The Relationship Between Smoking History, Periodontal Screening and Recording™ (PSR™) Codes and Overweight/Obesity in a Mississippi Dental School Population

Nelson Wood[a]/Roger B. Johnson[a]

Purpose: To examine the risks for being overweight (Ow) or obese (Ob) in subjects with elevated Periodontal Screening and Recording™ (PSR™) Codes (≥ 1) (with smoking as a modifier) in a population of dental school patients located within a region with a high incidence of Ow, Ob, diabetes mellitus (Db), and cardiovascular disease (CVD).

Materials and Methods: 1098 patient files were studied and data were grouped by average PSR™ Codes, smoking history, and body mass index (BMI). Data were compared by factorial ANOVA, the Mann-Whitney U test, and chi-square analysis using PSR™ Code as an exposure for BMI, Db, or CVD, with smoking as the effect modifier. Data were then analysed by adjusted multivariate logistic regression to determine the risks for being Ow or Ob.

Results: Subjects with an average PSR™ Code ≥ 1 and a smoking history did not have a significantly increased risk for either Db or CVD. Non-smoking subjects with an average PSR™ Code = 3 had a significantly higher risk for being Ow; those with an average PSR™ Code of 2–4 had a significantly higher risk for being Ob ($p < 0.05$). In contrast, only smokers with an average PSR™ Code = 4 had a significant risk for being either Ow or Ob.

Conclusion: Our data suggest that smoking may not directly increase the risk factors for Db or CVD in subjects with an average PSR™ Code > 1 (confirming the 'Smoker's Paradox'), but could be an indirect risk factor for these diseases as a consequence of its effect on body weight.

Key words: cardiovascular disease, diabetes mellitus, obesity, overweight, PSR™ Code, smoking

Oral Health Prev Dent 2008; 6: 67-74. *Submitted for publication: 25.08.06; accepted for publication: 26.01.07.*

Periodontitis is a chronic inflammation affecting the supporting tissues of the teeth and is present in 75% of the adults in the United States (Genco et al, 2002). There is increased evidence for common aetiologies of several systemic diseases and periodontitis (Wu et al, 2000; Fowler et al, 2001; Taylor, 2001), including cardiovascular disease (CVD) (Mattila et al, 1989; DeStefano et al, 1993; Beck et al, 1996, 1998; Joshipura et al, 1996; Genco, 1998; Arbes et al, 1999; Loos et al, 2000), diabetes mellitus (Db) (Papapanou, 1996; Mealey, 1999; Katz, 2001; Pradhan et al, 2001; Matthews, 2002) and being overweight (Ow) or obese (Ob) (Perlstein and Bissada, 1977; Saito et al, 1998, 2001; Grossi and Ho, 2000; Wood et al, 2003).

There is substantial information concerning the negative effects of systemic diseases on the periodontium; there is less information about the negative effects of a diseased periodontium on systemic health. The diseased periodontium is a reservoir of bacteria, bacterial products and inflammatory mediators, which can interact with organ systems outside the oral cavi-

[a] *Department of Periodontics and Preventive Sciences, University of Mississippi Medical Center, Mississippi, USA*

Correspondence: *Dr Roger B. Johnson, 2500 North State Street, Jackson, Mississippi 39216-4505. Tel: 601-984-6115. Fax: 601-984-6120. E-mail: rjohnson@sod.umsmed.edu*

ty. Periodontopathic bacteria produce lipopolysaccharides (LPS), which initiate a synthetic cascade of pro-inflammatory cytokines, which have both local and systemic effects. These effects include activation of monocytes/macrophages, increasing the number of neutrophils and the concentrations of fibrinogen and other coagulation factors in blood, alterations in lipid metabolism and enhancement of pro-inflammatory cytokine concentrations within the serum and gingival crevicular fluid (such as TNF-alpha) (Bostrom et al, 1999; de Maat and Kluft, 2002). The concentration of serum acute phase proteins such as C-reactive protein (CRP) and interleukin-6 (IL-6) (Castell et al, 1989; Ebersole et al, 1997; Loos et al, 2000; Slade et al, 2000; Wu et al, 2000; de Maat and Kluft, 2002) are also elevated. Thus, periodontal inflammation could contribute to the pathogenesis of other systemic diseases by serving as a source of either infective bacteria or proinflammatory cytokines. Once established, these sites of focal inflammation could induce feedback, which would amplify these immune and inflammatory responses into a hyperinflammatory state (Donahue and Wu, 2001).

