J Periodont Res 2008; 43: 417–421

JOURNAL OF PERIODONTAL RESEARCH
doi:10.1111/j.1600-0765.2007.01063.x

Relationship between body mass index and periodontitis in young Japanese adults

D. Ekuni[1,2], T. Yamamoto[1], R. Koyama[1], M. Tsuneishi[1], K. Naito[2], K. Tobe[2]

[1]Department of Oral Health, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan and [2]Health and Environment Center, Okayama University, Okayama, Japan

Ekuni D, Yamamoto T, Koyama R, Tsuneishi M, Naito K, Tobe K. Relationship between body mass index and periodontitis in young Japanese adults. J Periodont Res 2008; 43: 417–421. © 2008 The Authors. Journal compilation © 2008 Blackwell Munksgaard

Background and Objective: Obesity has been implicated as a risk factor for several chronic health conditions. Recent studies have reported a relationship between obesity and periodontitis, but few studies have investigated this relationship in adolescents. The purpose of the present study was to investigate the relationship between body composition (i.e. body mass index and body fat) and periodontitis in university students in Japan.

Material and methods: Medical and oral health data were collected in a cross-sectional examination conducted by the Health and Environment Center of Okayama University. Students aged 18–24 years ($n = 618$), who were interested in receiving an oral health examination, were included in the analysis. The community periodontal index was used to assess periodontal status. Subjects with a community periodontal index score of 0–2 were considered as controls and those with a community periodontal index score of > 2 were considered to have periodontitis. Logistic regression analysis was used to estimate the association between body mass index and periodontitis.

Results: The body mass index of all subjects was < 30 kg/m^2. Age and body mass index were significantly associated with the community periodontal index. Logistic regression analysis revealed a 16% increased risk for periodontitis per 1-kg/m^2 increase in body mass index (adjusted odds ratio, 1.16; 95% confidence interval, 1.03–1.31; $p < 0.05$).

Conclusion: Body mass index could be a potential risk factor for periodontitis among healthy young individuals (i.e. those with a body mass index of < 30 kg/m^2). It may be useful to include an evaluation of body mass index on a regular basis in university general and oral health examinations.

Tatsuo Yamamoto, Department of Oral Health, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, 2-5-1 Shikata-cho, Okayama 700-8525, Japan
Tel: +81 86 235 6711
Fax: +81 86 235 6714
e-mail: tatsuo@md.okayama-u.ac.jp

Key words: periodontitis; body mass index; risk factor; young adults

Accepted for publication October 31, 2007

Obesity is a major risk factor for chronic conditions, including type 2 diabetes, hyperlipemia, cardiovascular disease, hypertension and cholelithiasis (1–4). The proportion of obese individuals is showing a gradual increase in both developed and developing countries, and this is a well-recognized public health concern worldwide (3,5,6). In Japan, reports using data from the National Nutrition Surveys of Japan have shown evidence of increasing numbers of overweight or obese adults (7–9).

Epidemiological studies have shown that obesity, as determined by body mass index, waist-to-hip circumference ratio, body fat, or maximum oxygen

consumption, is significantly associated with periodontitis (10–18). Obesity has been found to contribute significantly to periodontitis severity in experimental animals (19). These findings suggest that periodontal disease may be exacerbated by conditions associated with obesity and that obesity may be a risk factor for periodontitis (14,20).

Periodontitis severity increases with age (21,22), and in younger populations it is important to prevent periodontitis by controlling risk factors. To our knowledge, few studies have investigated body composition as a risk factor for periodontitis in adolescents. The purpose of the present study was to investigate whether or not there is an association between body composition, as determined by body mass index measurement and body fat assessment, and periodontitis in healthy Japanese university students.

Material and methods

Data source and study samples

The data analyzed were obtained from general and oral examinations performed between 2003 and 2007 at the Health and Medical Center of Okayama University. Of 38,107 students aged 18–24 years (consultation rate: 74%) who had undergone a general health examination, 618 (296 men and 322 women) also volunteered to receive an oral examination.

Assessment of obesity

Body mass index and body fat were assessed as indicators of overall adiposity. Body mass index was computed as weight in kilograms divided by square height in meters. Body fat was measured using the bio-impedance method and a Body Fat Analyzer (TBF-202; Tanita Co., Tokyo, Japan).

Oral examination

Two experienced dentists performed the oral examinations. The total number of teeth and of decayed, missing and filled teeth was determined. To assess periodontal condition, the World Health Organization community periodontal index was used. Groups were defined according to periodontal status; individuals in the control group had a community periodontal index of 0–2, whereas those in the periodontitis group had a community periodontal index of 3–4 (23).

