore, a common genetic predisposition could provide an explanation for some of the alleged association of the two diseases (Offenbacher et al. 1999, Kornman & Duff 2001). Different polymorphisms in the interleukin-1 (IL-1) genes have been associated with the risk of periodontal and CVD (Kornman & Duff, 2001) and also TNF- α have been discussed (Craandijk et al. 2002). These cytokines are

Conflict of interest and source of funding statement

The authors declare that they have no conflict of interest.

This research was supported in part by a grant from The Swedish Dental Society and Margareta and Carl Modéns Memorial Foundation, Sweden.

associated with a high risk of periodontal disease and could be correlated with a risk for CVD, depending on the linkage pattern of the polymorphisms. However, as the specific genes associated with an increased risk remain unknown, it is impossible to control for potential genetic confounders in a classical epidemiological approach. An investigation of monozygotic twins minimizes the genetic influence and can thus provide a unique opportunity to study the association between periodontitis and CHD without genetic confounders.

The aim of this study was therefore to investigate the oral health in monozygotic twins where one twin had CHD and the other twin had no clinical signs of the disease.

Material and Methods

Study population

A total of 20 men and women participated in this case-control study and were born between 1923 and 1949. They were recruited from the Swedish twin registry (STR; Lichtenstein et al. 2002). This is currently the largest twin registry in the world, it is population based and it has registered more than 60,000 pairs of twins born in Sweden since 1886. The merging of the STR register with the Swedish National Birth Registry and the Health and Disease Register further augments the STR, by allowing an almost complete health follow-up of twins. The only inclusion criterion for the participation in the study was discordance regarding CHD within every twin pair, which is one twin with CHD and the other without. The diagnosis of CHD, which includes exclusively WHO diagnosis codes ICD 8–10, (WHO ICD 10, 1992), was based on information obtained from the medical records and confirmed by patient interviews. This excludes pairs in which at least one twin had signs of angina or CHD, but had not sought or received a medical diagnosis or care. This excludes also pairs in which the healthy one developed one of the mentioned CHD diagnoses recently.

The CHD patients in this study had a history of angina pectoris and/or previous myocardial infarction. Twenty-four monozygotic twin pairs living in the region of Mälardalen, Sweden, were identified in the STR to fulfil this criterion. The twins also had all participated

in the screening across the lifespan of twins (SALT) study (Lichtenstein et al. 2002). In this study, the verifiability of the zygosity of the twin pairs were tested and based on a detailed validated questionnaire. Based on the SALT questionnaire findings, all the participating twins in this present study population were monozygotic.

All subjects were contacted and the study protocol was explained in detail. Seven pairs (29.2%) could not participate in the study because of personal reasons, two pairs were excluded from the study because they did not fulfil inclusion criterion and five pairs (21.8%) declined to participate in the study because of varying health problems. They all received a questionnaire, and 10 pairs (mean age 65 ± 9) agreed to take part in the oral health examination (41.7%). According to their ICD diagnosis and cardiovascular health, no differences could be found between those who declined to participate and those who took part in the study. The participants were asked questions concerning general health, smoking habits and different socio-economic variables regarding income, education, profession, and civil status. The healthy twin of the pair acted as the control for the CHD ones. Therefore, the genetic influence on CHD and periodontitis in this study can be disregarded. Twins with CVD were treated with conventional cardiac medication, such as aspirin, ACE inhibitors, β -blockers and statins.

Both current and former smokers were considered in this study to be smokers and the non-smoking group consisted of only those who never smoked. Within this patient and control group, the number of smokers were few: only three patients and four controls. Therefore, it was impossible to carry out any statistical calculations or draw any conclusions from the perspective of smoking habits.

The study was approved by the Ethics Committee at Huddinge University Hospital and also by the Swedish Twin Registry's Review Board. The study was performed in accordance with the Helsinki declaration. All participants gave their informed consent.

