Self-reporting of periodontal diseases and clinical assessment outcome in a Swedish urban population of smokers and non-smokers

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The aim of this study was to determine whether there is an association between self-reporting of periodontal diseases and outcome in a clinical examination, and whether any difference is present in awareness of periodontal status between smokers and non-smokers. Participants comprised 1676 adults (838 M and 838 F aged between 31 and 40 years), 564 of whom reported being smokers. Subjects were asked via questionnaire whether they thought they had periodontal disease and why. A total of 1655 subjects answered the questionnaire and were subsequently divided into those who suspected having periodontal disease (Yes-group) and those who did not (No-group). A full-mouth clinical examination was carried out in all subjects. Female smokers in the Yes-group had a significantly higher number of teeth with pockets $\geq$5 mm ($P < 0.001$) and a higher calculus index (CI-S, $P < 0.01$) than female smokers in the No-group. Male smokers in the Yes-group had significantly less remaining teeth ($P < 0.01$), more teeth with pockets $\geq$5 mm ($P < 0.001$), and a higher CI-S ($P < 0.05$) than their counterparts in the No-group. For smokers, multivariate logistic regression analysis yielded an odds ratio (OR 3.21 [95% CI 1.73–5.74]) of self-reported periodontal disease to periodontitis outcome which was significant ($P < 0.001$). This association remained significant ($P < 0.01$) after adjustment for confounding factors. Subjects who reported having periodontal disease, especially those who also reported having movable teeth, were confirmed to have the disease. Smokers were more aware of their periodontal status than non-smokers. □ *Clinical outcome; periodontitis; questionnaire; self-report; smoking*

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Self-reported oral health questionnaires are used widely in epidemiological oral health investigations because they are time- and cost-effective and provide detailed information on subjects in a single health examination (1–9). The validity and reliability of these questionnaires are crucial if the self-assessed data on oral health and disease is to be useful (1, 2, 10, 11). Ho et al. (12) concluded that although information provided by the subject may not be as accurate as laboratory testing when screening general and oral health of populations, it is nevertheless a valuable source of data that can be utilized cost-effectively in research studies. Self-reporting can even be used for educational purposes. In a study among adolescents, Kallio et al. (3) demonstrated that gingival health can be improved by using questionnaires as an educative tool.

Questionnaire data on the number of teeth and removable dentures are generally in fairly good agreement with clinical examinations (2–5). However, Palmqvist et al. (13) found that self-reporting is reliable for dentures but not for number of teeth. In a meta-analysis, Patric et al. (14) concluded that self-reports of smoking are accurate in most studies. Tobacco smoking is a well-established risk factor for periodontal disease (15–18). Compared with non-smokers, smokers have a higher prevalence of periodontitis (17), an increased rate of disease progression, and less response to periodontal treatment (16, 19–24). Further, smokers are more susceptible to aggressive forms of periodontitis (20). Smokers have been shown to exhibit less inflammatory response and bleeding on probing than non-smokers at similar plaque levels (19, 23). Moreover, the effects of smoking on periodontal tissues depend on the daily dose and duration of tobacco smoking (25, 26). The impact of smoking on periodontal tissues seems to be more pronounced in men than in women (27).

Questions about gingival bleeding have been included in several epidemiological studies. Ankkuriniemi and Ainamo (6) and Buhlin et al. (28) have shown that self-reported gingival bleeding is correlated with gingival bleeding at clinical examination. Kallio et al. (3) concluded that self-reporting was a useful tool in screening the gingival health of populations but not individuals. A recent study has shown that a periodontal questionnaire is a valuable instrument in epidemiological studies of periodontal health, although it is less reliable for specific periodontal variables such as tooth movability (28).

While few studies have compared self-reported questionnaires and periodontal status, those available show good overall agreement (9, 11, 28, 29). The aim of this study was to further clarify the association between self-reporting of periodontal disease and outcome in a clinical examination and determine whether any difference is present in awareness of periodontal status between smokers and non-smokers.

