Periodontal Conditions in a Swedish City Population of Adolescents: A Comparison Between Smokers and Never-Smokers

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Purpose: The aim of this study was to evaluate the influence of smoking on the periodontal conditions of a randomly selected population sample of 19-year-old individuals.

Material and Methods: A population sample of 272 randomly selected 19-year-old individuals living in Göteborg, Sweden, was clinically examined with regard to oral hygiene, gingivitis, deepened periodontal pockets, probing attachment loss (PAL) and gingival recession. On bitewing radiographs, alveolar bone level and presence of dental calculus were assessed. The subjects were classified as 'never-smokers' or smokers based on information obtained by a questionnaire-based interview.

Results: The mean plaque and gingivitis scores were for never-smokers 58% (SE 1.7) and 46% (1.4) respectively, and for smokers 60% (2.2) and 42% (2.1) respectively. Of the never-smokers and smokers respectively, 38% and 35% showed a prevalence of gingivitis of >50%. The mean number of sites with periodontal pockets ≥4 mm was 12 (0.9) in never-smokers and 13 (1.4) in smokers. Of both never-smokers and smokers, 75% did not have any site with PAL ≥2 mm, and only six individuals (3%), all never-smokers, showed more than three sites with a PAL of ≥2 mm (facial sites). Logistic regression analyses revealed that smoking was a poor discriminator for identification of subjects with periodontal destruction (OR 0.62–1.33).

Conclusions: In the present population sample of adolescents, characterised by high prevalence of plaque and gingivitis, smoking habits did not contribute to a higher prevalence or severity of periodontal destruction.

Key words: adolescents, periodontal disease, smoking

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Cigarette smoking is considered to be an important risk factor for periodontal diseases (Ismail et al, 1983; Tonetti, 1998; Hujoel et al, 2003). In young individuals, among whom recent reports indicate an increase in tobacco consumption both in developing and developed countries (Wechsler et al, 1998; Warren et al, 2000), the available data in the literature with regard to the potentially negative effect of smoking on the periodontal tissues are contradictory. Similar (Preber and Kant, 1973; Müller et al, 2001) or higher (Preber et al, 1980; Haber et al, 1993; Linden and Mullally, 1994; Gunsolley et al, 1998; Hashim et al, 2001; Al-Wahadni and Linden 2003) prevalence of bleeding gingival sites was reported in young smokers compared with non-smokers, whereas in experimental gingivitis studies (Bergström et al, 1988; Danielsen et al, 1990; Lie et al, 1998) a reduced intensity of gingival vascular reactions and reduced gingival bleeding tendency in response to plaque accumulation was observed in smokers. Studies by Machuca et al (2000) and Hashim et al (2001) reported a detrimental impact of tobacco exposure on periodontal attachment levels in young individuals, while Lopez et al (2001) found no association between smoking habits and prevalence of attachment loss in a population sample of schoolchildren. Furthermore, gingival recession, which is

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associated with attachment loss, was shown by Gunsolley et al (1998) to be a more prevalent finding in smokers than in non-smokers, but data from a study by Müller et al (2002) did not support this finding. Results from studies evaluating the prevalence and severity of alveolar bone loss in young individuals are also contradictory. Al-Wahadni and Linden (2003) reported smoking to be significantly associated with a reduced ABL (alveolar bone level) at molars, whereas Preber et al (1980), in a study of young male army conscripts, found no differences between smokers and non-smokers in mean bone height at lower incisors.

Whether the amount of bacterial plaque accumulation may differ between smokers and non-smokers has also been addressed in a number of studies in young individuals. Although most studies reported higher prevalence scores of plaque in smokers compared with non-smokers (Preber et al, 1980; Gunsolley et al, 1998; Müller et al, 2001; Hashim et al, 2001; Al-Wahadni and Linden, 2003), some studies showed no differences (Preber and Kent, 1973; Linden and Mullally, 1994) or even lower plaque scores in smokers (Machuca et al, 2000). Furthermore, in experimental gingivitis studies, no difference in the rate of plaque accumulation between smokers and non-smokers was demonstrated (Bastiaan and Waite, 1978; Bergström and Preber, 1986; Bergström et al, 1988).

Most of the studies quoted have been performed on selected groups of young individuals, and data from randomly selected population samples of adolescents are lacking in the literature. The aim of this study was therefore to evaluate the influence of smoking on the periodontal conditions in a randomly selected population sample of 19-year-old individuals.