There is evidence of a greater prevalence of periodontal disease in Db than in non-Db subjects (Cianciola et al, 1982; Nelson et al, 1990; Emrich et al, 1991; Safkan-Seppala and Ainamo, 1992; Ebersole et al, 1997). Db is frequently associated with a group of complications including CVD and periodontal disease (Papapanou, 1996). To emphasise the relationship between the diseases, periodontitis is considered to be the sixth significant complication of Db (Löe, 1993).

Use of tobacco products is an important risk factor for periodontitis, as cigarette smokers are 2.5–6 times more likely to develop periodontal disease than non-smokers (Bergstrom and Preber, 1994). Smoking elevates the quantity of periodontopathic bacteria, and modulates the host inflammatory and immune responses to bacterial LPS (Zambon et al, 1996; Sayers et al, 1997; Kazor et al, 1999; Shiloah et al, 2000). As a result of this information, the American Academy of Periodontology has established a separate category for smoking-associated periodontal disease (Crook et al, 2000). In addition, the role of tobacco in the aetiology of periodontitis and systemic diseases may result from its ability to elevate serum CRP (Armitage, 1999) and increase insulin-resistance (Fredriksson et al, 1999; Loos et al, 2000; Slade et al, 2000; Wu et al, 2000). However, there are reports of no effect of smoking on periodontal disease (Muller et al, 2002).

Ow and Ob are reported to be associated with periodontal attachment loss, increased pocket depth, severity of gingival bleeding and elevated plaque indices (Wood et al, 2003). The prevalence of periodontal disease in Ob individuals is highest from 18–34 years of age, and declines in persons greater than 35 years of age (Al-Zahrani et al, 2003). The significance of periodontitis as a risk factor for systemic disease in smokers has also been studied. However, the contribution of smoking by subjects with periodontitis to the risk for development of other systemic diseases has not yet been examined.

Thus, there is potential value to reinvestigate the associations between periodontitis, Ow, Ob, CVD and Db, with cigarette smoking as a positive modifier. The present study population was from Mississippi, which has the highest rate of both Ob (body mass index [BMI]>30) (24.3%) and Db (8.8%) in the United States, and an abnormally high rate of CVD and hypertension within its African American population (Campbell et al, 2003). These extremes are not evident in national studies, as the national average for the prevalence of Ob is 19.8% and of Db is 7.3% (Mokdad et al, 2001), suggesting that our regional data could provide additional information to that reported by national surveys, such as NHANES III. The periodontal data for the patient records had been collected using PSR™ (Periodontal Screening and Recording™), which has been reported to be a useful index for periodontal health, and is often used to screen, and triage, large populations for epidemiological studies (Khocht et al, 1996; Landry and Jean, 2002; Covington et al, 2003).

MATERIALS AND METHODS

Patient records

The present study was approved by the Institutional Review Board of the University of Mississippi Medical Center. In total, 1098 patient records from the University of Mississippi School of Dentistry met the inclusion criteria and were sampled by a single calibrated investigator. Inclusion criteria for the record review included active status as a patient (treated in the School of Dentistry within the past 3 years), greater than 18 years of age, and recipient of a PSR™ examination. These records contained the following information: age, gender and race, PSR™ scores, and weight and height measurements (which were used to calculate a BMI for each patient). The medical history included a self-reported history of smoking cigarettes, CVD and Db. Each medical history had been evaluated with each patient by a clinical instructor.

Patient groups

Periodontal Screening and Recording (PSR™) Codes
Patients were initially grouped using PSR™ Codes, ranging from 0 to 4. Codes were defined as the highest PSR™ score per sextant, and a code was assigned to each sextant of each dental arch. Edentulous patients were excluded from the study to prevent over-adjustment of data, which could inflate the association between periodontal and other systemic diseases (Mokdad et al, 2001). In addition, there was often no reason for previous tooth extractions in the dental records.

PSR™ Code 0 indicated periodontal health (neither bleeding on probing nor defective restoration margins and gingival sulcus depths < 3.5 mm); Code 1 indicated bleeding on probing, no defective restoration margins and a gingival sulcus depth < 3.5 mm at a minimum of one site within the sextant; Code 2 indicated bleeding on probing, the presence of supra- or sub-gingival calculus, defective restoration margins and a gingival sulcus depth < 3.5 mm at a minimum of one site within the sextant; Code 3 indicated bleeding on probing and a pocket depth of 3.5–5.5 mm at a minimum of one site within the sextant; and Code 4 indicated that a pocket depth > 5.5 mm was present at a minimum of one site within the sextant (American Dental Association and American Academy of Periodontology, 1992). The number of sextants with each code were calculated and grouped as ≤ 2, 3–4 or 5–6 sextants/Code/patient. Factorial analysis of variance (ANOVA) suggested that the groups could be pooled, so an average PSR™ Code was calculated for each subject. To obtain this code, the average of the six PSR™ Codes was calculated for each subject and rounded to the closest integer.