Clinical and radiographic examinations

Patients and controls underwent a comprehensive dental examination, including digital panoramic radiographs (Planmeca Dimaxis, Planmeca Oy/Ab,

Helsinki, Finland). One dentist (F. T.) performed all the clinical examinations without prior knowledge of the coronary heart status of the examined twin. The oral examination of the participants consisted of the following parameters: evaluation of the teeth, gums and soft tissues, measurement of periodontal pocket depth with a Hu-Friedy (PCPUNC 15, Chicago, IL, USA) probe, assessment of hygiene (HI-index, Love et al. 1975) and bleeding on probing. Probing depth was defined as the distance between the gingival margin and to the bottom of the gingival pocket measured from six angles of each tooth. Gingival pockets 4 mm or deeper were considered pathogenic (Nyman & Lindhe 2003). All teeth except third molars were assessed.

Dentures, both complete and partial, in either jaw, were classified as removable dentures.

The clinical measurement was also combined with the marginal and vertical bone height evaluated from the panoramic radiographs for the assessment of periodontal disease.

The examination was performed, using the hygiene index (%), to determine the presence or absence of dental plaque at the gingival margin. Dental plaque was made visible by gently moving the tip of the probe along the gingival margin of the four sides of each tooth.

Gingival inflammation was noted as bleeding on probing and expressed as the proportion of bleeding sites in relation to the total number of sites in the dentition (Ainamo & Bay 1975).

The digital panoramic radiographs were examined with the Image Tool 3.0 programme (Department of Dental Diagnostic Science, University of Texas Health Science Center, San Antonio, USA) for digital radiographic measurements in pixels. In each patient, the periodontal marginal bone height was measured mesially and distally to each tooth and expressed as a percentage of the root length. Bone height was calculated using the formula: total bone height/total root length [the distance from the radiographic apex to the cemento-enamel junction (CEJ)] \times 100. This percentage corresponded to the remaining bone height supporting the tooth. A tooth was judged as non-measurable if the CEJ or bone crest could not be identified properly because of overlapping caries or restorations. In cases where any one of the dental or bony landmarks could not be identified

on one aspect (mesial or distal), the tooth was excluded. A total of 54 teeth, most often maxillary pre-molars, were excluded. To obtain one representative value per participant, the mean bone height for all teeth was calculated.

Restorations and dental caries were also calculated for each participant. Dental caries was determined under optimal light conditions with visual inspections and panoramic radiographs. The caries lesions were assessed under dry conditions by colour and gentle probing with an examination probe (Hu-Friedy) and missed teeth excluding third molar observed with visual inspections and radiographs. The result is shown according to the DMF-T (decayed, missed, filled teeth) and DMF-S (decayed, missed, filled surfaces) systems (modified from Klein et al. 1938).

The severity of attachment loss (loss of supportive tissue around the teeth) was classified as more or less than or equal to $\geq 1/3$ of the root length (Nyman & Lindhe 2003).

Statistical analysis

Statistica 7.0 and the SPSS 11.5 software programmes were used to perform statistical analysis. Descriptive statistics were used to characterize data. Normally distributed data were expressed as mean value \pm standard deviation (SD), and data with a skewed distribution as median value with range. The χ^2 independent test was used for comparison of proportions. Parametric and non-parametric data were analysed, using Student's *t*-test or Mann-Whitney *U*-test, respectively. Univariate regression analysis was used to calculate the odds ratio (OR) for any relationship between

oral health and CHD. The level of significance was set at $p = 0.05$ and the confidence interval (CI) at 95%. CI were calculated using Wald method.

Results

Clinical characteristics and dental features

The mean age of the study population was 65.0 (SD \pm 9.0) years (range 55–81 years). There was no significant difference between patients with CHD and those without with regard to smoking status, marital status, education, employment, income before tax, and body mass index (BMI) (26 SD \pm 6 and 26 SD \pm 4, respectively, Table 1).

No patient in the study population had a history of diabetes mellitus.