Statistical analysis

Because the data were skewed, the Mann–Whitney *U* and chi-squared tests were used to determine if there were any significant differences ($p < 0.05$) between groups. Any possible association of body mass index with periodontitis was examined in a series of logistic regression models, and the odds ratio and 95% confidence interval were calculated. The logistic regression models were reviewed for goodness-of-fit and validated using the Hosmer–Lemeshow statistic (12). The presence of periodontitis was used as the dependent variable, and age and gender were added as the independent variables in the multivariate analysis (21). The Statistical Package for the Social Sciences (15.0 J for Windows; SPSS Japan, Tokyo, Japan) was used for the statistical analyses.

Table 1. Characteristics of subjects (Japanese university students, $n = 618$)

Characteristics	No. of subjects (%)
Periodontal status	
CPI 0	118 (19)
CPI 1	59 (10)
CPI 2	392 (63)
CPI 3	49 (8)
CPI 4	0 (0)
Body mass index (kg/m^2)	
< 18.5	69 (11)
18.5–24.9	517 (84)
25.0–29.9	32 (5)
≥ 30.0	0 (0)

CPI, community periodontal index.

Results

Mean subject characteristics were as follows: age, 21.6 years (standard deviation 1.6); decayed, missing and filled teeth, 4.6 (standard deviation 4.2); number of teeth present, 27.8 (standard deviation 0.9); body mass index, 21.0 kg/m^2 (standard deviation 2.3); and body fat, 21.3% (standard deviation 5.8). Body mass index and community periodontal index scores are shown in Table 1. Subjects with a body mass index of ≥ 30 kg/m^2 and a community periodontal index of 4 were not identified in this study.

Characteristics of the study population and the prevalence of periodontitis are shown in Table 2. Of 618 subjects examined, 49 were classified as having periodontitis. There were significant differences in age and body mass index between the control (community periodontal index 0–2) and the periodontitis (community periodontal index 3) groups. No significant differences in gender, decayed, missing and filled teeth, number of teeth present and body fat were observed between the two groups.

In logistic regression analyses, subjects with periodontitis had an increased risk of periodontitis of 16% for each 1-kg/m^2 body mass index increase (adjusted odds ratio, 1.16; 95% confidence interval, 1.03–1.31; Table 3).

Table 2. Characteristics of subjects by periodontal status

	Control CPI 0–2 ($n = 569$)	Periodontitis CPI 3 ($n = 49$)	p
Age (years)	21.1 ± 1.6*	21.8 ± 1.4*	0.002†
DMFT	4.7 ± 4.2*	4.0 ± 3.8*	0.25†
Number of teeth present	27.8 ± 0.9*	27.8 ± 0.9*	0.32†
Body mass index (kg/m^2)	20.9 ± 2.6*	21.8 ± 2.4*	0.009†
Body fat (%)	21.3 ± 5.7*	21.3 ± 6.1*	0.99†
Proportion of male students (%)	47	53	0.45‡

*Data are given as mean ± SD.
†Mann–Whitney *U*-test.
‡Chi-squared test.
CPI, community periodontal index; DMFT, decayed, missing and filled teeth.

Table 3. Crude and multivariate odds ratios and 95% confidence interval (CI) of body mass index (BMI), age and gender in the study population

Variables	Crude odds ratio (95% CI)	*p*	Multivariate odds ratio (95% CI)	*p*
BMI	1.17 (1.04–1.31)	0.010	1.16 (1.03–1.31)	0.018
Age	1.36 (1.12–1.65)	0.002	1.34 (1.10–1.63)	0.004
Gender (male)	0.80 (0.45–1.43)	0.45	1.07 (0.58–1.98)	0.82

Discussion

It has been reported that the prevalence of periodontal disease is 76% higher among young obese (body mass index ≥ 30 kg/m^2) individuals aged 18–34 years than in normal-weight individuals (13) and that weight is associated with increased risk of periodontitis among those aged 17–21 years (24). In the present study of Japanese university students aged 18–24 years, a higher body mass index was associated with increased risk of periodontitis. An increase in body mass index may therefore be a potential risk factor for periodontitis, even among young individuals (i.e. in those with a body mass index of < 30 kg/m^2).