MATERIALS AND METHODS

The subject sample used in the present study originated from an epidemiological study of a computer-based random selection of 19-year-old individuals living in the community of Göteborg, Sweden (for details, see Abrahamsson et al, 2006). The population sample comprised 272 individuals who were clinically and radiographically examined. Information about the scope and aims of the project was given to all subjects and consent was obtained. Approval of the study protocol was obtained from the Ethics Committee at the Göteborg University. A questionnaire-based interview was used to collect information regarding oral hygiene and smoking habits. The subjects were classified according to their smoking habits as 'never-smokers', 'light smokers' (<10 cig/day) and 'heavy smokers' (≥10 cig/day). Potential use of smokeless tobacco was not considered. The persons who performed the clinical and radiographic assessments were kept unaware of the information regarding the subjects' smoking habits.

Clinical assessments

Two specially trained dental hygienists performed the clinical examinations. Besides the number of teeth (third molars excluded), the following variables were included in the clinical examination:

- Oral hygiene status: presence/absence of visible plaque after running a probe along the cervical part of the tooth was scored on four surfaces (mesial, buccal, distal and lingual) of six index teeth (Ramfjord, 1967).
- Gingivitis: presence of bleeding following probing of the sulcus area (Löe, 1967) was recorded at six sites (mesio-buccal, mid-buccal, disto-buccal, disto-lingual, mid-lingual and mesio-lingual) of all teeth.
- Probing pocket depth (PPD): assessed with the use of a manual periodontal probe (UNC 15; Hu-Friedy®, Chicago, IL, USA) to the nearest millimeter at six sites of each tooth.
- Probing attachment loss (PAL): PPD was assessed from the cemento-enamel junction (CEJ) at six sites of each tooth.
- Gingival recession: defined as location of the gingival margin apical to the CEJ and scored for facial tooth sites of all teeth.

Before the start of the study, the two examiners were trained to levels of accuracy and reproducibility for the various clinical parameters to be used. For both inter- and intra-examiner reproducibility, the standard deviation for probing measurements had to reach a level of <0.5 mm, with an agreement within ±1 mm of at least 99% of examined sites for PPD and 95% for PAL.

Radiographic assessments

Four bitewing radiographs (Ectaspeed Plus, Kodak Eastman, Rochester, USA) were taken of the premolar/molar regions using a standardised, parallel projection technique. On the radiographs, the ABL was assessed by measuring the distance in mm from the CEJ to the alveolar bone crest, i.e. the point at which the periodontal ligament space was considered to

have a normal width (Björn et al, 1969). The measurements were made by the use of a magnifying lens (7x) to the nearest lower 0.5 mm at all mesial/distal tooth surfaces reproduced in the bitewing radiographs. A site was considered 'non-readable' if the CEJ could not be defined. In addition, dental calculus was scored dichotomously as present/absent for each posterior jaw quadrant. A jaw quadrant was scored positive for calculus if at least two proximal tooth surfaces showed presence of radiographically detectable calculus. One examiner, blinded with respect to the purpose of the study, performed all radiographic assessments.

The intra-examiner reproducibility of ABL measurements was determined by repeated assessments of 10 randomly selected subjects (a total of 299 sites). Replicate pairs of measurements showed a mean difference of 0.04 mm (SD 0.11). Of the measurements, 96.4% were reproduced within a difference of ± 0.5 mm. The error of the method corresponded to 6% of the variance for the mean ABL in the population sample.

Data analysis

The highest value with respect to PPD, clinical attachment level and gingivitis at the buccal and lingual aspects of each approximal site was selected to represent the approximal site in the data description. Mean values and standard error (SE) were calculated based on the individual as the statistical unit.

The statistical analysis was based on a comparison between never-smokers and smokers and was performed by the use of the *t*-test for unpaired samples, or in case of abnormally distributed outcome variables, by the use of the Mann-Whitney U-test. In some of the descriptive analyses, the smokers were subgrouped according to the number of cigarettes smoked per day (<10 or ≥10 cig/day).

In addition, outcome variables were transformed into binary variables (0/1) for a logistic regression analysis with smoking habits as independent variable. The following binary variables were defined and tested: PPD ≥4 mm at >10 sites = 1, else 0; PPD ≥6 mm at ≥1site = 1, else 0; PAL ≥2 mm at ≥2 sites =1, else 0; facial gingival recession at >2 teeth = 1, else 0; and ABL >2 mm at ≥2 tooth sites = 1, else 0. All data analyses were performed with the use of the StatView 5.0.1 (SAS Institute Inc. 1998) software. A p-value of <0.05 was considered as statistically significant.