Medical conditions
The medical history included a self-reported history of smoking, CVD and Db. CVD was defined as a previous occurrence of one or more of the following conditions: angina pectoris, myocardial infarction, congestive heart failure, coronary artery disease or coronary heart disease. The history of Db included previous diagnosis of either Type I or Type II diseases. Patients were defined as 'non-smokers' if they had never smoked cigarettes, as 'smokers' if they presently smoked cigarettes, and as 'past smokers' if they were not currently smoking but had smoked cigarettes during their lifetime. In addition, subjects were grouped according to BMI (weight[kg]/height[m]^2). The BMI groups were classified according to the World Health Organization's recommendations as normal (BMI < 25), Ow (BMI 25–30) and Ob (BMI > 30) (Pleis and Coles, 2003).

Statistical analysis

These data were analysed using PSR™ Codes as an aetiologic factor for BMI, Db or CVD, with smoking as an effect modifier, using SPSS™ v 10.1 (SPSS Inc., Chicago, IL, USA). Initially, the groups were compared by factorial ANOVA, the Mann-Whitney U test, and chi-square analysis. Then, associations between factors and modifiers were assessed by multivariate logistic regression models (either unadjusted or following adjustment for age, race and gender) to determine relative risks for Db, CVD, Ow or Ob. A significance of $p < 0.05$ was used to determine significant differences between the groups.

RESULTS

Smoking history and demographic information

Most patients in this study were Caucasian and male without a smoking history. Factorial ANOVA suggested significant differences in the percentage of the total number of subjects with a smoking history, as a function of PSR™ Code ($p < 0.001$). The percentage of subjects without a smoking history was significantly lower in the mean PSR™ Code 2–4 groups than in PSR™ Code 0–1 groups and significantly lower in the PSR™ Code 4 group than in PSR™ Code 0–2 groups (Table 1). The mean age of the subjects was also significantly different as a function of smoking history and mean PSR™ Codes ($p < 0.001$). There were no significant differences in mean age of the subjects in the PSR™ Code 0 and 1 groups, regardless of smoking history. In the PSR™ Code 2–4 groups, the age of the non-smokers was significantly greater than in the PSR™ Code 0 and 1 groups (Table 1). Factorial ANOVA also suggested significant differences in the mean PSR™ Code as a function of gender and smoking history ($p < 0.05$). There were more males in the PSR™ Code 4 group than in other groups (Table 1). Factorial ANOVA also suggested significant differences in BMI as a function of smoking history and mean PSR™ Code ($p < 0.001$). Subjects with a PSR™ Code 2–4 and without a smoking history had a significantly higher mean BMI than those with PSR™ Codes 0–1 (Table 1).

Table 1 Relationship between mean PSR™ Codes and smoking status on demographic characteristics and body mass index (BMI) of our study population. Numbers within parentheses (x) indicate the number of subjects per group. Numbers within brackets {x} indicate the percentage of subjects with past (but not current) tobacco use. Data is expressed as mean ± standard error.

Mean PSR™ Code	Smoking history	Non-smokers (% total)	Age (years)	Males (% total)	Caucasian race (% total)	BMI (kg/m^2)
0	N (71) Y (14)	81.61 {2.30}	40.18 ± 2.16 43.21 ± 4.25	31.51 ± 5.48 21.43 ± 11.38	73.61 ± 5.23 92.86 ± 9.71	25.27 ± 0.60 26.62 ± 1.39
1	N (296) Y (38)	87.28 {0.88}	44.15 ± 1.01 40.89 ± 2.10	31.66 ± 2.63 50.00 ± 8.22*	73.70 ± 2.59 88.57 ± 5.46	26.89 ± 0.37 27.20 ± 0.95
2	N (334) Y (118)	71.37*,† {2.35}	46.63 ± 0.84**,† 41.21 ± 1.23	39.34 ± 2.67*,† 41.53 ± 4.56*,†	69.88 ± 2.56 82.05 ± 5.06	28.02 ± 0.33** 26.21 ± 0.62
3	N (142) Y (64)	66.67*,†† {1.88}	50.23 ± 1.12**,††,‡ 47.63 ± 1.63	38.73 ± 4.10 54.69 ± 6.27*,†,‡	61.03 ± 4.20 80.65 ± 5.06	29.23 ± 1.04**,†† 26.21 ± 0.62
4	N (11) Y (10)	50.00*,†,‡ {0}	42.27 ± 3.33†,‡,¶ 54.00 ± 2.72*,†	54.55 ± 15.75*,†,‡,¶ 80.00 ± 13.33*,†,‡,¶	81.82 ± 12.20 80.00 ± 13.33	28.07 ± 1.37* 26.82 ± 1.77