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Subjects and methods

The subjects consisted of 1676 adults (838 F, and 838 M), aged 31–40 years, who came to the clinical examination from a randomly selected epidemiological sample in the Stockholm region. They were contained within a registry file of all inhabitants of the Stockholm area born on the 20th of any month between 1945 and 1954 ($n = 3173$) (21).

The response rate was 52.4%. A clinical examination was performed in 1985–1986. The drop-out control sample consisted of 100 randomly selected subjects (55 F and 45 M) from the non-respondents. In the waiting room, before clinical examination, participants were asked on a questionnaire about their smoking habits and whether they thought that they had a periodontal disease (yes [Yes-group] or no [No-group]). The complementary question 'Why do you think you have a periodontal disease?' was answered if the participants thought they had a periodontal disease. A full-mouth clinical examination was performed by 6 periodontists. Examiners received 6 training sessions to ensure consistent registration of the parameters being investigated (30). The clinical examination comprised determination of the number of remaining teeth (excluding 3rd molars), presence of plaque (PLI) (31), calculus by simplified calculus index (S-CI) (32), and registration of gingival inflammation (GI) (33). The number of teeth with a probing pocket depth $\geq$5 mm was recorded. Pocket depth was measured with a Hu-Friedy probe (Hu-Friedy PCPUNC 15, Chicago, Il., USA) and was registered to the nearest millimeter at 6 sites per tooth. Periodontitis was defined as 3 or more teeth, excluding 3rd molars, with pockets $\geq$5 mm probing depth.

Statistical methods

Analysis of variance (ANOVA) and logistic multivariate regression analysis were used. Differences between data sets with a probability of less than 0.05 were regarded as significant. All data analysis was performed using the Stat View® 4.5 statistical package (34), and the diagnostic accuracy of self-reported periodontitis was expressed by the area under the receiver operating characteristics curve (ROC analysis) using SPSS 9.0 software (35).

Results

Of the 1676 subjects who came to the clinical examination, 162 had clinical manifestations of periodontitis (9.7%) according to our diagnostic criteria for periodontal disease. No significant differences were found between subjects and dropout controls regarding the presence of periodontal pockets, smoking habits, or the interval between dental visits. Mean age for responders was 35.7 ($s \pm 2.9$) years. The questionnaire was completed by 1655 subjects (827 F and 828 M) (98%). Of these, 72.0% had visited their dentist at least once a year, 16.6% every second year, and the remaining 11.4% had not visited a dentist during the previous 3 years. The 200 subjects (102 F and 98 M) who reported having a periodontal disease comprised the Yes-group. The 1437 (719 F and 718 M) who reported not having a periodontal disease formed the No-group. Eighteen subjects answered 'I don't know'. Clinical criteria for periodontal disease were fulfilled for 50 subjects (25.0%) in the Yes-group and 91 subjects (6.3%) in the No-group. A significant difference was found between the groups ($P < 0.001$). In addition, 104 subjects answered the complementary question 'Why do you think you have a periodontal disease?' The response 'Informed by the dentist or dental hygienist' is excluded from the main analysis and is analyzed separately. In all, 33 subjects reported having been informed of their periodontitic status by their dentist or dental hygienist, and, of these, 20 did not fulfill our criteria for periodontitis. Subjects who reported having gingival bleeding had periodontal disease in 20% of cases. Moreover, 60% of those who said they had movable teeth were found to have periodontal disease. The outcome of clinical examinations for the Yes- and No-groups is presented in Tables 1, 2, and 3 for non-smokers,

Table 1. Mean and 95% confidence interval (CI) of plaque index percentage (% PLI), gingival index percentage (% GI), calculus index (CI-S), number of teeth present, and number of teeth with pockets $\geq$5 mm (PD $\geq$5 mm) for non-smokers; females and males divided into Yes- and No-groups