Differences were apparent among the patients concerning the periodontal parameters; patients with CHD showed more signs of clinical periodontitis. The mean of bleeding upon probing in the CHD group was 52% (SD \pm 23) compared with 21% (SD \pm 11), ($p = 0.01$) in the control group, and the mean number of sites (4–5 mm) among patients with CHD was 16 (SD \pm 11) versus 7 (SD \pm 4), ($p = 0.05$) in the control group. The mean number of pathological pockets (≥ 4 mm) was also significantly different: 20 mm (SD \pm 15), and 8 mm (SD \pm 5) ($p = 0.047$), respectively (Table 2).

Regarding the remaining dental feature variables, no significant differences

Table 1. Characteristics (%) of patients within the study, with CHD patients compared with non-CHD patients

	n (%)		p-value
	twin with CHD, n = 10	twin without CHD, n = 10	
Smoker (current and former)	3 (30%)	4 (40%)	0.1
Diabetes mellitus	0 (0%)	0 (0%)	
BMI > 24	5 (50%)	6 (60%)	0.4
Marital status			
Married	9 (90%)	8 (80%)	1.0
Single/widow	1 (10%)	2 (20%)	
Education			
Elementary	1 (10%)	1 (10%)	0.5
Secondary	4 (40%)	6 (60%)	
University	5 (50%)	3 (30%)	
Employment			
Employee	4 (40%)	4 (40%)	1.0
Retired	6 (60%)	5 (50%)	
Other	0 (0%)	1 (10%)	
Income before tax/month (SEK)			
0–10,000	1 (10%)	0 (0%)	0.5
10,000–20,000	3 (30%)	4 (40%)	
20,001 or more	6 (60%)	6 (60%)	

CHD, coronary heart disease; BMI, body mass index.

Table 2. Oral health variables in all twin pairs

Twin pair	DMF-T No. A/B	DMF-S No. A/B	No. of teeth* A/B	Plaque (% A/B)	BOP (% A/B)	No. of sites 4–5 mm A/B	Total no. sites ≥ 4 mm A/B
1	18/18	52/52	28/27	43/25	67/8	18/6	19/6
2	14/25	36/40	28/27	31/11	59/14	31/3	38/5
3	25/20	71/28	26/28	100/3	100/12	36/6	53/6
4	25/21	70/54	25/24	41/27	54/36	18/5	20/7
5	13/16	25/39	28/28	15/35	18/28	1/2	1/2
6	15/18	29/46	25/25	30/15	34/23	10/12	11/16
7	20/19	69/65	26/26	22/6	54/9	15/10	21/11
8	28/28	89/87	26/23	37/11	56/15	18/2	18/2
9	26/18	83/65	23/27	18/16	28/35	8/10	8/11
10	20/19	66/59	22/24	43/25	45/31	8/13	13/14
Mean \pm SD	20 \pm 5/20 \pm 4	59 \pm 22/53 \pm 17	26 \pm 2/26 \pm 2	38 \pm 24/17 \pm 10	52 \pm 23/21 \pm 11	16 \pm 11/7 \pm 4	20 \pm 15/8 \pm 5
p-value	p = 0.9	p = 0.3	p = 0.76	p = 0.06	p = 0.01	p = 0.049	p = 0.047

*Third molar excluded.

A, twin with CHD; B, twin without CHD; BOP, bleeding on probing %; DMF-S, decayed, missed and filled surfaces; DMF-T, decayed, missed and filled teeth; SD, standard deviation; CHD, coronary heart disease.

