J Clin Periodontol 2011; 38: 236–242 doi: 10.1111/j.1600-051X.2010.01677.x

Journal of Clinical Periodontology

Association between periodontal infection and obesity: results of the Health 2000 Survey

Tuomas Saxlin[1,2], Pekka Ylöstalo[1], Liisa Suominen-Taipale[3,4], Satu Männistö[4] and Matti Knuuttila[1,5]

[1]Institute of Dentistry, University of Oulu, Oulu, Finland; [2]Finnish Doctoral Program of Oral Sciences (FINDOS), Turku, Finland; [3]Institute of Dentistry, University of Turku, Turku, Finland; [4]National Institute for Health and Welfare (THL), Helsinki, Finland; [5]Oral and Maxillofacial Department, Oulu University Hospital, Oulu, Finland

Saxlin T, Ylöstalo P, Suominen-Taipale L, Männistö S, Knuuttila M. Association between periodontal infection and obesity: results of the Health 2000 Survey. J Clin Periodontol 2011; 38: 236–242. doi: 10.1111/j.1600-051X.2010.01677.x

Abstract

Aim: To investigate the role of periodontal infection in obesity in an adult population.

Material and methods: This study was based on a subpopulation of the Health 2000 Survey that included dentate, non-diabetic subjects, aged 30–49 years ($n = 2784$). Obesity was measured using the body mass index (BMI), body fat percentage (BF%) and waist circumference (WC). The extent of periodontal infection was measured using the number of teeth with deepened (4 mm deep or deeper) periodontal pockets and was categorized into four categories (0, 1–3, 4–6, 7 or more).

Results: The number of teeth with deepened periodontal pockets was found to be associated with BMI in an exposure–response manner among the total study population. The association was found among men and women, and also among never-smokers**.** The number of teeth with deepened periodontal pockets was also associated with BF% and WC among never-smokers.

Conclusion: Periodontal infection measured by means of the number of teeth with deepened periodontal pockets appears to be associated with obesity. However, no inferences about causality can be made and further studies are needed to clarify the possible role of periodontal infection in obesity.

Key words: body mass index; obesity; oral health; periodontal infection; periodontitis

Accepted for publication 21 November 2010

In medicine, considerable research has been focused on the role of infections in the aetiology of obesity (Atkinson 2007). However, the knowledge of the possible role of oral infections in the aetiology of obesity is limited. The view that periodontal infection may be associated with obesity is supported by the findings of a study that suggested that periodontal infection is associated with metabolic syndrome and its components, including obesity (D'Aiuto et al. 2008). Also, preliminary analyses using these data have suggested that the relation of periodontal infection to body weight cannot be solely explained by the confounding effect of commonly accepted determinants for body weight (Ylöstalo et al. 2008).

The mechanism of how infection in the periodontium could be associated with weight gain may involve lipopolysaccharide (LPS), endotoxin, of Gram-negative periodontal pathogens. Periodontal infection has been found to be associated with endotoxaemia (Pussinen et al. 2007), which in turn has been suggested to be positively correlated with energy and fat intake (Amar et al. 2008). Also, ingestion of periodontal pathogens along with saliva might play a role (Goodson et al. 2009).

The above aspects suggest the possibility that infection in the periodontium is related to obesity, but evidence for this association is scant. The aim of this cross-sectional study was to investigate the association of periodontal infection measured using the number of teeth with deepened periodontal pockets with obesity in an adult population.

Conflict of interest and source of funding statement

The authors declare that there are no conflicts of interest.

The present study is part of the Health 2000 Survey, organized by the National Institute for Health and Welfare (THL) [former National Public Health Institute (KTL) of Finland] (http://www.terveys2000.fi), and is partly supported by the Finnish Dental Society Apollonia and the Finnish Dental Association. A personal grant from the Finnish Dental Society Apollonia and the Finnish Dental Association is acknowledged by Tuomas Saxlin. Satu Männistö wishes to thank the Academy of Finland (Nos. 118065 and 136895).

Material and Methods

The Health 2000 Survey was carried out in 2000–2001. This nationally representative, cross-sectional survey was con-

ducted by the National Institute for Health and Welfare (THL) [Former National Public Health Institute (KTL) of Finland]. The study population comprised persons aged 30 years or older living in continental Finland ($n = 8028$). The aim of the Health 2000 Survey is to yield information about the health and functional capacity of the adult population in Finland. The data for this research were collected from clinical oral and health examinations, and also by interviews, from self-administered questionnaires and from laboratory measurements. Additional information about the Health 2000 Survey is available in reports published by Aromaa & Koskinen (2004) and Heistaro (2008).