	Yes-group				No-group			
	Female		Male		Female		Male	
	n = 25		n = 24		n = 272		n = 282	
Parameters	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI
% PLI $\leq$1	87.3	81.6–93.0	85.4	76.1–94.8	91.6	89.7–93.5	86.9	84.3–89.5
% PLI $\geq$2	12.7	7.0–18.4	14.6	5.2–23.9	8.4	6.5–10.3	13.1	10.5–15.7
% GI $\leq$1	65.9*	52.2–79.7	54.1	37.3–70.8	79.7	76.5–83.0	68.7	65.1–72.3
% GI $\geq$2	34.1*	20.3–47.8	45.7*	29.2–62.7	20.3	17.0–23.5	31.3	27.7–34.9
CI-S	0.3	0.2–0.5	0.7*	0.3–1.0	0.3	0.2–0.3	0.4	0.3–0.4
No. of teeth present	26.7	25.9–27.5	26.5	25.6–27.5	26.8	26.6–27.1	27.0	26.8–27.3
No. of teeth (PD $\geq$5 mm)	0.6	0.0–1.1	1.3*	0.0–3.0	0.2	0.1–0.4	0.4	0.2–0.5

Significant difference between groups of the same gender * $P < 0.05$.

Table 2. Mean and 95% confidence interval (CI) of plaque index percentage (% PLI), gingival index percentage (% GI), calculus index (CI-S), number of teeth present, and number of teeth with pockets ≥5 mm (PD ≥5 mm) for ex-smokers; females and males divided into Yes- and No-groups

	Yes-group				No-group			
	Female		Male		Female		Male	
	n = 20		n = 20		n = 187		n = 186	
Parameters	Mean	95% CI	Mean	95% CI	Mean	95%CI	Mean	95%CI
% PLI ≤1	88.1	75.4–100.0	77.0	62.7–91.3	90.2	87.5–92.8	84.7	81.4–88.0
% PLI ≥2	11.9	0.0–24.6	23.0	8.7–37.3	9.8	7.2–12.5	15.3	12.0–18.6
% GI ≤1	61.5	44.0–79.0	51.6	33.9–69.2	76.8	72.9–80.8	60.7	56.0–65.4
% GI ≥2	38.5*	21.0–56.0	48.4	30.8–66.1	23.1	19.2–27.1	39.3	34.6–44.0
CI-S	0.5	0.2–0.8	0.9**	0.5–1.3	0.3	0.3–0.4	0.5	0.4–0.6
No. of teeth present	27.0	26.4–27.7	27.1	26.5–27.7	27.1	26.9–27.3	27.0	26.7–27.3
No. of teeth (PD ≥5 mm)	1.7**	0.0–3.7	1.6	0.1–3.2	0.4	0.2–0.6	0.9	0.6–1.3

Significant difference between groups of the same gender * $P < 0.05$,** $P < 0.01$.

ex-smokers, and smokers, respectively. Non-smoker females in the Yes-group had a significantly higher percentage of GI ≥2 than non-smoker females in the No-group ($P < 0.05$) (Table 1). Non-smoker males in the Yes-group also had a significantly higher percentage of GI ≥2 as well as a higher CI-S and more teeth with pockets ≥5 mm ($P < 0.05$) than their peers in the No-group (Table 1). Ex-smoker females in the Yes-group had a significantly higher percentage of GI ≥2 ($P < 0.05$) and a larger number of teeth with pockets ≥5 mm ($P < 0.01$) than ex-smoker females in the No-group (Table 2). Ex-smoker males in the Yes-group had a significantly higher CI-S than males in the No-group ($P < 0.01$). Female smokers in the Yes-group had a significantly higher CI-S ($P < 0.01$) and more teeth with pockets ≥5 mm ($P < 0.001$) than their counterparts in the No-group (Table 3). Male smokers in the Yes-group had a significantly higher CI-S ($P < 0.05$), less remaining teeth ($P < 0.01$), and more teeth with pockets ≥5 mm ($P < 0.001$) than male smokers in the No-group (Table 3). For smokers, multivariate logistic regression analysis yielded an odds ratio (OR 3.21 [95% CI 1.73–5.74]) of self-reported periodontal disease to periodontitis outcome which was significant ($P < 0.001$). This association remained significant ($P < 0.01$) after adjustment for the confounding factors of gender, age, and dental visits (Table 4). The diagnostic accuracy was also tested by ROC analysis of clinical parameters gingivitis, calculus, number of teeth with pockets ≥5 mm, and plaque index for never-smokers (Table 5).