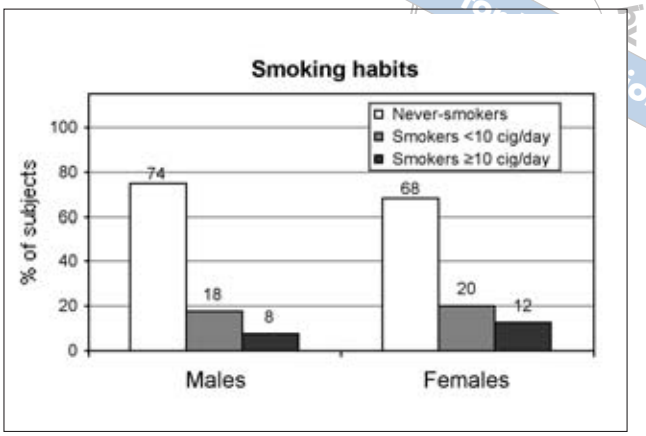

Fig 1 Percentage distribution of smoking categories among genders (n = 271).

RESULTS

Of the total 272 individuals examined (52% males and 48% females), one female never-smoker, who very recently had immigrated to Sweden and had never received any dental care in her life, was excluded from the data analysis since she could not be considered to be representative of the population under study (i.e. adolescents subjected to regular dental care). The distribution of the individuals with respect to the various smoking categories is depicted in Fig 1. Among the 271 adolescents, 78 (29%) were presently smokers, out of which 51 (65%) smoked <10 cig/day and had been smoking for an average of 3.4 years (SE 0.23, range 1–8 years), while 27 (35%) smoked ≥10 cig/day and had been smoking for a mean period of 5.2 years (0.35, range 2–9 years). No difference in mean number of teeth was found between smokers and never-smokers; 27.4 (S.E. 0.14) and 27.3 (0.12) teeth respectively.

Oral hygiene status

Table 1 and Fig 2 show the oral hygiene conditions of the adolescents. The mean percentage of tooth surfaces harbouring visible plaque was comparable for never-smokers and smokers; 58% (1.7) and 60% (2.2) respectively. Both groups revealed the highest proportions of plaque carrying tooth surfaces at proximal sites (78–82%). The analysis of the data with regard to the percentage of individuals displaying a good (plaque score <20%) and poor oral hygiene (plaque score ≥50%) revealed a somewhat higher proportion of individuals with poor oral hygiene among smokers compared with never-smokers, 74% versus 69% (Fig 2).

Table 1 Mean (SE) percentage of surfaces presenting visible plaque and signs of gingivitis (bleeding on probing) in never-smokers and smokers (unpaired *t*-test)

	Never-smokers	Smokers	p-value
Plaque (%)			
All surfaces	58.1 (1.67)	60.2 (2.21)	0.485
Buccal	25.4 (1.87)	21.8 (2.62)	0.394
Lingual	50.8 (2.30)	53.8 (3.34)	0.466
Proximal	77.9 (1.86)	82.4 (2.51)	0.272
Gingivitis (%)			
All surfaces	45.4 (1.35)	41.7 (2.07)	0.135
Buccal	17.7 (1.19)	14.8 (1.69)	0.197
Lingual	25.9 (1.48)	23.2 (1.99)	0.314
Proximal	69.0 (1.65)	64.4 (2.75)	0.144

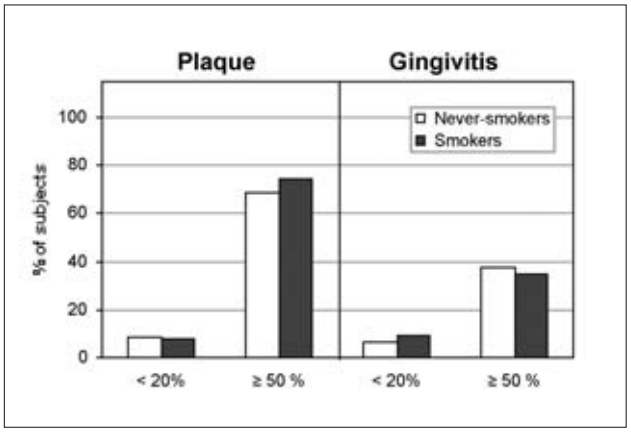

Fig 2 Proportion of subjects with < 20% and ≥ 50% in mean plaque and gingivitis scores, according to smoking habits.