Body mass index has been widely used to assess general body composition. It increases with age (25–27), and children aged 7–15 years with normal body mass index may become overweight or obese adults (body mass index > 25) (28). In young subjects with a normal body mass index, prospective morbidity is dependent on body mass index as well as on age (27), and maintenance of body mass index at the level at which it is present during adolescence is an important factor in preventing future disease. The World Health Organization recommends that national public health programs should incorporate oral health promotion and disease prevention based on the common risk factors approach (29,30). The evaluation of body mass index on a regular basis should be included at health and oral examinations in universities.

While body mass index showed an association with periodontitis severity in the present study, body fat (foot-to-foot bio-impedance) did not. However, in another study, of 40–79-year-old Japanese women, body fat was significantly associated with deep periodontal pockets (> 1.9 mm) (17). This discrepancy between study findings may be because of differences in the age of subjects or in the cut-off point for probing pocket depth. Moreover, body mass index is more predictive of a diagnosis of metabolic syndrome than bio-impedance (31), and the bio-impedance method is not an accurate measurement compared with dual-energy X-ray absorptiometry (31,32).

A variety of potential mechanisms could explain an association between obesity and periodontitis. Overweight young subjects may have unhealthy dietary patterns with insufficient micronutrients and excess sugar and fat content, and such dietary patterns may increase the risk for periodontal disease (13). Changes in host immunity and/or increased stress levels, which are often associated with gain of excess fat early in life, may also play a role (24). The underlying biological mechanisms for the association of obesity with periodontitis are not well established. However, adipose-tissue-derived cytokines and hormones may play a role (33). Fat tissue is not only a passive triglyceride reservoir, but also produces high levels of cytokines and hormones, collectively called adipokines or adipocytokines (34), which may in turn affect the periodontal tissues. Pro-inflammatory cytokines, such as tumor necrosis factor-α and interleukin-6, may form a multidirectional link among periodontitis, obesity and other chronic diseases (35–38). Several studies have demonstrated a close involvement of adipokines (such as leptin, adiponectin and resistin) in inflammatory processes (39–43). Obesity may also influence periodontal disease status by increasing lipid and glucose blood levels (44–48), which may in turn have deleterious consequences for the host response by altering T cells and monocyte/macrophage function, as well as increasing cytokine production (46,49–51).

This study had some limitations. As the design of this study limited the interpretation of causal relationships, prospective cohort studies may provide information beyond what we present here. The roles of genetics, oral health behaviors, or other factors (e.g. dental plaque, calculus, smoking, nutrition, stress levels, alcohol consumption, etc.), which have been shown to affect the prevalence of periodontitis, were not considered in our study. However, in a previous study, the proportion of university students who were found to be smokers was relatively small (male students, 10.8%; female students, 2.2%) (52). In addition, the young age of the subjects in the present study meant that the length of time that they had been smokers and number of cigarettes were relatively small. Therefore, in the group of subjects in the present study, the effects of smoking on periodontal conditions may not be as high as found in middle-aged and elderly individuals. As the prevalence of cigarette smoking is also inversely associated with body mass index (53,54), smoking may not act as a confounding factor in this study. Finally, the number of subjects in the study was small and was limited by the available general and oral health data. While the prevalence of periodontitis was only 8% ($n = 49$), this value is close to that reported in the World Health Organization Global Oral Health Data Bank for 15–19-year-old individuals (around 10%) (30). Additional studies are needed to clarify the causality between abdominal adiposity and periodontitis.

In conclusion, a higher body mass index could be a potential risk factor for periodontitis among a healthy young population (body mass index < 30 kg/m^2). Thus, it may be useful for body mass index evaluations to be included on a regular basis during general and oral health examinations at universities.

References

1. Kopelman PG. Obesity as a medical problem. *Nature* 2000;**404:**635–643.

2. Doll S, Paccaud F, Bovet P, Burnier M, Wietlisbach V. Body mass index, abdominal adiposity and blood pressure: consistency of their association across developing and developed countries. *Int J Obes Relat Metab Disord* 2002;**26:**48–57.
3. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA* 1999;**282:**1523–1529.
4. Field AE, Coakley EH, Must A *et al*. Impact of overweight on the risk of developing common chronic diseases during a 10-year period. *Arch Intern Med* 2001;**161:**1581–1586.
5. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults. The National Health and Nutrition Examination Surveys, 1960–1991. *JAMA* 1994;**272:** 205–211.
6. Saw SM, Rajan U. The epidemiology of obesity: a review. *Ann Acad Med Singapore* 1997;**26:**489–493.
7. Horibe H. Trend of average height, weight and skinfolds in the Japanese. *Diabetes Res Clin Pract* 1990;**10:**S113–S118.
8. Sakata K, Labarthe DR. Changes in cardiovascular disease risk factors in three Japanese national surveys 1971–1990. *J Epidemiol* 1996;**6:**93–107.
9. Yoshiike N, Seino F, Tajima S *et al*. Twenty-year changes in the prevalence of overweight in Japanese adults: The National Nutrition Survey 1976–95. *Obes Rev* 2002;**3:**183–190.
10. Saito T, Shimazaki Y, Sakamoto M. Obesity and periodontitis. *N Engl J Med* 1998;**339:**482–483.
11. Saito T, Shimazaki Y, Yamashita Y, Koga T, Tsuzuki M, Sakamoto M. Association between periodontitis and exercise capacity. *Periodontal Insights* 1999;**62:** 9–12.
12. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between upper body obesity and periodontitis. *J Dent Res* 2001;**80:**1631–1636.
13. Al-Zahrani MS, Bissada NF, Borawskit EA. Obesity and periodontal disease in young, middle-aged, and older adults. *J Periodontol* 2003;**74:**610–615.
14. Wood N, Johnson RB, Streckfus CF. Comparison of body composition and periodontal disease using nutritional assessment techniques: Third National Health and Nutrition Examination Survey (NHANES III). *J Clin Periodontol* 2003; **30:**321–327.
15. Dalla Vecchia CF, Susin C, Rosing CK, Oppermann RV, Albandar JM. Overweight and obesity as risk indicators for periodontitis in adults. *J Periodontol* 2005;**76:**1721–1728.
16. Nishida N, Tanaka M, Hayashi N *et al*. Determination of smoking and obesity as periodontitis risks using the classification and regression tree method. *J Periodontol* 2005;**76:**923–928.
17. Saito T, Shimazaki Y, Kiyohara Y *et al*. Relationship between obesity, glucose tolerance, and periodontal disease in Japanese women: the Hisayama study. *J Periodontal Res* 2005;**40:**346–353.
18. Linden G, Patterson C, Evans A, Kee F. Obesity and periodontitis in 60–70-year-old men. *J Clin Periodontol* 2007;**34:**461–466.
19. Perlstein MI, Bissada NF. Influence of obesity and hypertension on the severity of periodontitis in rats. *Oral Surg Oral Med Oral Pathol* 1977;**43:**707–719.
20. Vanhala MJ, Kumpusalo EA, Pitkaijarvi TK, Takala JK. 'Metabolic syndrome' in a middle-aged Finnish population. *J Cardiovasc Risk* 1997;**4:**291–295.
21. Genco RJ. Current view of risk factors for periodontal diseases. *J Periodontol* 1996;**67:**1041–1049.
22. Albandar JM, Tinoco EM. Global epidemiology of periodontal diseases in children and young persons. *Periodontol 2000* 2002;**29:**153–176.
23. Ojima M, Hanioka T, Tanaka K, Inoshita E, Aoyama H. Relationship between smoking status and periodontal conditions: findings from national databases in Japan. *J Periodontal Res* 2006;**41:**573–579.
24. Reeves AF, Rees JM, Schiff M, Hujoel P. Total body weight and waist circumference associated with chronic periodontitis among adolescents in the United States. *Arch Pediatr Adolesc Med* 2006;**160:**894–899.
25. Hediger ML, Hartnett HJ, Louis GM. Association of endometriosis with body size and figure. *Fertil Steril* 2005;**84:**1366–1374.
26. Hypponen E, Power C, Smith GD. Prenatal growth, BMI, and risk of type 2 diabetes by early midlife. *Diabetes Care* 2003;**26:**2512–2517.
27. Tobe K, Ogura T, Tsukamoto C, Inoue H, Arata J, Matsuura K. Effect of change in body mass index on morbidity in non-obese university graduates. *Acta Med Okayama* 2002;**56:**149–158.
28. Venn AJ, Thomson RJ, Schmidt MD *et al*. Overweight and obesity from childhood to adulthood: a follow-up of participants in the 1985 Australian Schools Health and Fitness Survey. *Med J Aust* 2007;**186:**458–460.
29. Petersen PE, Yamamoto T. Improving the oral health of older people: the approach of the WHO Global Oral Health Programme. *Community Dent Oral Epidemiol* 2005;**33:**81–92.
30. Petersen PE, Ogawa H. Strengthening the prevention of periodontal disease: the WHO approach. *J Periodontol* 2005; **76:**2187–2193.
31. Soto Gonzalez A, Bellido D, Buno MM *et al*. Predictors of the metabolic syndrome and correlation with computed axial tomography. *Nutrition* 2007;**23:**36–45.
32. Steiner MC, Barton RL, Singh SJ, Morgan MD. Bedside methods versus dual energy X-ray absorptiometry for body composition measurement in COPD. *Eur Respir J* 2002;**19:**626–631.
33. Pischon N, Heng N, Bernimoulin JP, Kleber BM, Willich SN, Pischon T. Obesity, inflammation, and periodontal disease. *J Dent Res* 2007;**86:**400–409.
34. Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. *J Clin Endocrinol Metab* 2004;**89:**2548–2556.
35. Hotamisligil GS. The role of TNF alpha and TNF receptors in obesity and insulin resistance. *J Intern Med* 1999;**245:**621–625.
36. Hotamisligil GS. Molecular mechanisms of insulin resistance and the role of the adipocyte. *Int J Obes Relat Metab Disord* 2000;**24**(suppl 4)**:**S23–S27.
37. Kern PA, Ranganathan S, Li C, Wood L, Ranganathan G. Adipose tissue tumor necrosis factor and interleukin-6 expression in human obesity and insulin resistance. *Am J Physiol Endocrinol Metab* 2001;**280:**E745–E751.
38. Rotter V, Nagaev I, Smith U. Interleukin-6 (IL-6) induces insulin resistance in 3T3-L1 adipocytes and is, like IL-8 and tumor necrosis factor-alpha, overexpressed in human fat cells from insulin-resistant subjects. *J Biol Chem* 2003;**278:**45777–45784.
39. Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. Positional cloning of the mouse obese gene and its human homologue. *Nature* 1994;**372:**425–432.
40. Berg AH, Combs TP, Scherer PE. ACRP30/adiponectin: an adipokine regulating glucose and lipid metabolism. *Trends Endocrinol Metab* 2002;**13:**84–89.
41. Chandran M, Phillips SA, Ciaraldi T, Henry RR. Adiponectin: more than just another fat cell hormone? *Diabetes Care* 2003;**26:**2442–2450.
42. Verma S, Li SH, Wang CH *et al*. Resistin promotes endothelial cell activation: further evidence of adipokine-endothelial interaction. *Circulation* 2003;**108:**736–740.
43. Kawanami D, Maemura K, Takeda N *et al*. Direct reciprocal effects of resistin and adiponectin on vascular endothelial cells: a new insight into adipocytokine-endothelial cell interactions. *Biochem Biophys Res Commun* 2004;**314:**415–419.
44. Denke MA, Sempos CT, Grundy SM. Excess body weight. An under-recognized contributor to dyslipidemia in white American women. *Arch Intern Med* 1994;**154:**401–410.
45. Galanis DJ, McGarvey ST, Sobal J, Bausserman L, Levinson PD. Relations of

body fat and fat distribution to the serum lipid, apolipoprotein and insulin concentrations of Samoan men and women. *Int J Obes Relat Metab Disord* 1995;**19:**731–738.
46. Tanaka S, Isoda F, Ishihara Y, Kimura M, Yamakawa T. T lymphopaenia in relation to body mass index and TNF-alpha in human obesity: adequate weight reduction can be corrective. *Clin Endocrinol (Oxf)* 2001;**54:**347–354.
47. Hsieh SD, Yoshinaga H, Muto T, Sakurai Y, Kosaka K. Health risks among Japanese men with moderate body mass index. *Int J Obes Relat Metab Disord* 2000;**24:**358–362.
48. Dixon JB, O'Brien P. A disparity between conventional lipid and insulin resistance markers at body mass index levels greater than 34 kg/m^2. *Int J Obes Relat Metab Disord* 2001;**25:**793–797.
49. Samartin S, Chandra RK. Obesity, overnutrition and immune system. *Nutr Res* 2001;**21:**243–262.
50. Marti A, Marcos A, Martinez JA. Obesity and immune function relationships. *Obes Rev* 2001;**2:**131–140.
51. Lamas O, Marti A, Martinez JA. Obesity and immunocompetence. *Eur J Clin Nutr* 2002;**56**(suppl 3)**:**S42–S45.
52. Kinumi K, Naitou K, Kuroki K *et al*. Non-smoking campaign in Okayama University: A trial of the "Non-smoking school". *Campus Health (in Japanese)* 2004;**41:**63–68.
53. Hayashi R, Iwasaki M, Otani T *et al*. Body mass index and mortality in a middle-aged Japanese cohort. *J Epidemiol* 2005;**15:**70–77.
54. Cui R, Iso H, Toyoshima H *et al*. Body mass index and mortality from cardiovascular disease among Japanese men and women: the JACC study. *Stroke* 2005;**36:** 1377–1382.