Discussion

The sample was considered to present the entire population of individuals born in 1945–1954 in the Stockholm area. Here periodontitis was defined as 3 or more teeth, excluding 3rd molars, with pockets ≥5 mm. Periodontal disease can also be defined by other parameters, including attachment level, radiographic bone level, other probing depth levels and various pocket frequency cut-off points.

Table 3. Mean and 95% confidence interval (CI) of plaque index percentage (% PLI), gingival index percentage (% GI), calculus index (CI-S), number of teeth present, and number of teeth with pockets ≥5 mm (PD ≥5 mm) for smokers; females and males divided into Yes- and No-groups

	Yes-group				No-group			
	Female		Male		Female		Male	
	n = 36		n = 33		n = 253		n = 242	
Parameters	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI
% PLI ≤1	85.6	78.0–93.1	82.8	74.6–91.1	86.6	84.4–88.9	80.4	77.3–83.5
% PLI ≥2	14.4	6.9–22.0	17.2	8.9–25.4	13.4	11.2–15.6	19.6	16.5–22.7
% GI ≤1	51.0	38.4–63.5	35.8	23.0–48.5	66.1	62.0–70.2	51.9	47.2–56.6
% GI ≥2	49.0	36.5–61.6	64.2	51.5–77.0	33.9	29.8–38.0	48.1	43.4–52.8
CI-S	0.7**	0.5–1.0	0.9*	0.6–1.1	0.5	0.4–0.5	0.6	0.5–0.7
No. of teeth present	26.6	26.0–27.2	24.9**	22.9–26.3	26.7	26.5–26.9	26.5	26.2–26.9
No. of teeth (PD ≥5 mm)	2.6***	0.9–4.3	2.9***	1.4–4.5	0.9	0.6–1.2	1.0	0.6–1.3

Significant difference between groups of the same gender * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

Table 4. Multivariate logistic regression analysis of self-awareness of periodontal disease with number of teeth with PD ≥5 mm as a dependent variable

	Odds ratio			Odds ratio		
Variable	Unadjusted	*P*	95% CI	Adjusted[a)]	*P*	95% CI
Nonsmoker, n = 49	2.35	N.S.[b)]	0.66–8.42	2.90	N.S.	0.78–11.12
Ex-smoker, n = 43	2.43	N.S.	0.99–5.96	2.08	<0.001	0.74–5.87
Smoker, n = 75	3.21	<0.001	1.73–5.74	3.10	<0.01	1.57–6.09

[a)]Odds ratios adjusted for gender, age, and regular dental visits.
[b)]N.S. = not significant.

How periodontal disease is defined has an impact on disease prevalence (36). The complementary question confirmed that most subjects in the Yes-group reported having periodontitis because of bleeding gums when the answer 'Informed by dentist or dental hygienist' was excluded. Smoking habits had an impact on results. Both non-smoking females and males in the Yes-group had a significantly higher percentage of GI ≥2 than non-smoking individuals in the No-group. However, non-smokers in the Yes-group did not have periodontitis according to the periodontitis criteria applied (Table 1).

Between smokers in the Yes- and No-groups, no difference was seen in GI, while a significant difference was observed in the number of teeth with pockets ≥5 mm (Table 3). No clinical differences in GI ≥2 were present in smokers of the Yes- and No-groups; however, in both groups, a high percentage of GI ≥2 was observed, which seems to be smoking-dependent. In addition, some differences in results between the Yes- and No-groups for male and female smokers were found. Male smokers in the Yes-group compared with male smokers in the No-group had significantly less teeth. No such difference was present among female smokers (Table 3). This difference can be explained by the effect of tobacco on periodontal tissues being more pronounced in males than in females (27). Both female and male smokers had significantly more periodontal pockets ≥5 mm in the Yes-group than in the No-group (Table 3). Laurell et al. (37) found that smokers have a higher incidence of bone loss in upper and lower incisors, making it easier for smokers to self-assess periodontal diseases. This may be one of the reasons why smokers were more aware of their periodontal disease. One of the most common answers to the complementary question in our study was 'hereditary periodontitis'. Background determinants associated with periodontal diseases, including hereditary factors, have been discussed elsewhere (38). Evidence suggests that there is a significant genetic component to susceptibility and resistance to chronic periodontal disease (39). According to McGuire and Nunn (40), a positive IL-1 genotype increases risk of tooth loss 2.7-fold, in comparison with heavy smoking, which increases this risk 2.9-fold. In our study, smokers who reported having periodontitis had a 3.1-fold risk of periodontal disease even after adjustment for confounding factors (Table 4).