Y, yes; N, no
Significantly different from mean PSR™ Code 0: *p < 0.05, **p < 0.01
Significantly different from mean PSR™ Code 1: †p < 0.05, ††p < 0.01
Significantly different from mean PSR™ Code 2: ‡p < 0.05
Significantly different from mean PSR™ Code 3: ¶p < 0.05

Table 2 Crude odds ratios for the risk of being overweight or obese in subjects with a history of smoking, elevated mean PSR™ Code, or both. The F ratio represents the statistical interaction between mean PSR™ Code and smoking status on body weight, using factorial ANOVA. Numbers in parentheses (x) indicate the number of subjects per group. Risk ratios are derived from multivariate logistic regression of data adjusted for age, race and gender.

Mean PSR™ Code	Smoking history	Overweight risk ratio {95% Confidence Interval}	Overweight F ratio	Obesity risk ratio {95% Confidence Interval}	Obesity F ratio
0	N (71) Y (14)	1.00 {Reference} 1.36 {0.53–3.51}	0.801 0.733	1.00 {Reference} 2.22 {0.80–6.16}	0.439 0.351
1	N (296) Y (38)	1.05 {0.26–4.19} 1.50 {0.85–2.65}	5.370† 1.631	2.81 {0.57–13.99} 1.28 {0.73–2.23}	1.302 0.618
2	N (334) Y (118)	2.13 {0.58–7.84} 1.07 {0.76–1.49}	0.515 0.861	6.35* {1.37–29.44} 0.84 {0.58–1.20}	0.929 0.266
3	N (142) Y (64)	4.31* {1.05–17.68} 0.80 {0.50–1.26}	3.243† 0.720	4.38* {0.88–21.76} 1.28 {0.82–2.01}	6.141†† 2.498
4	N (11) Y (10)	2.60 {0.23–29.61} 0.90 {0.27–2.99}	3.883† 1.664	15.86* {0.45–55.58} 0.29* {0.03–3.02}	1.315 0.228

Y, yes; N, no
Significantly different from 'Reference': *p < 0.05
Significant interaction between mean PSR™ code and smoking status on the body weight outcome variable: †p < 0.05; ††p < 0.005

Table 3 Crude odds ratios for the risk having either diabetes mellitus or cardiovascular disease in subjects with a history of smoking, elevated mean PSR™ Code, or both. The F ratio represents the statistical interaction between mean PSR™ Code and smoking status on either diabetes mellitus or cardiovascular disease using factorial ANOVA. Numbers in parentheses (x) indicate the number of subjects per group. Risk ratios are derived from multivariate logistic regression of data adjusted for age, race and gender.

Mean PSR™ Code	Smoking history	Diabetes risk ratio {95% Confidence Interval}	Diabetes F ratio	Cardiovascular disease risk ratio {95% Confidence Interval}	Cardiovascular disease F ratio
0	N (71)	1.00 {Reference}	0.307	1.00 {Reference}	0.397
	Y (14)	0.89 {0.51–1.27}	0.518	0.53 {0.39–0.67}	0.511
1	N (296)	1.12 {0.92–1.22)	1.411	1.23 {0.92–1.47}	1.131
	Y (38)	0.82 {0.69–1.13}	1.344	0.61 {0.41–0.98}	0.788
2	N (334)	1.38 {1.11–1.47}	1.155	1.18 {0.89–1.55}	0.999
	Y (118)	0.73 {0.54–1.19}	1.161	0.72 {0.55–0.93}	0.977
3	N (142)	2.11 {1.13–3.04}	1.988	1.19 {1.02–1.49}	1.654
	Y (64)	0.71 {0.51–1.23}	1.278	0.79 {0.69–0.97}	1.879
4	N (11)	2.15 {1.17–3.45}	1.918	1.13 {0.96–1.30}	1.842
	Y (10)	0.69 {0.41–0.89}	1.422	0.87 {0.71–1.03}	0.993

Y, yes; N, no

Db, CVD, Ow and Ob risks

Subjects with a mean PSR™ Code 3 and no smoking history had a significantly higher risk for being Ow than those with mean PSR™ Codes 0, 1, 2 or 4, and those with PSR™ Codes 2–4 had a significantly higher risk of being Ob ($p < 0.05$). In contrast, subjects with a smoking history and a PSR™ Code 4 had a lower risk of being Ob ($p < 0.05$) (Table 2). The present data suggested no significantly increased risk for either Db or CVD in subjects with any PSR™ Code, regardless of smoking history (Table 3).