This study was based on a subpopulation of dentate non-diabetic subjects, who were 30–49 years old ($n = 2784$). Diabetes was determined on the basis of information obtained from a health examination and health interview. In addition to previously diagnosed diabetes, subjects were diagnosed to have diabetes, if the fasting glucose was 7.0 mmol/l or more, or if the fasting glucose was normal, but the result of the glucose tolerance test was 11.1 mmol/l or more. Informed consent was obtained from the participants. The Ethical Committee for Epidemiology and Public Health of the Hospital District of Helsinki and Uusimaa approved the study protocol.

Outcome variable

Our primary outcome was obesity measured using body mass index (BMI, kg/m^2). Information about height and weight was obtained primarily by clinical examination. In some situations, this was not possible, in which case information from a questionnaire was used. For the statistical analyses, BMI was categorized into two categories according to the World Health Organization (WHO) definition for obesity: BMI < 30 *versus* 30 or over.

As secondary outcome variables, we used body fat percentage (BF%) and waist circumference (WC). BF was measured during the clinical health examination using a high-precision body composition analyzer, InBody 3.0, Biospace Co. Ltd., Seöul, Korea. For statistical analyses, different cut-off values for obesity of BF% and WC among men and women were used. Men were defined to be obese if the BF% was $> 25\%$ or WC was 102 cm or more. For women, the corresponding cut-off values were 35% and 88 cm. Different cut-off values for men and women were based on the WHO reference standards.

Explanatory variable

Oral clinical examinations were performed by five calibrated dentists in a dental chair using a headlamp, mouth mirror and a WHO periodontal probe in line with WHO instructions. Periodontal infection was assessed by means of the presence of teeth with periodontal pockets of 4 mm deep or deeper. Periodontal pocket depth on probing was measured on four surfaces of each tooth (disto-buccal, mid-buccal, mesio-oral, mid-oral) apart from the third molars and radices, and the deepest pocket depth on each tooth was recorded. The percentual agreement for deepened periodontal pockets was 77% (κ 0.41) in the parallel measurements, where the assessments of the field examiners were compared individually with those of the reference examiner under field conditions (Vehkalahti et al. 2008, p. 19). The results for intra-examiner reliability assessments concerning periodontal pocket measurements showed a κ value of 0.83 (Vehkalahti et al. 2004, p. 29). In this study, the extent of periodontal infection was measured using the number of teeth with deepened (4 mm deep or deeper) periodontal pockets and was categorized into four categories: 0, 1–3, 4–6 and 7 or more.

The presence of dental plaque was used as a secondary explanatory variable. The method for measuring the presence of dental plaque was modified from the method described by Silness & Löe (1964). The presence of dental plaque was measured from three indicator teeth on one surface each as follows: the buccal surface on the most posterior tooth on the upper right side, the lingual surface on the most posterior tooth on the lower left side and the buccal surface on the lower left canine. The presence of dental plaque was categorized into three categories: no visible plaque (given value 0), visible plaque on gingival margins only (given value 1) or visible plaque also elsewhere (given value 2). The highest value of any of the indicator teeth was recorded. The percentual agreement for the presence of dental plaque was 58% (κ 0.36) in the parallel measurements between the five examiners and the reference examiner under field conditions (Vehkalahti et al. 2008, p.19). The results for intra-examiner reliability assessments related to the presence of dental plaque showed a κ value of 0.79 (Vehkalahti et al. 2004, p. 29).

Other variables

The number of teeth was categorized into three categories as follows: 1–20, 21–25 and 26 or more. Age was used in the analyses as a continuous variable. Education was categorized into three categories. Basic education included those who had less than a high school education and did not have formal vocational qualifications. Intermediate education included those who had graduated from high school. The highest category of education included subjects with a university degree or who had graduated from polytechnics.

Smoking was categorized into four categories: daily smokers, occasional smokers, subjects who had quit smoking and subjects who had never smoked. The frequency of physical exercise was categorized into four categories: daily or four to six times a week, two to three times a week, once a week and two to three times a month or less frequently. Self-reported health was categorized into two categories: good or fairly good and moderate, fairly poor or poor.

Information about diet was obtained from a validated (Männistö et al. 1996, Paalanen et al. 2006) food frequency questionnaire (FFQ). The FFQ consisted of 128 food items to assess the subjects' entire diet over the previous 12 months. The items were grouped under 12 subheadings. Nine response options ranged from ''never or rarely'' to ''six or more times per day''. The portion sizes were fixed and, if possible, specified using natural units (e.g. cups of coffee). The daily food consumption of the subject was broken down into their components using the Finnish national food composition database (http://www.fineli.fi/index.php). The daily energy intake (in kcal/day) and the proportions of fats, carbohydrates and proteins of the daily energy intake in percents were entered in the analyses as continuous variables. The basic characteristics of the study population according to the categories of the main explanatory variable are presented in Table 1.