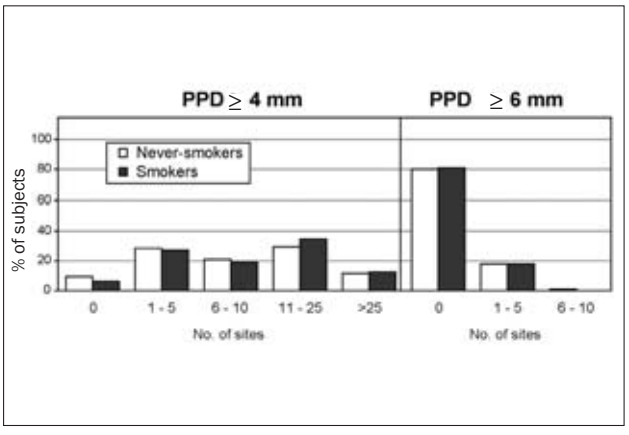

Fig 3 Percentage distribution of never-smokers and smokers according to number of sites with PPD ≥ 4 mm and ≥ 6 mm.

Table 2 Mean (SE) number of tooth sites with probing pocket depth (PPD) of ≥ 4 mm and ≥ 6 mm, probing attachment loss (PAL) of ≥ 2 mm and facial gingival recessions (unpaired *t*- test)

	Never-smokers	Smokers	p-value
PPD ≥ 4 mm (no. of sites)			
All surfaces	11.8 (0.89)	13.1 (1.43)	0.345
Proximal	11.4 (0.85)	12.5 (1.37)	0.370
PPD ≥ 6 mm (no. of sites)			
All surfaces	0.4 (0.08)	0.3 (0.07)	0.824
Proximal	0.4 (0.08)	0.3 (0.07)	0.807
PAL ≥ 2 mm (no. of sites)			
All surfaces	0.6 (0.14)	0.3 (0.07)	0.969
Proximal	0.2 (0.07)	0.1 (0.03)	0.925
Gingival recession (no. of facial sites)	0.8 (0.13)	0.6 (0.14)	0.873

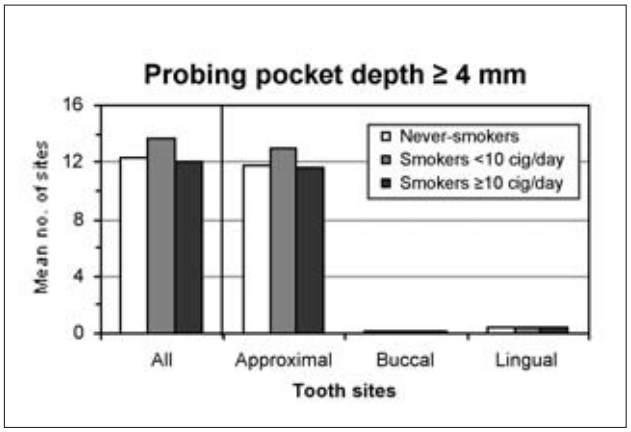

Fig 4 Individual mean number of sites with PPD ≥4 mm in each smoking category and according to tooth sites.

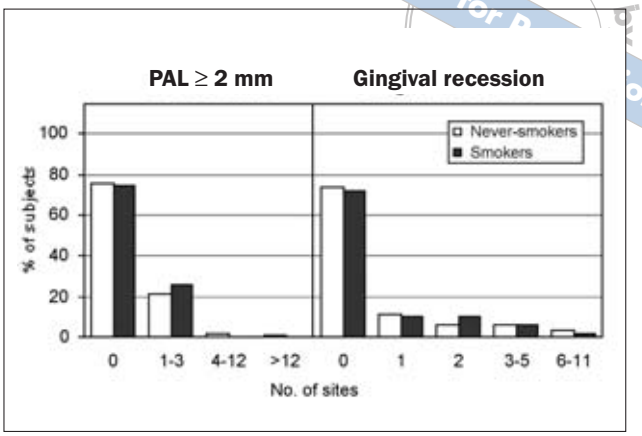

Fig 5 Percentage distribution of never-smokers and smokers according to number of sites with PAL ≥ 2 mm.

Gingivitis

The prevalence data for gingivitis are presented in Table 1 and Fig 2. The mean percentage of gingival sites showing bleeding following superficial probing was in never-smokers 45% (1.4) compared with 42% (2.1) in smokers. At proximal sites, the corresponding figure was 69% (1.7) and 64% (2.8) respectively. Of the never-smokers, 73 (38%), and 27 (35%) of the smokers showed a prevalence of gingivitis of ≥ 50% (Fig 2), while only 7% (never-smokers) and 9% (smokers) displayed comparatively healthy gingival conditions (< 20% with gingivitis). No statistically significant differences were found between the two groups.

Probing pocket depth

The mean number of sites with PPD ≥4 mm and ≥6 mm, respectively, is presented in Table 2. The average number of sites with PPD ