

Association between body weight and periodontal infection

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Ylöstalo P, Suominen-Taipale L, Reunanen A, Knuuttila M. Association between body weight and periodontal infection. *J Clin Periodontol* 2008; 35: 297–304. doi: 10.1111/j.1600-051X.2008.01203.x.

Abstract

Background: Besides being a risk factor for cardiovascular diseases, certain cancers and type II diabetes, obesity has been suggested to be a risk factor for periodontitis. A number of epidemiological studies have studied the association between obesity and periodontitis, but the results have been partly inconclusive. The aim of this study was to examine the association of body weight with periodontal infection.

Material and Methods: The association between body weight and periodontal infection was examined using a nationally representative Health 2000 Health Examination Survey. The study was based on a subpopulation of dentate non-diabetic subjects aged 30–49 ($n = 2841$). Periodontal infection was measured by the number of teeth with periodontal pockets of 4 mm or deeper and 6 mm or deeper. Body weight was measured using body mass index (BMI).

Results: We detected a weak exposure–response association of BMI with teeth with deepened periodontal pockets after controlling for smoking habits by restricting the sample to subjects who have never smoked and for other potential confounders by including them in the multivariate models.

Conclusions: The results showed an association between body weight and periodontal infection among the non-diabetic, non-smoking population aged 30–49. Additional research is needed to determine the nature of this association.

Key words: obesity; periodontal infection; weight

Accepted for publication 21 December 2007

Besides being a risk factor for cardiovascular diseases, certain cancers and type II diabetes; obesity has also been suggested to be a risk factor for periodontitis (Saito et al. 2001, Genco et al. 2005, Nishida et al. 2005). The mechanism of how obesity affects the periodontium is currently poorly understood,

but what is known is that obesity has several harmful biological effects that might be related to the pathogenesis of periodontitis. According to current knowledge, the adverse effects of obesity on the periodontium might be mediated through impaired glucose tolerance, dyslipidaemia or through increased levels of various bioactive substances secreted by adipose tissue (Saito & Shimazaki 2007).

A number of epidemiological studies have examined the association between obesity and periodontitis (Saito et al. 2001, Al-Zahrani et al. 2003, Wood et al. 2003, Dalla Vecchia et al. 2005, Genco et al. 2005, Nishida et al. 2005, Linden et al. 2007). The results of the studies have been inconsistent. For example, Saito et al. (2001), Genco et al. (2005) and Nishida et al. (2005) reported an overall association, whereas

Al-Zahrani et al. (2003) found an association among young subjects and Dalla Vecchia et al. (2005) found one among non-smoking women. Linden et al. (2007) reported an association between a high body mass index (BMI) and periodontitis in a group of 60–70-year-old men. The results of some of these studies could be interpreted as support for the presence of biological pathways whereas the results of other studies do not unequivocally support the presence of a causal association.

The existence of a causal relation between obesity and periodontitis would have several implications. It would elucidate the role of the systemic condition in the pathogenesis of periodontitis, especially with reference to the role of an altered lipid profile and inflammatory condition. It would also clarify the role of obesity as a confounder in the relation

Conflict of interest and source of funding statement

The authors declare that they have no conflict of interests.

The present study is part of the Health 2000 Health Examination Survey, organized by the National Public Health Institute (KTL) of Finland (<http://www.ktl.fi/health2000>), and partly supported by the Finnish Dental Society Apollonia and the Finnish Dental Association. Personal grants to Pekka Ylöstalo by the Finnish Dental Society are acknowledged.

of periodontitis to other diseases or conditions.

The aim of this study was to examine the association of body weight with periodontal infection among a Finnish population aged 30–49 years.

Material and Methods

A nationally representative Health 2000 Health Examination Survey was carried out in 2000–2001 by the National Public Health Institute of Finland. The study population comprised persons aged 30 years or older living in continental Finland ($n = 8028$). Eighty-seven per cent of the sample participated in an interview and 79% in a health examination. Informed consent was obtained from the participants. The ethical committee of Helsinki University Hospital approved the study protocol.

The data for this research were collected by interviews and clinical oral and health examinations. Oral examinations were made by five calibrated dentists. The study was based on a subpopulation of dentate non-diabetic subjects aged under 50 (effective $n = 2841$). Additional information about the Health 2000 Health Examination Survey is available in the published report at: <http://www.ktl.fi/terveys2000/julkaisut/baseline.pdf>.