The results of this investigation will form the basis for recall of patients for a 10-year follow-up protocol.

It was concluded that subjects who reported having periodontal disease, especially those also reporting having movable teeth, were confirmed to have the disease. Smokers were more aware of their periodontal status than non-smokers.

Table 5. Prognostic analysis of clinical data results for nonsmokers who answered yes to the question "Do you think you have periodontitis?"

	AUROC (Prognostic accuracy)
Gingivitis	0.62
Calculus	0.63
No. of teeth with PD ≥5 mm	0.62
Plaque Index	0.56

Near 0 or 1: Good accuracy.
Near 0.5: Random accuracy.

References

1. Helöe LA. Comparison of dental health data obtained from questionnaires, interviews and clinical examination. Scand J Dent Res 1972;80:495–9.
2. Könönen M, Lipasti J, Murtomaa H. Comparison of dental information obtained from self-examination and clinical examination. Community Dent Oral Epidemiol 1986;14:258–60.
3. Kallio P, Nordblad A, Croucher R, Ainamo J. Self-reported gingivitis and bleeding gums among adolescents in Helsinki. Community Dent Oral Epidemiol 1994;22 (5 Pt 1):277–82.
4. Axelsson G, Helgadottir S. Comparison of oral health data from self-administered questionnaire and clinical examination. Community Dent Oral Epidemiol 1995;23:365–8.
5. Kallio P, Uutela A, Nordblad A, Alvesalo I, Murtomaa H, Croucher R. Self-assessed bleeding and plaque as methods for improving gingival health in adolescents. Int Dent J 1997;47:205–12.
6. Ankkuriniemi O, Ainamo J. Dental health and dental treatment needs among recruits of the Finnish Defence Forces, 1919–91. Acta Odontol Scand 1997;55:192–7.
7. Kallio P, Murtomaa H. Determinants of self-assessed gingival health among adolescents. Acta Odontol Scand 1997;55:106–10.
8. Kawamura M, Fukuda S, Kawabata K, Iwamoto Y. Comparison of health behavior and oral/medical conditions in non-