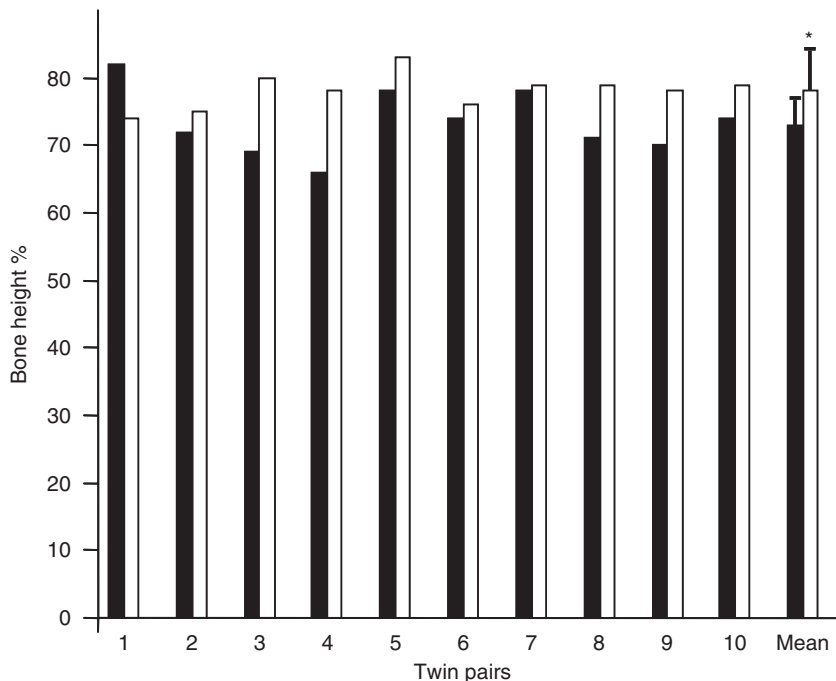


Fig. 1. Radiographical bone height (%) in twins with (solid bars) and without (open bars) coronary heart disease (CHD). The mean values (with standard deviations), shown in the last set of columns, differ significantly ($p = 0.03$).

Table 3. Odds ratios (OR) [95% confidence interval (CI)] for the relationship between various indicators of oral health, periodontal health and the patient group

	Odds ratio patient group	95% Confidence interval	<i>p</i> -value
Dental plaque (increase of 1%)	1.11	1.01–1.29	0.03
DMF-S (increase of 1 unit)	1.04	0.96–1.17	0.4
DMF-T (increase of 1 tooth)	1.02	0.77–1.35	0.9
Gingival bleeding (increase of 1%)	1.1	1.01–1.32	0.01
Mean marginal alveolar bone height (decrease of 1%)	1.3	1.01–1.98	0.04
No. of pockets (4 and 5 mm) (increase of one pocket)	1.18	1.00–1.59	0.049
No. of teeth (decrease of one tooth)	1.12	0.54–2.51	0.9
Total no. of pockets ≥ 4 mm (increase of 1 pocket)	1.17	1.01–1.54	0.03

The associations are shown for all participants.

DMF-S, decayed, missed and filled surfaces; DMF-T, decayed, missed and filled teeth.

were observed between the two groups and only one participant had a partial denture (Table 2).

Radiographic examination

We observed that the mean horizontal alveolar bone levels were lower on the panoramic radiographs of the CHD patients compared with the patients without CHD, $73 \pm 5\%$, and $78 \pm 3\%$ ($p = 0.03$), respectively (Fig. 1).

Regressions analysis

In the univariate analyses, a significant relationship was apparent between the clinical criteria for the diagnosis of periodontitis (measured as dental plaque, gingival bleeding and pockets 4–5 mm) and the prevalence of CHD (Table 3). These associations were also found between the total numbers of pockets 4 mm or deeper (OR 1.17, CI 1.01–1.54, and $p = 0.033$) and the mean marginal bone height (OR

1.3, CI 1.01–1.98, and $p = 0.041$) (Table 3).

Discussion

The periodontal–CVD association has generated considerable debate. One explanation for the positive findings is confounded by the shared genetic factors. As specific genes are unknown for either disease, it is impossible to control for multi-factual genetic traits in a classical epidemiological approach. Our present study design investigating monozygotic twin pairs directly addresses this limitation.

The present pilot study is the first that has shown an association between periodontal disease and CHD in monozygotic twins, which completely takes into consideration genetic variability, although the findings indicate that a genetic influence is not enough to explain the associations observed in this study and in previous studies (e.g. Buhlin et al. 2005, Spahr et al. 2006).

Studies calculated by (Michalowicz et al. 1991, 1999, 2000) have suggested that 40–80% of the population variance for gingivitis, probing depth, attachment loss, and plaque might be attributed to genetic influence. In another study, the concordance for periodontal disease was 38% among monozygotic compared with 16% among dizygotic twins, lending further support for a significant genetic component of periodontal disease (Corey et al. 1993). In a Swedish study, heritability estimates of periodontal disease were 39% and 33% for women and men, respectively (Mucci et al. 2005). Analysis of data from this large, population-based study demonstrates a moderate role of genetic factors in periodontal disease, and suggests potential gene–environment interactions.

Regarding oral status and CHD, a possible mechanism of pathogenesis is that periodontal inflammation may trigger the atherosclerotic process in the blood vessels (Li et al. 2002, Buhlin et al. 2003, Lalla et al. 2003). The atherosclerotic process is slow and accumulates over time, therefore; the loss of teeth as a consequence of periodontal disease could be an important risk indicator for atherosclerosis.

The results from this investigation show an association between marginal bone loss and CHD. This is interesting

as bone loss is one of the principal signs of periodontal disease, which cannot be changed and is also a reliable way of determining periodontal disease accumulation over time. This result is in agreement with another Swedish study (Persson et al. 2003), where they found an increased risk for acute myocardial infarction by an OR of 5.5 if one also suffered from severe marginal bone loss. In the present study, the mean horizontal alveolar bone height on the panoramic radiographs, was lower in the CHD patients compared with the no-CHD group: 73% and 78%. Our result of bone loss related to CHD is in agreement with previous studies in which a strong association between loss of bone and CHD has been observed. In a North American study on men, the severity of periodontitis was demonstrated to have a linear relation to future risk of CHD and stroke (Beck et al. 1996). The result was that individuals with severe bone loss at baseline had a 50% increase in the incidence of CHD. Another study showed that the percentage of persons with a history of heart attack increased with each increasing category of attachment loss (Arbes et al. 1999).

Other findings like the plaque score and bleeding on probing can easily be altered by the patient just by improving his/her oral hygiene. Therefore, the increased risk for being in the CHD group if one has a higher plaque or bleeding on probing score should be interpreted with caution.

The plaque score and gingival bleeding, together with the other oral health data may be influenced by various socio-economical factors, and the data in Table 3 are based on univariate analyses. Another explanation for the differences could be that the twins within the same pair developed and retain different attitudes towards health and experienced very different related histories and risk behaviours. However, after examining the participating twins this is regarded as unlikely.

This current pilot study has a limited number of participants, mainly as there are not that many twins who are discordant regarding CVD; being based on 10 twin pairs, this is a pilot study, and larger numbers of subjects should be studied. Another problem was that many of the monozygotic twin pairs in this study declined to participate. There were several reasons for declining such as old age, compromised health, geographical, economical, study fatigue,

among others. The limited number of participants did not allow us carry out multivariate comparisons, i.e. to compensate for variables such as smoking, weight, socio-economy and diabetes mellitus. However, these variables did not differ significantly between twins with CHD and those without. Therefore, they are less likely to account for the co-occurrence of CHD and periodontitis in these patients. Despite these limitations, this is the first study showing an association between CVD and periodontitis, without genetic confounders. Thus, a substantial influence of heredity could be ruled out.

Conclusion

This study indicates worsened periodontal conditions among twins with CHD compared with their siblings with no history of CHD. This strengthens the association between periodontal inflammation and the presence of atherosclerosis.

References

- Ainamo, J. & Bay, I. (1975) Problems and proposals for recording gingivitis and plaque. *International Dental Journal* **25**, 229–235.
- Arbes, S. J. Jr., Slade, G. D. & Beck, J. D. (1999) Association between extent of periodontal attachment loss and self-reported history of heart attack: an analysis of NHANES III data. *Journal of Dental Research* **78**, 1777–1782.
- Beck, J., Garcia, R., Heiss, G., Vokonas, P. S. & Offenbacher, S. (1996) Periodontal disease and cardiovascular disease. *Journal of Periodontology* **67**, 1123–1137.
- Beck, J. D., Offenbacher, S., Williams, R., Gibbs, P. & Garcia, R. (1998) Periodontitis: a risk factor for coronary heart disease? *Annals of Periodontology* **3**, 127–141.
- Buhlin, K., Gustafsson, A., Ahnve, S., Janszky, I., Tabrizi, F. & Klinge, B. (2005) Oral health in women with coronary heart disease. *Journal of Periodontology* **76**, 544–550.
- Buhlin, K., Gustafsson, A., Pockley, A. G., Frostegård, J. & Klinge, B. (2003) Risk factors for cardiovascular disease in patients with periodontitis. *European Heart Journal* **24**, 2099–2107.
- Corey, L. A., Nance, W. E., Hofstede, P. & Schenkein, H. A. (1993) Self-reported periodontal disease in a Virginia twin population. *Journal of Periodontology* **64**, 1205–1208.
- Craandijk, J., van Krugten, M. V., Verweij, C. L., van der Velden, U. & Loos, B. G. (2002) Tumor necrosis factor- α gene polymorphisms in relation to periodontitis. *Journal of Clinical Periodontology* **29**, 28–34.
- Haskell, W. L. (2003) Cardiovascular disease prevention and lifestyle interventions: effectiveness and efficacy. *Journal of Cardiovascular Nursing* **18**, 245–255.

- Hujoel, P. P., Drangsholt, M., Spiekerman, C. & DeRouen, T. A. (2002) Periodontitis-systemic disease associations in the presence of smoking – causal or coincidental. *Periodontology* **2000** **30**, 51–60.
- Klein, H., Palmer, C. E. & Knutson, J. W. (1938) Studies on dental caries I. Dental status and dental needs of elementary school children. *Public Health Reports* **53**, 751–765.
- Klinge, B. & Norlund, A. (2005) A socio-economic perspective on periodontal diseases: a systematic review. *Journal of Clinical Periodontology* **32** (Suppl. 6), 314–325.
- Kornman, K. S. & Duff, G. W. (2001) Candidate genes as potential links between periodontal and cardiovascular diseases. *Annals of Periodontology* **6**, 48–57.
- Lalla, E., Lamster, I. B., Hofmann, M. A., Bucciarelli, L., Jerud, A. P., Tucker, S., Lu, Y., Papapanou, P. N. & Schmidt, A. M. (2003) Oral infection with a periodontal pathogen accelerates early atherosclerosis in apolipoprotein E-null mice. *Arteriosclerosis, Thrombosis & Vascular Biology* **23**, 1405–1411.
- Li, L., Messas, E., Batista, E. L. Jr., Levine, R. A. & Amar, S. (2002) Porphyromonas gingivalis infection accelerates the progression of atherosclerosis in a heterozygous apolipoprotein E-deficient murine model. *Circulation* **105**, 861–867.
- Lichtenstein, P., De Faire, U., Floderus, B., Svartengren, M., Svedberg, P. & Pedersen, N. L. (2002) The Swedish twin registry: a unique resource for clinical, epidemiological and genetic studies. *Journal of Internal Medicine* **252**, 184–205.
- Love, W. D., Ramirez, J. M. & Fultz, R. P. (1975) An oral hygiene measurement system for possible research and clinical use. *Journal of Public Health Dentistry* **35**, 227–230.
- Mattila, K. J., Nieminen, M. S., Valtanen, V. V., Rasi, V. P., Kesaniemi, Y. A., Syrjala, S. L., Jungell, P. S., Isoluoma, M., Hietaniemi, K. & Jokinen, M. J. (1989) Association between dental health and acute myocardial infarction. *British Medical Journal* **298**, 779–781.
- Mattila, K. J., Valtanen, V. V., Nieminen, M. & Huttunen, J. K. (1995) Dental infection and the risk of new coronary events: prospective study of patients with documented coronary artery disease. *Clinical Infectious Diseases* **20**, 588–592.
- Meurman, J. H., Sanz, M. & Janket, S. J. (2004) Oral health, atherosclerosis, and cardiovascular disease. *Critical Reviews in Oral Biology and Medicine* **15**, 403–413.
- Michalowicz, B. S., Aeppli, D., Virag, J. G., Klump, D. G., Hinrichs, J. E., Segal, N. L., Bouchard, T. J. Jr. & Pihlstrom, B. L. (1991) Periodontal findings in adult twins. *Journal of Periodontology* **62**, 293–299.
- Michalowicz, B. S., Diehl, S. R., Gunsolley, J. C., Sparks, B. S., Brooks, C. N., Koertge, T. E., Califano, J. V., Burmeister, J. A. & Schenkein, H. A. (2000) Evidence of a substantial genetic basis for risk of adult periodontitis. *Journal of Periodontology* **71**, 1699–1707.

- Michalowicz, B. S., Wolff, L. F., Klump, D., Hinrichs, J. E., Aeppli, D. M., Bouchard, T. J. Jr. & Pihlstrom, B. L. (1999) Periodontal bacteria in adult twins. *Journal of Periodontology* **70**, 263–273.
- Mucci, L. A., Bjorkman, L., Douglass, C. W. & Pedersen, N. L. (2005) Environmental and heritable factors in the etiology of oral diseases – a population-based study of Swedish twins. *Journal of Dental Research* **84**, 800–805.
- Nyman, S. & Lindhe, J. (2003) Examination of patients with periodontal disease. In: Lindhe, J., Karring, T. & Lang, N. P. (eds). *Clinical Periodontology and Implant Dentistry*, pp. 403–413. Copenhagen: Blackwell Munksgaard.
- Offenbacher, S., Madianos, P. N., Champagne, C. M., Southerland, J. H., Paquette, D. W., Williams, R. C., Slade, G. & Beck, J. D. (1999) Periodontitis–atherosclerosis syndrome: an expanded model of pathogenesis. *Journal of Periodontal Research* **34**, 346–352.
- Page, R. C. & Schroeder, H. E. (1976) Pathogenesis of inflammatory periodontal disease. A summary of current work. *Laboratory Investigation* **34**, 235–249.
- Patel, S. T. & Kent, K. C. (1998) Risk factors and their role in the diseases of the arterial wall. *Seminars in Vascular Surgery* **11**, 156–168.
- Paunio, K., Impivaara, O., Tiekso, J. & Mäki, J. (1993) Missing teeth and ischaemic heart disease in men aged 45–64 years. *European Heart Journal* **14** (Suppl. K), 54–56.
- Persson, R. G., Olsson, O., Pettersson, T. & Renvert, S. (2003) Chronic periodontitis, a significant relationship with acute myocardial infarction. *European Heart Journal* **24**, 2108–2115.
- Ross, R. (1999) Atherosclerosis – An inflammatory disease. *New England Journal of Medicine* **340**, 115–126.
- Salvi, G. E., Lawrence, H. P., Offenbacher, S. & Beck, J. D. (1997) Influence of risk factors on the pathogenesis of periodontitis. *Periodontology 2000* **14**, 173–201.
- Spahr, A., Klein, E., Khuseynova, N., Boeckh, C., Muche, R., Kunze, M., Rothenbacher, D., Pezeshki, G., Hoffmeister, A. & Koenig, W. (2006) Periodontal infections and coronary heart disease: role of periodontal bacteria and importance of total pathogen burden in the Coronary Event and Periodontal Disease (CORODONT) study. *Archives of Internal Medicine* **166**, 554–559.
- World Health Organization (WHO) (1992) *International Statistical Classification of Diseases and Health Related Problems 10*, Vol. 1. Geneva: World Health Organization (WHO).

Address:
Dr. Farnaz Tabrizi
Department of Periodontology
Karolinska Institutet
PO Box 4064
SE 141 04 Huddinge
Sweden
E-mail: Farnaz.Tabrizi@ki.se

Clinical Relevance

Scientific rationale for the study: Several studies have shown an association between CHD and periodontal health. The influences of genetic factors are considerable in both diseases. Therefore, investigating monozygotic twins where the

genetic variability is the same should result in new information regarding the association between the two diseases.

Principal findings and practical implications: Significantly worsened oral health and periodontal conditions were found in the twins with

CHD compared with their healthy siblings. This was shown both as clinical signs and on panoramic radiographs. Further studies in larger populations of monozygotic twins are needed to confirm these findings.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.