DISCUSSION

There is a small body of literature suggesting 'beneficial' effects of smoking on several systemic diseases (the 'Smoker's Paradox'), including gingival recession (Muller et al, 2002). Significant interactive effects between mean PSR™ Codes and smoking history on Ow and Ob were found in the present dental school patient population, which extends data from previous studies (Grossi and Ho, 2000; Muller et al, 2002; Al-Zahrani et al, 2003; Wood et al, 2003). In addition, these data were adjusted for factors associated with either periodontal disease or body weight (i.e. Db, gender, age and race), removing them as confounding variables in the final statistical model. Individual variations in personal hygiene and health care practice often confound studies of associations between periodontal inflammation and other systemic diseases (DeStefano et al, 1993) and were also uncontrolled variables in the present study.

There is evidence that periodontal disease and smoking (as separate variables) affect body weight (Perlstein and Bissada, 1977; Saito et al, 1998, 2001; Grossi and Ho, 2000; Wood et al, 2003). In addition, smoking itself is a significant risk factor for periodontal disease (Haber and Wattles, 1993; Tomar and Asma, 2000). The addition of smoking history as a variable removed the risk of being Ow or Ob and increased these risks in subjects with a healthy periodontium in the present study population. In addition, subjects with periodontitis and a smoking history did not have an increased risk for either Db or CVD, which does not support previous studies (Kim et al, 2000; Kannel et al, 2002; Ylöstalo et al, 2003).

Difficulty in distinguishing the effects of periodontitis from those of smoking with respect to health-related outcome variables has been reported (Tomar and Asma, 2000). A self-reported smoking history can be a confounding variable, which could affect the estimates for a statistical association between any of the other variables of the model. Since both body weight and mean PSR™ Code are positively associated with a smoking history in our population, a self-reported smoking history could possibly inflate their risk for Ow or Ob, producing false-positive associations between smoking, mean PSR™ Code and body weight (Tomar and Asma, 2000). In addition, adjustment of other variables for a smoking history using a poorly defined smoking/non-smoking variable (such as number of cigarettes consumed daily, which is often underreported) could place smokers into more than one group, confounding the outcome data. However, the present data were based on rigid parameters for definition of the smoking and non-smoking groups, which should have minimised those data artefacts. Reduced risk for Ow or Ob in subjects with periodontal disease and a smoking history was reported in the present population, suggesting that a significant number of smokers were excluded from the non-smoking group and that the changes in risk for Ow and Ob in subjects with periodontal disease and a smoking history were not statistical artefacts.

Smoking for weight control is prevalent, especially in adolescents (Strauss and Mir, 2001; Lowry et al, 2002; Fulkerson and French, 2003). Adult and adolescent subjects gain weight when they cease smoking (Owen-Smith and Hannaford, 1999; Peterson and Helton, 2000; Ferrara et al, 2001). In contrast, subjects with periodontitis have a risk for being Ow or Ob (Wood et al, 2003). Thus, periodontitis and a smoking history could have opposite effects on body weight. Recent studies report an inverse effect of smoking on serum levels of leptin and proinflammatory cytokines, offering a possible explanation for the variation in body weight between smokers and non-smokers (Hodge et al, 1997; Chu et al, 2001; Wallenfeldt et al, 2001; Martin et al, 2002), as Ob has now been classified as an inflammatory disease (Das, 2001).

Subjects in the present study, on average, had a BMI greater than the U.S. median (25.5 kg/m^2) (Kuczmarski et al, 1997) (except in subjects with a PSR™ Code 0 and no smoking history), supporting previous data suggesting that the Mississippi population is obese (Campbell et al, 2003). Ow and Ob have been associated with poor levels of perceived physical well-being, which is often manifested as emotional distress (Doll et al, 2000). There also is evidence that Ob may be a source of chronic stress, which adversely affects systemic health. Smoking effects on BMI varies according to education level (Laaksonen et al, 1998) and gender of the subjects (Molarius et al, 1997). These socio-economic variables could have affected our data outcomes, but were not available in the charts.