Statistical analyses

We estimated odds ratios and their 95% confidence intervals using logistic

Table 1. Basic characteristics of the study population; proportions/means and their standard errors (in parentheses) in the total population and in the categories of the number of teeth with deepened (4 mm or deeper) periodontal pockets

	Teeth with periodontal pockets ⩾4 mm				
	total ($n = 2784$)	0 ($n = 1143$)	1–3 ($n = 708$)	4–6 ($n = 351$)	⩾7 ($n = 582$)
Age, mean (SE) ($n = 2784$)	39.8 (0.1)	38.8 (0.2)	40.2 (0.2)	40.3 (0.3)	40.9 (0.3)
Gender, % (SE) ($n = 2784$)					
Male	49.9 (0.9)	40.4 (1.5)	49.2 (1.9)	55.4 (2.5)	65.4 (1.9)
Female	50.1 (0.9)	59.6 (1.5)	50.8 (1.9)	44.6 (2.5)	34.6 (1.9)
Education, % (SE) ($n = 2773$)					
Basic	18.3 (0.8)	15.4 (1.0)	16.8 (1.4)	18.5 (2.1)	25.2 (2.0)
Intermediate	41.9 (1.0)	38.8 (1.4)	40.6 (1.9)	42.2 (2.5)	49.1 (1.9)
Higher	39.9 (0.9)	45.8 (1.5)	42.6 (1.7)	39.3 (2.5)	25.7 (1.8)
Number of teeth, % (SE) ($n = 2784$)					
1–20	7.5 (0.5)	7.5 (0.7)	7.4 (1.0)	7.7 (1.4)	7.6 (1.0)
21–25	9.8 (0.6)	8.3 (0.9)	10.9 (1.2)	9.3 (1.6)	11.7 (1.5)
⩾26	82.7 (0.8)	84.2 (1.1)	81.7 (1.5)	83.0 (2.0)	80.8 (1.8)
Smoking, % (SE) ($n = 2773$)					
Daily	29.6 (0.9)	22.5 (1.2)	24.6 (1.8)	34.7 (2.7)	45.8 (2.4)
Occasional	6.6 (0.5)	6.1 (0.7)	7.1 (0.9)	7.5 (1.4)	6.3 (1.0)
Quitted	17.6 (0.7)	18.3 (1.2)	16.1 (1.4)	17.8 (1.9)	17.6 (1.6)
Never	46.3 (0.9)	53.1 (1.5)	52.1 (1.9)	40.0 (2.6)	30.2 (2.3)
Frequency of physical exercise, % (SE) ($n = 2756$)					
Daily or 4–6 times a week	21.9 (0.8)	23.0 (1.3)	23.0 (1.7)	22.1 (2.1)	18.2 (1.7)
2–3 times a week	35.5 (0.9)	35.1 (1.4)	36.1 (1.9)	38.0 (2.6)	34.1 (2.0)
Once a week	19.4 (0.7)	20.9 (1.3)	19.0 (1.4)	15.9 (1.9)	19.3 (1.6)
2–3 times a month or less frequently	23.2 (0.8)	20.9 (1.3)	21.9 (1.6)	24.0 (2.2)	28.5 (2.1)
Self-reported health, % (SE) ($n = 2772$)					
Good or fairly good	80.2 (0.8)	81.8 (1.2)	81.1 (1.6)	81.8 (1.9)	75.1 (1.8)
Moderate, fairly poor or poor	19.8 (0.8)	18.2 (1.2)	19.0 (1.6)	18.2 (1.9)	24.9 (1.8)
Daily energy intake (kcal), mean (SE) ($n = 2599$)	2333.5 (15.4)	2346.0 (28.0)	2302.1 (30.2)	2356.7 (50.0)	2332.5 (33.7)
Daily energy intake (kJ), mean (SE) ($n = 2599$)	9763.3 (64.4)	9815.7 (117.0)	9631.9 (126.4)	9860.5 (209.1)	9759.2 (141.0)
Proportion of fats (%) of the daily energy intake, mean (SE) ($n = 2599$)	37.1 (0.1)	36.8 (0.1)	36.8 (0.2)	37.4 (0.3)	37.8 (0.2)
Proportion of carbohydrates (%) of the daily energy intake, mean (SE) ($n = 2599$)	43.9 (0.1)	44.4 (0.2)	44.1 (0.2)	43.2 (0.3)	42.9 (0.3)
Proportion of proteins (%) of the daily energy intake, mean (SE) ($n = 2599$)	17.1 (0.0)	17.1 (0.1)	17.1 (0.1)	17.2 (0.1)	17.3 (0.1)
Body mass index, % (SE) ($n = 2783$)					
<30.0	84.6 (0.7)	87.3 (1.0)	85.7 (1.3)	84.7 (2.0)	78.3 (1.9)
30.0 or more	15.4 (0.7)	12.7 (1.0)	14.3 (1.3)	15.3 (2.0)	21.7 (1.9)
Body mass index, among men, % (SE) ($n = 1301$)					
<30.0	84.3 (1.0)	88.3 (1.4)	86.1 (2.0)	85.9 (2.5)	77.3 (2.4)
30.0 or more	15.7 (1.0)	11.7 (1.4)	13.9 (2.0)	14.1 (2.5)	22.7 (2.4)
Body mass index, among women, % (SE) ($n = 1482$)					
<30.0	85.0 (1.0)	86.7 (1.4)	85.3 (2.0)	83.2 (3.0)	80.2 (2.9)
30.0 or more	15.0 (1.0)	13.3 (1.4)	14.7 (2.0)	16.8 (3.0)	19.8 (2.9)
Body fat percentage, among men, % (SE) ($n = 1301$)					
25.0 or less	81.5 (1.1)	85.6 (1.4)	83.5 (2.2)	78.8 (3.3)	76.3 (2.3)
>25.0	18.5 (1.1)	14.4 (1.4)	16.5 (2.2)	21.2 (3.3)	23.7 (2.3)
Body fat percentage, among women, % (SE) ($n = 1483$)					
⩽35.0	81.0 (1.0)	83.4 (1.5)	79.4 (2.1)	76.7 (3.4)	79.3 (2.9)
>35.0	19.0 (1.0)	16.6 (1.5)	20.6 (2.1)	23.3 (3.4)	20.7 (2.9)
Waist circumference (cm), among men, % (SE) ($n = 1301$)					
<102	76.4 (1.1)	80.1 (1.6)	78.5 (2.3)	75.0 (3.0)	70.8 (2.4)
102 or more	23.6 (1.1)	19.9 (1.6)	21.5 (2.3)	25.0 (3.0)	29.2 (2.4)
Waist circumference (cm), among women, % (SE) ($n = 1483$)					
<88	67.8 (1.2)	69.6 (1.8)	70.6 (2.7)	64.1 (3.7)	59.7 (3.5)
88 or more	32.2 (1.2)	30.4 (1.8)	29.4 (2.7)	35.9 (3.7)	40.3 (3.5)