Outcome variables

Five calibrated dentists performed a clinical oral examination in a dental chair using a headlamp, mouth mirror and a WHO periodontal probe in line with the WHO instructions. The clinical oral examination involved assessing the condition of the periodontium and teeth. Periodontal pocket depth on probing was measured on four surfaces of each tooth apart from the third molars, but only the most severe site of each tooth was recorded. Data on probing depth were lacking for 30 subjects.

We chose the number of teeth with periodontal pockets of 4 mm or deeper as a primary outcome variable. We used the number of teeth with periodontal pockets of 6 mm or deeper and the number of carious teeth as secondary outcomes. The percentual agreement between the examiners and the reference examiner in periodontal pockets was 82% (κ 0.32) (Vehkalahti et al. 2004).

Dental caries was examined visually after blowing the teeth dry and by

probing on each surface of all teeth. Caries was also diagnosed by surface but was recorded by the tooth level. The number of carious teeth included radices and all teeth with carious lesions reaching the dentine. The percentual agreement between the examiners and the reference examiner in the state of the teeth was 95% (κ 0.86) (Vehkalahti et al. 2004).

Explanatory variables

Body weight was measured using BMI, which is a measure of body weight in relation to height (kg/m^2). BMI was categorized into quintiles [first quintile, <22.3; second quintile, 22.3–24.3; third quintile, 24.3–26.3; fourth quintile, 26.3–29.1; and fifth quintile 29.1 or more (rounded values)]. Data needed to calculate BMI were missing for one subject. The correlation between BMI (as a continuous variable) and body fat was 0.90 and the correlation between BMI and waist circumference was 0.87. Body fat was measured using a high-precision body composition analyser, Biospace InBody 3.0, Seoul, South Korea.

Behavioural factors included tooth-brushing frequency (categorized as twice a day or more, daily, more seldom) and dental attendance pattern (categorized into those who regularly have dental check-ups *versus* those who never used dental health services or used them in a symptom-based manner).

Smoking habits were categorized in the following manner: daily smokers, irregular smokers, former smokers who had quit 1–12 months ago, former smokers who had quit smoking more than 1 year ago and those who had never smoked. We controlled for the effect of smoking habits by restricting the analyses to a subgroup of never smokers in order to achieve complete controlling. The results for the whole cohort, where we controlled for smoking habits by including them as a covariate in the multivariate model, are also presented to allow comparisons between the results of this study and those of others.

The presence of dental plaque was assessed from three teeth at one site, each as follows: from the buccal surface of teeth 17–14, from the lingual surfaces of teeth 37–34 and the buccal surface of tooth 33. The presence of dental plaque was classified into three categories (no visible plaque, visible plaque in gingival

margins, visible plaque also elsewhere) according to the highest recorded value. The number of teeth (as a continuous variable) was treated in the regression models as the offset variable.

Diabetes was determined on the basis of information obtained from the health interview. The question posed was “Do you have diabetes diagnosed by a doctor?” with the answer options being yes/no. Diabetic patients were excluded because interrelations between obesity and diabetes, which depend on the type and the severity of the diabetes, can be complex and prevent accurate control.

Education was categorized into three categories: basic education, secondary education and higher education. The basic characteristics of the study population are shown in Table 1.

Statistical methods

We estimated relative risks (RR) and confidence intervals (95% CI) using the Poisson regression models. We performed an additional analysis (among never smokers) where we excluded periodontally healthy subjects from the analysis to overcome possible problems related to the distribution assumption. The distributions of the main outcome variable are shown in Figs 1 and 2.

Possible modification (for the three outcome variables) was studied by adding product terms for BMI and the explanatory variables one by one in the model including all explanatory variables (BMI included as a categorized variable, number of teeth as the offset variable, age as a continuous variable, other variables as categorized variables as shown in Table 2).

SUDAAN statistical package was used in the analyses to take into account two-stage cluster sampling. Weights were used to correct the effects of non-response. The weighting of the sample was based on post-stratification according to gender, age and region.

Results

The unadjusted RR and their 95% CI for explanatory variables are presented in Table 2. Table 3 presents the adjusted mean number of teeth with periodontal pockets in the different quintiles of BMI. Subjects who had never smoked and belonged to the highest quintile had on average 1.0 teeth with periodontal

Table 1. Basic characteristics of the study population ($n = 2841$); proportions/means and their standard errors (in parentheses) in the total population and in different categories (quintiles) of body mass index (BMI)

	Body mass index					
	total ($n = 2841$)	I quintile (lowest) ($n = 570$)	II quintile ($n = 569$)	III quintile ($n = 572$)	IV quintile ($n = 562$)	V quintile (highest) ($n = 567$)
Age (mean) (2841)	39.8 (0.1)	38.3 (0.2)	39.5 (0.2)	39.8 (0.2)	40.7 (0.3)	40.7 (0.3)
Gender, proportion of males (%) (2841)	50.1 (0.9)	30.2 (2.0)	47.4 (2.1)	55.9 (2.0)	61.3 (2.1)	55.1 (2.2)
Educational level (%) (2840)						
Low (516)	18.4 (0.8)	16.7 (1.6)	15.4 (1.5)	17.6 (1.5)	19.9 (1.7)	22.1 (1.9)
Intermediate (1181)	42.0 (1.0)	35.1 (2.0)	42.2 (2.0)	44.4 (2.0)	44.1 (2.1)	44.0 (2.3)
High (1143)	39.7 (0.9)	48.3 (2.0)	42.5 (2.0)	38.0 (2.1)	36.0 (2.0)	33.8 (2.0)
BMI (mean) (2840)	26.0 (0.1)	20.8 (0.1)	23.3 (0.0)	25.3 (0.0)	27.6 (0.0)	32.9 (0.2)
Number of teeth (mean) (2841)	26.5 (0.1)	27.0 (0.2)	26.8 (0.2)	26.5 (0.2)	26.5 (0.2)	25.9 (0.2)
Number of teeth with periodontal pocket 4 mm or more (mean) (2811)	3.8 (0.2)	3.2 (0.2)	3.4 (0.2)	3.5 (0.2)	4.1 (0.3)	4.7 (0.3)
Number of teeth with periodontal pocket 6 mm or more (mean) (2811)	0.4 (0.0)	0.4 (0.1)	0.4 (0.1)	0.3 (0.1)	0.5 (0.01)	0.6 (0.1)
Number of carious teeth (mean) (2841)	0.7 (0.0)	0.7 (0.1)	0.5 (0.1)	0.7 (0.1)	0.8 (0.1)	0.9 (0.1)
Smoking history (%) (2839)						
Proportion of never smokers (1325)	46.2 (0.9)	48.4 (2.0)	51.3 (2.1)	46.4 (2.2)	43.6 (2.2)	41.4 (2.1)
Proportion of current smokers (833)	36.2 (0.9)	38.3 (2.0)	32.2 (2.0)	37.8 (2.2)	37.7 (2.3)	34.9 (2.1)
Proportion of former smokers (681)	17.7 (0.7)	13.4 (1.5)	16.5 (1.6)	15.7 (1.5)	18.7 (1.8)	23.8 (1.7)
Tooth brushing (%) (2745)						
Proportion of those who brush teeth at least twice a day (1793)	64.4 (1.1)	74.6 (2.0)	70.8 (2.2)	62.1 (2.0)	59.2 (2.0)	55.6 (2.1)
Once a day (792)	29.4 (0.9)	20.3 (1.8)	26.3 (2.1)	32.2 (1.9)	32.5 (2.0)	35.5 (2.1)
More seldom (160)	6.2 (0.6)	5.2 (1.1)	2.9 (0.8)	5.7 (0.9)	8.3 (1.1)	8.9 (1.2)
Dental attendance pattern (%) (2745)						
Proportion of those who visit dentists regularly (1678)	60.7 (1.2)	66.9 (2.3)	64.3 (2.3)	59.0 (2.1)	58.8 (2.5)	54.6 (2.3)
Never use or symptom-based use (1067)	39.3 (1.2)	33.1 (2.3)	35.7 (2.3)	41.0 (2.1)	41.2 (2.5)	45.4 (2.3)
Presence of plaque (%) (2832)						
No plaque (1062)	37.0 (1.3)	39.2 (2.4)	43.0 (2.1)	36.1 (2.1)	37.0 (2.3)	29.9 (2.0)
Plaque in gingival margins (1447)	51.2 (1.3)	51.1 (2.3)	46.8 (2.2)	53.1 (2.0)	50.2 (2.3)	55.0 (2.1)
Plaque also elsewhere (323)	11.8 (0.8)	9.7 (1.3)	10.2 (1.4)	10.8 (1.4)	12.8 (1.5)	15.2 (1.9)

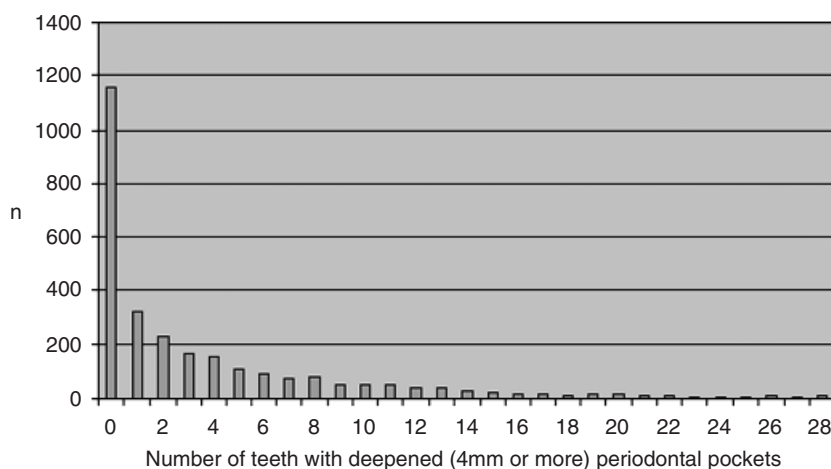


Fig. 1. Number of subjects with teeth with deepened (4 mm or more) periodontal pockets in the whole study population.

pockets (4 mm or deeper) more than those belonging to the lowest quintile of BMI. Among daily smokers, the mean number of teeth with periodontal pockets was more than twice as high as among never smokers. There was no consistent association between the teeth with deepened periodontal pockets and

BMI in the group of daily smokers (Table 3).

We found that the association of BMI with the number of teeth with deepened periodontal pockets was modified by smoking habits [statistically significant product term for both teeth with deepened periodontal pockets (4 mm or

more) ($p = 0.05$) and for deep periodontal pocket (6 mm or more) ($p = 0.02$)]. Also, the association of BMI with carious teeth was modified by smoking habits ($p = 0.09$). We did not observe any consistent modification by other factors than smoking habits in the association between BMI and with the number of teeth with deepened periodontal pockets.

We detected an exposure-response association between BMI and teeth with deepened periodontal pockets (4 mm or deeper) after controlling for smoking habits by restricting the analyses to a subgroup of never smokers and controlling for other potential confounders by including them as covariates in a multivariate model (Table 4). The association between BMI and teeth with deep periodontal pockets (6 mm or deeper) showed a fairly similar pattern, although there were some deviations from linearity. We did not observe a consistent association between BMI and the number of carious teeth among never smokers.

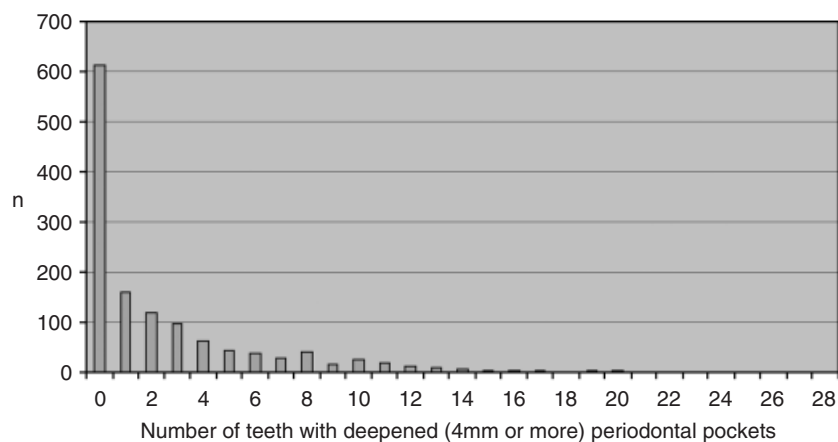


Fig. 2. Number of subjects with teeth with deepened (4 mm or more) periodontal pockets in the group of never smokers.

When excluding those who were periodontally healthy from the analysis, we found that the association among never smokers between BMI and the number of teeth with periodontal pockets (4 mm or more) was slightly attenuated. RR from the lowest category to the highest were 1.0 (reference category), 1.1 (95% CI 0.8–1.4), 1.1 (95% CI 0.9–1.3), 1.3 (95% CI 1.1–1.6) and 1.3 (95% CI 1.1–1.5) (highest category).

We also performed analyses among the whole study population where smoking was controlled for by including smoking habits as a covariate to allow comparisons between the results of this study and other studies. The results of these analyses showed that there was no consistent association between body weight and teeth with periodontal pockets of 4 mm or deeper. No consistent associations were observed in the case of teeth with deeper pockets (6 mm or more) or in the case of carious teeth in this population (Table 4).

Discussion

We detected a weak exposure–response association of BMI with the number of teeth with deepened periodontal pockets but not with the number of carious teeth when we controlled for the effect of smoking by restricting the sample to a subpopulation of never smokers and for the effect of other potential confounders by including them as covariates in the multivariate models. Our study does not address any mechanisms of how obesity may have an adverse effect on the periodontium. It has been suggested in earlier studies that impaired glucose tolerance, perturbations in lipid profiles

and substances secreted by adipose tissue, for example, could be mediating mechanisms (Saito & Shimazaki 2007). An alternative to a biological explanation is confounding, which could be related to biological or behavioural determinants in common.

Potential confounders and modifiers

The study population was restricted to subjects aged 30–49 years in order to eliminate the confounding effect of age-related factors. We also restricted the analyses to subjects who had never smoked. This was done in order to eliminate confounding and the possible modifying effect of smoking habits. By restricting the analyses to never smokers, we eliminated confounding not only related to the toxic effects of smoking but most likely also to the effects of various behavioural factors. In addition, we adjusted for a number of other potential confounders such as gender, education, number of teeth and behavioural factors related to dental health behaviour. The study population is also quite homogeneous in relation to ethnic origin.

One of the most important confounders in this context is smoking, which is considered to be a risk factor for periodontitis (Borrell & Papapanou 2005), and also for various other diseases, including cardiovascular diseases, different lung diseases and several cancers. Owing to its associations with a large number of behavioural factors (Thornton et al. 1994), smoking most probably confounds the association between body weight and periodontal infection. Confounding may arise from the measurement and/or reporting of smoking

habits, which are most likely incomplete and subject to errors (Scott et al. 2001, Hujoel et al. 2002, Spiekerman et al. 2003).

In this study, we found a consistent, although a weak association among never smokers whereas no consistent association was found among daily smokers or among the whole study population. The interesting question is whether smoking modifies the association between body weight and periodontal infection. However, our possibilities to study the possible modifying effect of smoking on the association between body weight and periodontal infection were limited. A large variation in the group of daily smokers, possibly due to residual confounding related to the quantity of smoked tobacco, prevents the detection of the effect of less powerful determinants and also the detection of any causal interdependence between body weight and smoking. However, the possibility that a modifying effect of smoking does exist, either synergistic or antagonistic, cannot totally be excluded.

The presence of dental plaque is an aetiological factor for both dental caries and periodontitis. In this study, the presence of dental plaque was the most important determinant for periodontal infection and dental caries. It is therefore not surprising that by including it in the multivariate models, the estimates attenuated considerably. In the case of teeth with periodontal pockets of 4 mm or deeper, for instance, the RR in the highest category of BMI increased from 1.7 to 1.5, even when dental attendance patterns and toothbrushing frequency were controlled for (data not shown). This stresses the importance of behavioural factors as confounders. It also suggests that studies in which behavioural factors are not accurately controlled for might overestimate the true risk.

The possibility that there might be other extraneous factors that could confound the association between body weight and periodontal infection in an exposure–response manner and could cause a spurious exposure–response association is not excluded. For example, poor diet and physical inactivity could be such factors, although they are not currently considered to be risk factors for periodontitis (Borrell & Papapanou 2005). Moreover, attitudinal or behavioural factors could also be such factors, because these factors are

Table 2. Factors related to teeth with deepened periodontal pockets and to carious teeth; crude relative risk (RR) with 95 % confidence intervals (CI)

	Teeth with periodontal pockets of 4 mm or more		Teeth with periodontal pockets of 6 mm or more		Carious teeth	
	RR	95% CI	RR	95% CI	RR	95% CI
Gender						
Male	1.7	1.5–1.9	2.5	1.8–3.5	2.4	1.9–2.9
Female	1.0		1.0		1.0	
	(n = 2811)		(n = 2811)		(n = 2841)	
Age						
30–34	1.0		1.0		1.0	
35–39	1.4	1.1–1.6	2.4	1.3–4.1	0.9	0.7–1.2
40–44	1.6	1.4–1.9	3.3	2.0–5.6	1.3	1.0–1.8
45–49	2.0	1.7–2.4	5.2	3.2–8.4	1.7	1.3–2.2
	(n = 2811)		(n = 2811)		(n = 2841)	
Education						
Low	2.2	1.9–2.6	4.4	2.8–6.8	4.4	3.3–5.8
Intermediate	1.6	1.5–1.8	3.2	2.1–4.7	2.7	2.1–3.5
High	1.0		1.0		1.0	
	(n = 2810)		(n = 2810)		(n = 2840)	
Number of teeth						
1–9	1.0		1.0		1.0	
10–19	2.7	1.7–4.2	2.9	1.3–6.2	1.8	1.1–2.8
20–24	2.9	1.9–4.5	1.3	0.6–2.9	1.1	0.7–1.5
25–32	2.5	1.7–3.6	1.1	0.6–2.3	0.5	0.4–0.7
	(n = 2811)		(n = 2811)		(n = 2841)	
Dental attendance pattern						
Never use or symptom-based use	1.7	1.5–1.9	2.3	1.7–3.0	5.2	4.2–6.4
Regular check-ups	1.0		1.0		1.0	
	(n = 2715)		(n = 2715)		(n = 2745)	
Tooth brushing						
At least twice a day	1.0		1.0		1.0	
Once a day	1.3	1.1–1.4	1.3	1.0–1.9	2.1	1.7–2.5
More seldom	2.2	1.8–2.6	2.6	1.6–4.2	6.2	4.5–8.6
	(n = 2715)		(n = 2715)		(n = 2745)	
Presence of plaque						
No plaque	1.0		1.0		1.0	
Plaque in gingival margins	2.0	1.7–2.3	2.4	1.7–3.4	2.1	1.7–2.6
Plaque also elsewhere	4.3	3.7–5.1	8.8	6.1–12.7	6.8	5.2–8.9
	(n = 2805)		(n = 2805)		(n = 2832)	
BMI						
I Quintile (lowest)	1.0		1.0		1.0	
II Quintile	1.1	0.9–1.3	1.0	0.6–1.6	0.7	0.5–1.0
III Quintile	1.1	0.9–1.3	0.9	0.5–1.5	0.9	0.7–1.2
IV Quintile	1.3	1.1–1.6	1.3	0.8–2.2	1.1	0.8–1.5
V Quintile (highest)	1.5	1.3–1.8	1.7	1.0–2.7	1.3	1.0–1.8
	(n = 2810)		(n = 2810)		(n = 2840)	

Number of teeth treated as an offset variable.

BMI, body mass index.

difficult if not impossible to conceptualize and consequently to measure.

Validity issues

The way in which periodontitis is defined and assessed in epidemiological studies varies considerably. In the present study, pocket depth was measured in four sites in each tooth and the most severe site was recorded. As a main outcome, we used the number of teeth

with deepened periodontal pockets with a cut-off value of 4 mm, which is commonly used as a limit for deepened pockets. In addition, we used the number of teeth with pocket depths of 6 mm or more as a secondary outcome, but because the number of teeth with deeper pockets (6 mm or more) was low, the results are subject to large random variation. Also, the estimates, especially those for carious teeth and for teeth with deep periodontal pockets (6 mm

or more), are subject to errors due to an excessive number of zeros.

To overcome the problems related to distribution assumption, we performed an additional analysis (among never smokers) where we excluded periodontally healthy subjects from the analysis. This complementary analysis also showed a positive association between BMI and the number of teeth with deepened (4 mm or more) periodontal pockets, although the association was slightly attenuated. This finding could be interpreted in that body weight has an effect on the extent of periodontal infection among subjects with periodontal infection. However, the most important aspect is that this finding increases the credibility of the results obtained in the main analyses.

Despite these shortcomings, there are several benefits of using the number of teeth with periodontal pockets as an outcome variable. One is that using the number of teeth with deepened periodontal pockets as an outcome variable, the study also focuses on the extent of the infection of the periodontium at the time of the survey. Using other criteria, such as clinical attachment loss, alveolar bone loss or combinations of different parameters, means one does not focus on the prevailing infection but on other aspects, such as consequences of past disease processes. This is an important point because the use of aetiological history may cause validity problems (Steurer et al. 2006).

Another advantage in using the number of teeth with deepened periodontal pockets as an outcome is that we were able to avoid an arbitrary cut-off point. Therefore, the use of a continuous variable reflects the true pattern of periodontal infection better than classifying subjects into two categories based on an arbitrary cut-off point. By doing so, we were also able to reduce the effect of misclassification by reducing the effect of measurement error, and consequently reduce attenuation of the results.

The operationalization of body weight may involve misclassification due to individual variation in the distribution of adipose tissue and muscle mass. The fact that BMI, i.e. body weight in relation to height, is not an optimal base for determining overweight and obesity has been demonstrated earlier (Rimm et al. 1995). Despite this shortcoming, it is probably the most commonly used measure of overweight and obesity. In these data,

Table 3. Adjusted mean number of teeth with periodontal pockets of 4 mm or more among subjects who had never smoked ($n = 1265$) and among subjects who smoke daily ($n = 800$); standard error in parentheses

	Among never smokers		Among daily smokers	
	number of subjects	mean number of teeth with periodontal pockets	number of subjects	mean number of teeth with periodontal pockets
BMI				
I Quintile (lowest)	278	2.1 (0.2)	181	5.8 (0.6)
II Quintile	294	2.3 (0.3)	149	6.4 (0.6)
III Quintile	267	2.5 (0.2)	181	5.1 (0.5)
IV Quintile	248	2.9 (0.3)	164	5.4 (0.5)
V Quintile (highest)	238	3.1 (0.3)	158	6.6 (0.5)

Adjusted for gender, age as continuous, education, dental attendance pattern, tooth brushing frequency and presence of plaque.

Fifty subjects from the group of never smokers and 33 subjects from the group of daily smokers were excluded from the analyses due to missing data.

BMI, body mass index.

Table 4. Relation of body mass index to teeth with periodontal pockets and to carious teeth; adjusted relative risk (adj. RR) with 95 % confidence intervals (CI)

	Teeth with periodontal pockets of 4 mm or more		Teeth with periodontal pockets of 6 mm or more		Carious teeth	
	adj. RR	95% CI	adj. RR	95% CI	adj. RR	95% CI
<i>Among never smokers*</i>						
BMI						
I Quintile (Lowest)	1.0		1.0		1.0	
II Quintile	1.1	0.8–1.4	0.7	0.3–1.8	0.8	0.5–1.5
III Quintile	1.2	0.9–1.5	0.8	0.3–2.2	1.1	0.7–1.6
IV Quintile	1.3	1.0–1.7	1.3	0.5–3.2	0.9	0.6–1.5
V Quintile (highest)	1.5	1.2–1.9	2.0	0.9–4.6	1.2	0.7–1.9
BMI (as a continuous variable)	1.03	1.02–1.05	1.08	1.03–1.14	1.00	0.97–1.03
<i>Total population†</i>						
BMI						
I Quintile (lowest)	1.0		1.0		1.0	
II Quintile	1.1	0.9–1.3	0.9	0.6–1.4	0.7	0.5–1.0
III Quintile	1.0	0.8–1.1	0.6	0.4–1.0	0.8	0.6–1.0
IV Quintile	1.0	0.9–1.2	0.8	0.5–1.3	0.8	0.6–1.1
V Quintile (highest)	1.2	1.0–1.4	1.0	0.7–1.6	0.9	0.7–1.2
BMI (as a continuous variable)	1.01	1.00–1.02	1.01	0.96–1.05	1.00	0.98–1.02

*Adjusted for gender, age as continuous, education, dental attendance pattern, tooth brushing frequency, presence of plaque and number of teeth as the offset variable.

†Adjusted for gender, age as continuous, education, dental attendance pattern, tooth brushing frequency, presence of plaque, smoking history and number of teeth as the offset variable.

Fifty subjects from the group of never smokers and 134 subjects from the total study population were excluded from the analyses due to missing data.

BMI, body mass index.

the correlation between BMI and body fat was high, being 0.90, indicating that BMI is a fairly good, although possibly conservative measure of adiposity. Misclassification related to individual variation in the distribution of adipose tissue most likely causes bias towards null and attenuates the association. This indicates that the true effect of body weight on periodontal infection might be more distinct than that observed.

Limitations of the study

A self-evident limitation in this study is the use of cross-sectional data, which makes it impossible to determine the direction of causal relations. The direction of the relation might also be opposite to the one expected or it might be bidirectional. Our complementary analyses (data not shown) revealed that the number of teeth with periodontal pock-

ets was positively associated with BMI in this study population (never smokers, aged 30–49) after controlling for potential confounders, suggesting that periodontal infection could be a risk factor for obesity. This complementary analysis showed that the direction of the relation cannot be solved in this kind of cross-sectional study, and that in such cases the interpretation of the results is a matter of judgement.

Another limitation that has been connected to cross-sectional studies is that prevalence is dependent on both aetiological and prognostic factors. However, this effect is most likely limited in the case of chronic, non-fatal conditions.

A further limitation is that the effects of other diseases could not be eliminated fully. One of the most important ones is diabetes, which often coexists with obesity and periodontitis (Nishimura et al. 2003). The relation between diabetes and obesity might differ depending on the type of diabetes and how well each diabetic adheres to treatment recommendations, for example, which makes it difficult to control for. Because the number of subjects with diagnosed diabetes was low, we excluded them from the study. It is possible, however, that undiagnosed diabetic patients existed in the study population. The proportion of such subjects is most likely quite small, because the prevalence of diabetes is quite low in this age group (30–49) in comparison with older age groups, which suggests that the effect of undiagnosed diabetes is small.

Implications and conclusions

Our study does not suggest any specific biological mechanism of how obesity may have an adverse effect on the periodontium. Yet there are facts, namely specificity and exposure–response association, that support, but do not prove, the presence of a biological mechanism between body weight and periodontal infection. The adverse effects of obesity could be mediated through several mechanisms including impaired glucose tolerance, perturbations in lipid profiles and secretion of substances from adipose tissue (Saito & Shimazaki 2007) or through some other mechanism. The possibility that these mechanisms might act simultaneously is not excluded either. Despite the existence of possible mediating mechanisms,

until biological mechanisms connecting body weight to periodontal infection have been found and confirmed, confounding as an alternative to a biological explanation has to be considered.

The results of this study indicate that overweight and obesity are potential confounders in the association between periodontitis and other diseases. For example, obesity is a risk factor for atherosclerotic cardiovascular diseases (Eckel 1997, Pearson et al. 2003) and, due to its associations with periodontitis, it is a potential confounder in the association between periodontitis and atherosclerotic cardiovascular diseases. Because adipose tissue secretes pro-inflammatory cytokines such as interleukins (IL-1 β , IL-6) and tumour necrosis factor (Baumann & Gaudie 1994), it means that obesity could also be a potential confounder in the association between periodontitis and the above-mentioned pro-inflammatory mediators. Moreover, because IL-6 up-regulates C-reactive protein (CRP) (Visser et al. 1999), obesity is a potential confounder in the association of periodontal diseases with CRP too, an association that has recently been studied intensively.

The results of our study conform well with the overall picture of results obtained in earlier studies: a weak overall association and more consistent association, although by no means is a strong association observed when the effects of potential confounders, especially smoking habits and age, are thoroughly controlled for. When interpreting the results, it must be remembered, however, that obesity and other risk factors for periodontitis alone are rarely sufficient causes for periodontitis. The onset of the disease most likely requires the coaction of risk factors, which probably vary depending on the disease mechanisms and the presence of other risk factors. Both cause variation in the results, and might be a plausible explanation for differences in the results of different epidemiological studies.

We found that among the non-diabetic, non-smoking population aged 30–49 body weight was associated with the number of teeth with deepened periodontal pockets in an exposure-dependent manner. Despite apparent unconfounded results, it is possible that this association is not necessarily

causal. Hence, additional research is needed to determine the nature of this association.

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Clinical Relevance

Scientific rationale for the study: There are several possible biological mechanisms connecting obesity to increased risk of periodontitis. To date, the results of earlier epidemiolo-

gical studies connecting body weight to periodontitis are inconclusive.

Principal findings: Body mass index was weakly associated with the number of teeth with periodontal pockets

among a non-diabetic non-smoking population aged 30–49.

Practical implications: The nature of this association is not known and more evidence is needed.