While PSR™ Codes are frequently used for screening patients for periodontal disease, the technique is not without shortcomings. There is evidence that PSR™ underestimates the severity of periodontal disease because it does not measure the epithelial attachment loss (Khocht et al, 1995). The present data was adjusted for age to minimise epithelial attachment loss as a confounding variable (Baelum et al, 1995). However, since PSR™ Codes are used by a large number of dental practitioners (Frisco and Bramson, 1993), the present use of these data will allow them to better assess the periodontal status of their patients, and the potential for successful therapies utilising smoking cessation and body weight management.

Thus, dental health professionals cannot assume that smoking will enhance the risk factors for other systemic diseases in subjects with periodontitis, as the present data suggested that it has either no effect or reduces the risk for other diseases. More studies of these associations are required to determine specific biological mechanisms for these epidemiological data; in particular, the psychosocial effects of smoking on the incidence and severity of periodontal diseases.

REFERENCES

1. Al-Zahrani MS, Bissada NF, Borawski EA. Obesity and periodontal disease in young, middle-aged, and older adults. J Periodontol 2003;74:610-615.
2. American Dental Association and American Academy of Periodontology. Periodontal Screening and Recording Training Program Kit. Chicago: American Academy of Periodontology, 1992.
3. Arbes SV, Slade GD, Beck JD. Association between extent of periodontal attachment loss and self-reported history of heart attack: an analysis of NHANES III data. J Dent Res 1999; 78:1777-1782.
4. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999;4:1-6.
5. Baelum V, Manji F, Wanzala P, Fejerskov O. Relationship between CPITN and periodontal attachment loss findings in an adult population. J Clin Periodontol 1995;22:146-152.
6. Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. J Periodontol 1996;67(10 Suppl):1123-1137.
7. Beck J, Offenbacher S, Williams R, Gibbs P, Garcia R. Periodontitis: a risk factor for coronary heart disease. Ann Periodontol 1998;3:127-141.
8. Bergstrom J, Preber H. Tobacco use as a risk factor. J Periodontol 1994;65:545-550.