insulin-dependent (typ 2) diabetics and non-diabetics. Aust Dent J 1998;43:315–20.
9. Gilbert AD, Nuttall NM. Self-reporting of periodontal health status. Br Dent J 1999;186:241–4.
10. Douglass CW, Berlin J, Tennstedt S. The validity of self-reported oral health status in the elderly. J Public Health Dent 1991;51: 220–2.
11. Unell L, Söderfeldt B, Halling A, Paulander J, Birkhed D. Oral disease, impairment, and illness: congruence between clinical and questionnaire findings. Acta Odontol Scand 1997;55:127–32.
12. Ho A, Grossi S, Dunford R, Genco R. Reliability of a self-reported health questionnaire in a periodontal disease study. J Periodontal Res 1997;32:646–50.
13. Palmqvist S, Söderfeldt B, Arnbjerg D. Self-assessment of dental conditions: validity of a questionnaire. Community Dent Oral Epidemiol 1991;19:249–51.
14. Patric DL, Chealle A, Thompson D, Dier P, Koepsell T, Kinne S. The validity of self-reported smoking: a review and meta-analysis. Am J Public Health 1994;84:1086–93.
15. Bergström J. Cigarette smoking as a risk factor in chronic periodontal disease. Community Dent Oral Epidemiol 1989;17: 245–7.
16. Haber J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontol 1993;64:16–23.
17. Papapanou PN. Periodontal diseases: epidemiology. Ann Periodontol 1996;1:1–36.
18. Norderyd O, Hugoson A. Risk of severe periodontal disease in a Swedish adult population. A cross-sectional study. J Clin Periodontol 1998;25:1022–8.
19. Preber H, Bergström J. Occurrence of gingival bleeding in smoker and non-smoker patients. Acta Odontol Scand 1985;43: 315–20.
20. Sakki TK, Knuuttila ML, Vimpari SS, Hartikainen MS. Association of lifestyle with periodontal health. Community Dent Oral Epidemiol 1995;23:155–8.
21. Söder B, Jin LJ, Söder P-Ö, Wikner S. Clinical characteristics of destructive periodontitis in a risk group of Swedish urban adults. Swed Dent J 1995;19:9–15.
22. Genco RJ. Current view of risk factors for periodontal diseases. J Periodontol 1996;67 (10 Suppl):1041–9.
23. Grossi SG, Zambon J, Machtei EE, Schifferle R, Andreana S, Genco RJ, et al.. Effects of smoking and smoking cessation on healing after mechanical periodontal therapy. J. Am Dent Assoc 1997;128:599–607.
24. Jin L, Wong KYN, Leung WK, Corbet EF. Comparison of treatment response patterns following scaling and root planing in smokers and non-smokers with untreated adult periodontitis. J Clin Dent 2000;11:35–41.
25. Haber J, Kent R. Cigarette smoking in a periodontal practise. J Periodontol 1992;63:100–6.
26. Martinez-Canut P, Lorca A, Magan R. Smoking and periodontal disease severity. J Clin Periodontol 1995;22:743–9.
27. Calsina G, Ramon J-M, Echeverria JJ. Effects of smoking on periodontal tissues. J Clin Periodontol 2002;29:771–6.
28. Buhlin K, Gustafsson A, Andersson K, Håkansson J, Klinge B. Validity and limitations of self-reported periodontal health. Community Dent Oral Epidemiol 2002;30:431–7.
29. Joshipura KJ, Douglass CW, Garcia RI, Valachovic R, Willett WC. Validity of a Self-reported periodontal disease measure. J Public Health Dent 1996;56:205–12.
30. Söder P-Ö, Frithiof L, Lavstedt S, Wikner S, Wouters F, Engström P-E. Prevalence of periodontitis in the 31–40-year-old population of 1380 subjects in Stockholm. In: Lehner T, Cimasoni G, editors. The borderland between caries and periodontal disease III. Genève: editions Médecine et Hygiène; 1986. p. 39–43.
31. Silness J, Löe H. Periodontal disease in pregnancy. II: Correlation between oral hygiene and periodontal condition. Acta Odontol Scand 1964;22:121–35.
32. Green JC, Vermillion JR. The simplified oral hygiene index. J Am Dent Assoc 1964;68:7–13.
33. Löe H, Sillness J. Periodontal disease in pregnancy. (I) Prevalence and severity. Acta Odontol Scand 1963;21:533–51.
34. SAS Institute Inc. Stat View Reference. 1999 3rd ed. Gary, NC: SAS Institute Inc..
35. Metz C. Basic principles of ROC analysis. Semin Nucl Med 1978. p. 283–98.
36. Bergström J. Tobacco smoking and risk for periodontal disease. J Clin Periodontol 2003;30:107–13.
37. Laurell L, Romao C, Hugoson A. Longitudinal study on the distribution of proximal sites showing significant bone loss. J Clin Periodontol 2003;30:346–52.
38. Genco RJ. Current view of risk factors for periodontal diseases. J Periodontol 1996;67:1041–9.
39. Baker P, Roopenian DC. Genetic susceptibility to chronic periodontal disease. Microbes Infect 2002;11:1157–67.
40. McGuire MK, Nunn ME. Prognosis versus actual outcome. IV. The effectiveness of clinical parameters and IL-1 genotype in accurately predicting prognoses and tooth survival. J Periodontol 1999;70:49–56.

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