regression models. The selection of covariates was based on knowledge about the factors that might be associated with body weight and thus confound the association between periodontal infection and obesity. Associations between the number of teeth with deepened (4 mm deep or deeper) periodontal pockets and BMI, BF% and WC were analysed and stratified according to gender. Additional analyses among never-smokers were carried out, based on knowledge that smoking may modify associations with obesity, and the control of the confounding effect of smoking may otherwise be insufficient.

A stratified two-stage cluster sampling design was used in the survey. Weighting of the sample was based on post-stratification according to gender, age and region. The data analyses were

performed using the SUDAAN statistical package version 9.0.1 (Research Triangle Institute, Raleigh, NC, USA) to take into account the two-stage cluster sampling design.

Results

Among the total study population, the number of teeth with deepened (4 mm deep or deeper) periodontal pockets was associated with BMI after controlling for potential confounding factors such as gender, age, education, number of teeth, smoking, the frequency of physical exercise, self-reported health, daily energy intake and the proportions of fats, carbohydrates and proteins of the daily energy intake. Risk estimates were statistically significant only in the highest category of the number of teeth with deepened periodontal pockets (seven or more) (Table 2).

We performed stratified analyses according to gender, and the number of teeth with deepened periodontal pockets was found to be associated with BMI among both genders, although the association was weaker among women (Table 3).

The number of teeth with deepened periodontal pockets was associated with BMI also among a subpopulation of never-smokers. The association followed a pattern fairly similar to the one found in the total study population, although the association was stronger among never-smokers (Tables 2 and 3).

A high number of teeth with deepened (4 mm deep or deeper) periodontal pockets was found to be associated with other measures of obesity, such as BF% and WC, among never-smokers (Tables 4 and 5). This association was found among both genders, although being more inconsistent among women (Tables 4 and 5).

We also performed analyses where we studied the association of the presence of dental plaque with BMI. This association was found to be weak and inconsistent. A similar pattern was found in the stratified analyses according to gender and also among never-smokers (Table 6).

Table 2. Association between number of teeth with deepened (4 mm or deeper) periodontal pockets and obesity (body mass index 30 or more)

	Obesity	
	unadjusted OR (95% CI)	adjusted OR (95% CI)
Total population		
Number of teeth with deepened periodontal pockets*		
0	1	1
1–3	1.1 (0.9–1.5)	1.2 (0.9–1.6)
4–6	1.2 (0.9–1.8)	1.3 (0.9–1.9)
⩾7	1.9 (1.4–2.5)	1.8 (1.3–2.4)
Never-smokers		
Number of teeth with deepened periodontal pockets†		
0	1	1
1–3	1.5 (1.0–2.3)	1.5 (1.0–2.3)
4–6	1.4 (0.8–2.4)	1.3 (0.7–2.4)
⩾7	2.5 (1.7–3.8)	2.4 (1.5–3.8)

Unadjusted and adjusted odds ratios (OR) with 95% confidence intervals (95% CI).
*Adjusted for gender, age (continuous variable), education, number of teeth, smoking, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables), effective $n = 2571$.
†Adjusted for gender, age (continuous variable), education, number of teeth, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables), effective $n = 1217$.

Table 3. Association between number of teeth with deepened (4 mm or deeper) periodontal pockets and obesity (body mass index 30 or more), stratified according to gender

	Obesity		Obesity	
	OR	95% CI	OR	95% CI
Total population				
Number of teeth with deepened periodontal pockets*	among men (effective $n = 1176$)		among women (effective $n = 1395$)	
0	1.0		1.0	
1–3	1.2	0.8–2.0	1.2	0.8–1.9
4–6	1.5	0.9–2.5	1.2	0.7–2.0
⩾7	2.2	1.4–3.4	1.3	0.8–2.2
Never-smokers				
Number of teeth with deepened periodontal pockets†	Among men (effective $n = 468$)		Among women (effective $n = 749$)	
0	1.0		1.0	
1–3	1.3	0.6–2.6	1.7	0.9–3.1
4–6	1.4	0.6–3.3	1.3	0.5–3.0
⩾7	2.8	1.4–5.4	2.0	0.9–4.2

Adjusted odds ratios (OR) with 95% confidence intervals (95% CI).
*Adjusted for age (continuous variable), education, number of teeth, smoking, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables).
†Adjusted for age (continuous variable), education, number of teeth, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables).

Discussion

The association between obesity and periodontitis/periodontal disease has been suggested in several cross-sectional studies (Saito et al. 2001, Dalla Vecchia et al. 2005, Genco et al. 2005, Khader et al. 2009, Han et al. 2010), and also using these data (Ylöstalo et al. 2008). However, the possible role of oral infections in the aetiology of obesity is still unclear. This study showed that the number of teeth with deepened (4 mm deep or deeper) periodontal pockets was associated with obesity in an exposure–response manner: the higher the number of teeth with deepened periodontal pockets, the higher the proportion of subjects with BMI 30 or over. This association was found among the total study population, among men, among women and among a subpopulation of never-smokers, although there was a slight deviation from the exposure–response association among

Table 4. Association between number of teeth with deepened (4 mm or deeper) periodontal pockets and obesity (body fat percentage (BF %) > 25% among men and > 35% among women)

	Obesity		Obesity	
	OR	95% CI	OR	95% CI
Total population				
Number of teeth with deepened periodontal pockets*	Among men (BF % > 25%, effective $n = 1176$)		Among women (BF % > 35%, effective $n = 1396$)	
0	1.0		1.0	
1–3	1.1	0.8–1.6	1.4	0.9–2.1
4–6	1.7	1.0–2.8	1.3	0.8–2.1
⩾7	1.7	1.1–2.4	1.0	0.6–1.6
Never-smokers				
Number of teeth with deepened periodontal pockets†	Among men (effective $n = 468$)		Among women (effective $n = 750$)	
0	1.0		1.0	
1–3	1.1	0.6–2.0	1.5	0.9–2.5
4–6	2.3	1.1–4.6	0.9	0.4–2.0
⩾7	1.5	0.9–2.6	1.3	0.6–2.5

Adjusted odds ratios (OR) with 95% confidence intervals (95% CI).
*Adjusted for age (continuous variable), education, number of teeth, smoking, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables).
†Adjusted for age (continuous variable), education, number of teeth, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables).

Table 5. Association between number of teeth with deepened (4 mm or deeper) periodontal pockets and obesity [waist circumference (WC) ⩾ 102 cm among men and ⩾ 88 cm among women]

	Obesity		Obesity	
	OR	95% CI	OR	95% CI
Total population				
Number of teeth with deepened periodontal pockets*	Among men (WC ⩾ 102 cm, effective $n = 1176$)		Among women (WC ⩾ 88 cm, effective $n = 1396$)	
0	1.0		1.0	
1–3	1.0	0.7–1.5	0.9	0.6–1.2
4–6	1.5	1.0–2.3	1.1	0.7–1.6
⩾7	1.5	1.0–2.1	1.1	0.8–1.7
Never-smokers				
Number of teeth with deepened periodontal pockets†	Among men (effective $n = 468$)		Among women (effective $n = 750$)	
0	1.0		1.0	
1–3	1.0	0.5–1.8	1.3	0.8–2.0
4–6	1.6	0.8–3.1	1.0	0.5–1.9
⩾7	2.3	1.2–4.3	2.0	1.1–3.5

Adjusted odds ratios (OR) with 95% confidence intervals (95% CI).
*Adjusted for age (continuous variable), education, number of teeth, smoking, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables).
†Adjusted for age (continuous variable), education, number of teeth, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables).

never-smokers. The number of teeth with deepened periodontal pockets was also found to be associated in an almost similar manner with obesity among never-smokers when obesity was measured using BF% or WC.

We adjusted for several factors that potentially associate with obesity. Interestingly, adjustments for intake of energy, composition of diet and also other potential determinants for body weight seemed to have virtually no effect on the strength of the association of periodontal infection with BMI. This suggests that this association may not be confounded by the commonly accepted risk factors for obesity. In addition, the fact that periodontal infection but not essentially dental plaque was associated with BMI supports the conception that the association found may be specifically related to periodontal infection, not to overall poor oral health. Naturally, another possibility is that the association between periodontal infection and obesity is explained by some unknown extraneous factor or known factors that are difficult to adjust for, such as behavioural factors, for instance. The possibility that factors exist that are not controllable in a normal setting would be in line with the findings of the study by Hujoel et al. (2006), where a lack of daily flossing was associated in an exposure–response manner with overweight, obesity and morbid obesity.

The direct cause of obesity is a long-term positive imbalance between energy intake and consumption. Although the present study did not investigate the possible mechanisms of how infection in the periodontium could have an effect on the development of obesity, there is evidence of possible biological explanations. One of these, for instance, may be the effect of LPS, endotoxin, of the outer cell membrane of Gram-negative bacteria. Subgingival biofilm may provide a substantial and continuous source of circulating endotoxin (Page et al. 1997) and an elevated systemic concentration of endotoxin (endotoxaemia) has been reported to be associated with periodontal infection (Pussinen et al. 2007). Endotoxaemia has been found to be associated with weight gain in experimental studies. Cani et al. (2007) found that a continuous 4-week subcutaneous infusion of LPS to mice that were fed a normal diet resulted in weight gain, which was similar to the one of mice on a 4-week high-fat diet regimen. Tsukumo et al. (2007), on the other hand, reported that mice with a loss-of-function mutation in toll-like receptor 4 were protected against the development of diet-induced obesity. In addition, endotoxaemia has been found to correlate positively with energy and fat intake (Amar et al. 2008). However, the results of our study do not support the correlation between endotoxaemia and energy and fat intake.

Another possible mechanism is the ingestion of periodontal pathogens. This

Table 6. Association between the presence of dental plaque and obesity (body mass index 30 or more)

	Obesity		Obesity		Obesity	
	OR	95% CI	OR	95% CI	OR	95% CI
Total population						
Presence of dental plaque*	Total (effective $n = 2565$)		Among men (effective $n = 1171$)		Among women (effective $n = 1394$)	
None	1.0		1.0		1.0	
Plaque on gingival margins only	1.3	1.0–1.7	1.3	0.9–1.9	1.2	0.9–1.7
Plaque also elsewhere	1.4	0.9–2.2	1.5	0.9–2.6	1.0	0.5–2.1
Never-smokers						
Presence of dental plaque†	Total (effective $n = 1214$)		Among men (effective $n = 466$)		Among women (effective $n = 748$)	
None	1.0		1.0		1.0	
Plaque on gingival margins only	1.4	1.0–2.0	1.5	0.7–3.1	1.3	0.8–2.1
Plaque also elsewhere	1.2	0.6–2.5	1.1	0.4–3.0	1.5	0.6–3.9

Adjusted odds ratios (OR) with 95% confidence intervals (95% CI).
*Adjusted for age (continuous variable), education, number of teeth, smoking, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables).
†Adjusted for age (continuous variable), education, number of teeth, frequency of physical exercise, self-reported health, daily energy intake (continuous variable) and proportions of fats, carbohydrates and proteins of the daily energy intake (continuous variables).

view is supported by a study by Goodson et al. (2009), in which they found one pathogen, also found in the periodontal biofilm, *Selenomonas noxia*, which, at levels $>1.05\%$ of the total salivary bacteria, could identify obese individuals with a high sensitivity and specificity. *S. noxia* belongs to the phylum *Firmicutes*, whose relative proportion of the gut microbiota has been reported to be elevated in obese individuals (Ley et al. 2006).