9. Bostrom L, Linder LE, Bergstrom J. Smoking and crevicular fluid levels of IL-6 and TNF-alpha in periodontal disease. J Clin Periodontol 1999;26:352-357.
10. Campbell BW, Addison CC, Charles L, Thurston DA. Cardiovascular risk factors among women in Mississippi in the 1990s. J Am Med Womens Assoc 2003;58:105-111.
11. Castell JV, Andus T, Kunz D, Heinrich P. Interleukin-6: the major regulator of acute-phase protein synthesis in man and rat. Ann NY Acad Sci 1989;557:87-99.
12. Chu NF, Stampfer MJ, Spiegelman D, Rifai N, Hotamisligil GS, Rimm EB. Dietary and lifestyle factors in relation to plasma leptin concentrations among normal weight and overweight men. Int J Obes Relat Metab Disord 2001;25:106-114.
13. Cianciola LJ, Park BH, Bruck E, Mosovich L, Genco RJ. Prevalence of periodontal disease in insulin-dependent diabetes mellitus (juvenile diabetes). J Am Dent Assoc 1982;104:653-660.
14. Covington LL, Breault LG, Hokett SD. The application of Periodontal Screening and Recording™ (PSR) on a military population. J Contemp Dent Pract 2003;4:24-39.
15. Crook MA, Scott DA, Stapleton JA, Palmer RM, Wilson RF, Sutherland G. Circulating concentrations of C-reactive protein and total sialic acid in tobacco smokers remain unchanged following one year of validated smoking cessation. Eur J Clin Invest 2000;30:861-865.
16. Das UN. Is obesity an inflammatory condition? Nutrition 2001; 17:1416-1420.
17. de Maat MP, Kluft C. The association between inflammation markers, coronary artery disease and smoking. Vascul Pharmacol 2002;39:137-139.
18. DeStefano F, Anda RF, Kahn HS, Williamson DF, Russell CM. Dental disease and risk of coronary heart disease and mortality. Br Med J 1993;306:688-691.
19. Doll HA, Petersen SEK, Stewart-Brown SL. Obesity and physical and emotional well-being: associations between body mass index, chronic illness, and the physical and mental components of the SF-36 questionnaire. Obes Res 2000;8:160-170.
20. Donahue RP, Wu T. Insulin resistance and periodontal disease: an epidemiologic overview of research needs and future directions. Ann Periodontol 2001;6:119-124.
21. Ebersole JL, Machen RL, Steffen MJ, Willmann DE. Systemic acute-phase reactants, C-reactive protein and haptoglobin, in adult periodontitis. Clin Exp Immunol 1997;107:347-352.
22. Emrich LJ, Schlossman M, Genco RJ. Periodontal disease in non-insulin-dependent diabetes mellitus. J Periodontol 1991;62:123-130.
23. Ferrara CM, Kumar M, Nicklas B, McCrone S, Goldberg AP. Weight gain and adipose tissue metabolism after smoking cessation in women. Int J Obes Rel Metabol Dis 2001;25:1322-1326.
24. Fowler EB, Breault LG, Cuenin MF. Periodontal disease and its association with systemic disease. Mil Med 2001;166:85-89.
25. Fredriksson MI, Figueredo CMS, Gustafsson A, Bergstrom KG, Asman BE. Effect of periodontitis and smoking on blood leukocytes and acute-phase proteins. J Periodontol 1999;70:1355-1360.
26. Frisco CL, Bramson JB. Periodontal screening and recording: perceptions and effects on practice. J Am Dent Assoc 1993;24:226-232.
27. Fulkerson JA, French SA. Cigarette smoking for weight loss or control among adolescents: gender and racial/ethnic differences. J Adolesc Health 2003;32:306-313.
28. Genco RJ. Periodontal disease and risk for myocardial infarction and cardiovascular disease. Cardiovasc Rev Rep 1998;19:34-40.
29. Genco R, Offenbacher S, Beck J. Periodontal disease and cardiovascular disease. Epidemiology and possible mechanisms. J Am Dent Assoc 2002;133:14S-22S.
30. Grossi SG, Ho AW. Obesity, insulin resistance and periodontal disease. J Dent Res 2000;79(Spec issue):625.
31. Haber J, Wattles J. Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontol 1993;64:16-23.
32. Hodge AM, Westerman RA, de Courten MP, Collier GR, Zimmet PZ, Alberti KG. Is leptin sensitivity the link between smoking cessation and weight gain? Int J Obes Relat Metab Dis 1997;21:50-53.
33. Joshipura KJ, Rimm EB, Douglass CW, Trichopoulos D, Ascherio A, Willett WC. Poor oral health and coronary artery disease. J Dent Res 1996;75:1631-1636.
34. Kannel WB, Wilson PW, Nam BH, D'Agostino RB. Risk stratification of obesity as a coronary risk factor. Am J Cardiol 2002;90:697-701.
35. Katz J. Elevated blood glucose levels in patients with severe periodontal disease. J Clin Periodontol 2001;28:710-712.
36. Kazor C, Taylor GW, Loesche WJ. The prevalence of BANA-hydrolyzing periodontopathic bacteria in smokers. J Clin Periodontol 1999;26:814-821.
37. Khocht A, Zohn H, Deasy M, Chang KM. Assessment of periodontal status with PSR and traditional clinical periodontal examination. J Am Dent Assoc 1995;126:1658-1665.
38. Khocht A, Zohn H, Deasy M, Chang KM. Screening for periodontal disease: radiographs vs. PSR. J Am Dent Assoc 1996;127:749-756.
39. Kim KS, Owen WL, Williams D, Adams-Campbell LL. A comparison between BMI and Conicity index on predicting coronary heart disease: the Framingham Heart Study. Ann Epidemiol 2000;10:424-431.
40. Kuczmarski RJ, Carroll MD, Flegal KM, Troiano RP. Varying body mass index cutoff points to describe overweight prevalence among U.S. adults: NHAMNES III (1988 to 1994). Obes Res 1997;5:542-548.
41. Laaksonen M, Rahkonen O, Prattala R. Smoking status and relative weight by educational level in Finland, 1978–1995. Prev Med 1998;27:431-437.
42. Landry RG, Jean M. Periodontal Screening and Recording (PSR) index: precursors, utility and limitations in a clinical setting. Int Dent J 2002;52:35-40.
43. Löe H. The sixth complication of diabetes mellitus. Diabetes Care 1993;16:329-334.
44. Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillen PM, van der Velden U. Evaluation of systemic markers related to cardiovascular disease in the peripheral blood of periodontitis patients. J Periodontol 2000;71:1528-1534.
45. Lowry R, Galuska DA, Fulton JE, Wechsler H, Kann L. Weight management goals and practices among U. S. high school students: associations with physical activity, diet and smoking. J Adolesc Health 2002;31:133-144.
46. Martin LJ, Cole SA, Hixson JE, Mahaney MC, Czerwinski SA, Almasy L et al. Genotype by smoking interaction for leptin levels in the San Antonio Family Heart Study. Genet Epidemiol 2002;22:105-115.
47. Matthews DC. The relationship between diabetes and periodontal disease. J Can Dent Assoc 2002;68:161-164.
48. Mattila K, Nieminen MS, Valtonen VV, Rasi VP, Kesäniemi YA, Syrjälä SL et al. Association between dental health and acute myocardial infarction. Br Med J 1989;298:779-781.
49. Mealey B. Diabetes and periodontal disease. J Periodontol 1999;70:935-949.

50. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. The continuing epidemics of obesity and diabetes in the United States. J Am Med Assoc 2001;286:1195-2000.
51. Molarius A, Seidell JC, Kuulasmaa K, Dobson AJ, Sans S. Smoking and relative body weight: an international perspective from the WHO MONICA Project. J Epidemiol Community Health 1997;51:252-260.
52. Muller H-P, Stadermann S, Heinecke A. Gingival recession in smokers and non-smokers with minimal periodontal disease. J Clin Periodontol 2002;29:129-136.
53. Nelson RG, Shlossman M, Budding LM, Pettitt DJ, Saad MF, Genco RJ, Knowler WC. Periodontal disease and NIDDM in Pima Indians. Diabetes Care 1990;13:836-840.
54. Owen-Smith V, Hannaford PC. Stopping smoking and body weight in women living in the United Kingdom. Br J Gen Pract 1999;49:989-990.
55. Papapanou PN. Periodontal diseases: epidemiology. Ann Periodontol 1996;1:1-37.
56. 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.
57. Peterson AL, Helton J. Smoking cessation and weight gain in the military. Mil Med 2000;165:536-538.
58. Pleis JR, Coles R. Summary health statistics for U.S. adults: National Health Interview Survey, 1999. Vital Health Stat 10 2003;212:1-137.
59. Pradhan AD, Manson JE, Rifai N, Buring JE, Ridker PM. C-reactive protein, interleukin-6, and risk of developing type 2 diabetes mellitus. J Am Med Assoc 2001;286:327-334.
60. Safkan-Seppala B, Ainamo J. Periodontal conditions in insulin-dependent diabetes mellitus. J Clin Periodontol 1992;19:24-29.
61. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between upper body obesity and periodontitis. J Dent Res 2001;80:1631-1636.
62. Saito T, Shimazaki Y, Sakamoto M. Obesity and periodontitis. N Eng J Med 1998;339:482-483.
63. Sayers NM, Gomes BP, Drucker DB, Blinkhorn AS. Possible lethal enhancement of toxins from putative periodontopathogens by nicotine: implications for periodontal disease. J Clin Pathol 1997;50:245-249.
64. Shiloah J, Patters MR, Waring MB. The prevalence of pathogenic periodontal microflora in healthy young adult smokers. J Periodontol 2000;71:562-567.
65. Slade GD, Offenbacher S, Beck J, Heiss G, Pankow JS. Acute-phase inflammatory response to periodontal disease in the U.S. population. J Dent Res 2000;79:49-57.
66. Strauss RS, Mir HM. Smoking and weight loss attempts in overweight and normal-weight adolescents. Int J Obes Relat Metab Disord 2001;25:1381-1385.
67. Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: an epidemiologic perspective. Ann Periodontol 2001;6:99-112.
68. Tomar S, Asma S. Smoking-attributable periodontitis in the United States: Findings from NHANES III. J Periodontol 2000;71:743-751.
69. Wallenfeldt K, Hulthe J, Bokemark L, Wikstrand J, Fagerberg B. Carotid and femoral atherosclerosis, cardiovascular risk factors and C-reactive protein in relation to smokeless tobacco use or smoking in 58-year-old men. J Intern Med 2001;250:492-501.
70. 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.
71. Wu T, Trevisan M, Genco RJ, Falkner KL, Dorn JP, Sempos CT. Examination of the relation between periodontal health status and cardiovascular risk factors: serum total and high density lipoprotein cholesterol, C-reactive protein, and plasma fibrinogen. Am J Epidemiol 2000;151:273-282.
72. Ylöstalo PV, Ek E, Laitinen J, Knuuttila ML. Optimism and life satisfaction as determinants for dental and general health behavior-oral health habits linked to cardiovascular risk factors. J Dent Res 2003;82:194-199.
73. Zambon JJ, Grossi SG, Machtei EE, Ho AW, Dunford R, Genco RJ. Cigarette smoking increases the risk for subgingival infection with periodontal pathogens. J Periodontol 1996;67(10 Suppl):1050-1054.