Validity issues

This study obviously has some limitations. One of them is the cross-sectional study design, which means that any definite conclusions about causality cannot be made. This is due to the inability to assess the temporal sequence between the supposed cause and effect. Another is that the measurement of periodontal infection was based on one clinical parameter, pocket depth. However, the presence of deepened periodontal pockets is a widely used method in clinical work and in research to measure the existing infection and treatment need. The boundary value of 4 mm is a commonly accepted limit for pathological deepening of periodontal sulcus. Also, the dental plaque variable has some limitations. One of them admittedly is the slight crudeness of the variable as it is measured only from three indicator teeth, which may also cause some inconsistency in the results.

We used multivariate models, restrictions and stratifications to control confounding in this study. The rationale for separate analyses for never-smokers was that smoking has been suggested to be associated with lower body weight (Albanes et al. 1987), and also that the cessation of smoking has been suggested to be associated with weight gain (Flegal et al. 1995). We also performed analyses stratified according to gender, as women are known to have greater amount of fat tissue than males. We also included only the age group of 30–49 years in order to control age-related confounding. Diabetic and prediabetic subjects were excluded due to the complex interrelations of diabetes with both periodontitis and obesity, which would have been otherwise difficult to control in the analyses. However, despite the fairly profound adjustments, it is possible that the association between the number of teeth with deepened (4 mm deep or deeper) periodontal pockets and obesity is due to residual confounding related to the controlled factors and unknown factors, such as attitudinal and behavioural factors.

Conclusions

The number of teeth with deepened (4 mm deep or deeper) periodontal pockets was associated with obesity in this low-risk population. However, due to the cross-sectional study design, the possible causal role of periodontal infection in the aetiology of weight gain cannot be assessed. Possible biological explanations for this association have been suggested, but also residual confounding as an explanation cannot be overlooked. The lack of longitudinal and intervention studies warrants further investigation on the association between periodontal infection and obesity.

References

Albanes, D., Jones, D. Y., Micozzi, M. S. & Mattson, M. E. (1987) Associations between smoking and body weight in the US population: analysis of NHANES II. *American Journal of Public Health* **77**, 439–444.

Amar, J., Burcelin, R., Ruidavets, J. B., Cani, P. D., Fauvel, J., Alessi, M. C., Chamontin, B. & Ferrieres, J. (2008) Energy intake is associated with endotoxemia in apparently healthy men. *The American Journal of Clinical Nutrition* **87**, 1219–1223.

Aromaa, A. & Koskinen, S. (eds). (2004) *Health and functional capacity in Finland. Baseline results of the Health 2000 Health Examination Survey B12/2004*. Helsinki: Publications of the National Public Health Institute.

Atkinson, R. L. (2007) Viruses as an etiology of obesity. *Mayo Clinic Proceedings* **82**, 1192–1198.

Cani, P. D., Amar, J., Iglesias, M. A., Poggi, M., Knauf, C., Bastelica, D., Neyrinc, A. M., Fava, F., Tuohy, K. M., Chabo, C., Waget, A., Delmee, E., Cousin, B., Sulpice, T., Chamontin, B., Ferrieres, J., Tanti, J.-F., Gibson, G. R., Casteila, L., Delzenne, N. M., Alessi, M. C. & Burcelin, R. (2007) Metabolic endotoxemia initiates obesity and insulin resistance. *Diabetes* **56**, 1761–1772.

D'Aiuto, F., Sabbah, W., Netuveli, G., Donos, N., Hingorani, A. D., Deanfield, J. & Tsakos, G. (2008) Association of the metabolic syndrome with severe periodontitis in a large U.S. population-based survey. *Journal of Clinical Endocrinology and Metabolism* **93**, 3989–3994.

Dalla Vecchia, C. F., Susin, C., Rösing, C. K., Oppermann, R. V. & Albandar, J. M. (2005) Overweight and obesity as risk indicators for periodontitis in adults. *Journal of Periodontology* **76**, 1721–1728.

Flegal, K. M., Troiano, R. P., Pamuk, E. R., Kuczmarski, R. J. & Campbell, S. M. (1995) The influence of smoking cessation on the prevalence of overweight in the United States. *New England Journal of Medicine* **333**, 1165–1170.

Genco, R. J., Grossi, S. G., Ho, A., Nishimura, F. & Murayama, Y. (2005) A proposed model linking inflammation to obesity, diabetes, and periodontal infections. *Journal of Periodontology* **76**, 2075–2084.

Goodson, J. M., Groppo, D., Halem, S. & Carpino, E. (2009) Is obesity an oral bacterial disease? *Journal of Dental Research* **88**, 519–523.

Han, D.-H., Lim, S.-Y., Sun, B.-C., Paek, D.-M. & Kim, H.-D. (2010) Visceral fat area defined obesity and periodontitis among Koreans. *Journal of Clinical Periodontology* **37**, 172–179.

Heistaro, S. (ed). (2008) *Methdology report. Health 2000 Survey B26/2008*. Helsinki: Publications of the National Public Health Institute.

Hujoel, P. P., Cunha-Cruz, J. & Kressin, N. R. (2006) Spurious associations in oral epidemiological research: the case of dental flossing and obesity. *Journal of Clinical Periodontology* **33**, 520–523.

Khader, Y. S., Bawaldi, H. A., Haroun, T. F., Alomari, M. & Tayyem, R. F. (2009) The association between periodontal disease and obesity among adults in Jordan. *Journal of Clinical Periodontology* **36**, 18–24.

Ley, R. E., Turnbaugh, P. J., Klein, S. & Gordon, J. I. (2006) Microbial ecology: human gut microbes associated with obesity. *Nature* **444**, 1022–1023.

Männistö, S., Virtanen, M., Mikkonen, T. & Pietinen, P. (1996) Reproducibility and validity of a food frequency questionnaire in a case–control study on breast cancer. *Journal of Clinical Epidemiology* **49**, 401–409.

Paalanen, L., Männistö, S., Virtanen, M. J., Knekt, P., Räsänen, L., Montonen, J. & Pietinen, P. (2006) Validity of a food frequency questionnaire varied by age and body mass index. *Journal of Clinical Epidemiology* **59**, 994–1001.

Page, R. C., Offenbacher, S., Schroeder, H. E., Seymour, G. J. & Kornman, K. S. (1997) Advances in the pathogenesis of periodontitis: summary of developments, clinical implications and future directions. *Periodontology 2000* **14**, 216–248.

Pussinen, P. J., Tuomisto, K., Jousilahti, P., Havulinna, A. S., Sundvall, J. & Salomaa, V. (2007) Endotoxemia, immune response to periodontal pathogens, and systemic inflammation associate with incident cardiovascular disease events. *Arteriosclerosis, Thrombosis, and Vascular Biology* **27**, 1433–1439.

Saito, T., Shimazaki, Y., Koga, T., Tsuzuki, M. & Ohshima, A. (2001) Relationship between upper body obesity and periodontitis. *Journal of Dental Research* **80**, 1631–1636.

Silness, J. & Löe, H. (1964) Periodontal disease in pregnancy II. Correlation between oral hygiene and periodontal condition. *Acta Odontologica Scandinavia* **22**, 121–135.

Tsukumo, D. M. L., Carvalho-Filho, M. A., Carvalheira, J. B. C., Prada, P. O., Hirabara, S. M., Schenka, A. A., Araujo, E. P., Vassallo, J., Curi, R., Velloso, L. A. & Saad, M. J. A. (2007) Loss-of-function mutation in toll-like receptor 4 prevents diet-induced obesity and insulin resistance. *Diabetes* **56**, 1986–1998.

Vehkalahti, M., Knuuttila, M. & Hausen, H. (2004) Kliinisten mittausten laadun varmistaminen (Quality assurance of clinical examinations). In: Suominen-Taipale, L., Nordblad, A., Vehkalahti, M. & Aromaa, A. (eds). *Suomalaisten aikuisten suunterveys, Terveys 2000 – tutkimus (Oral Health of Finnish Adults, Health 2000 Health Examination Survey) B16/2004*, pp. 24–32. Helsinki: Publications of the National Public Health Institute.

Vehkalahti, M., Knuuttila, M. & Hausen, H. (2008) Quality assurance. In: Suominen-Taipale, L., Nordblad, A., Vehkalahti, M. & Aromaa, A. (eds). *Oral health in the Finnish population, Health 2000 Survey B25/2008*, pp. 17–20. Helsinki: Publications of the National Public Health Institute.

Ylöstalo, P., Suominen-Taipale, L., Reunanen, A. & Knuuttila, M. (2008) Association between body weight and periodontal infection. *Journal of Clinical Periodontology* **35**, 297–304.

Address:
Tuomas Saxlin
Institute of Dentistry
University of Oulu
PO Box 5281
90014 Oulu
Finland
E-mail: tuomas.saxlin@oulu.fi

Clinical Relevance

Scientific rationale for the study: Overweight and obesity have been shown to be associated with periodontal infection in several cross-sectional studies, whereas the knowledge of the possible role of oral infections in the aetiology of obesity is limited.

Principal findings: The number of teeth with deepened (4 mm deep or deeper) periodontal pockets was found to be associated with BMI in an exposure–response manner among the total study population. The association was found among men and women, and also among never-smokers. The number of teeth with deepened periodontal pockets was also associated with obesity among never-smokers when obesity was measured by means of BF% or WC.

Practical implications: Periodontal infection measured using the number of teeth with deepened periodontal pockets was found to be associated with obesity. However, due to the cross-sectional study design, the possible causal nature of this association remains to be unclear.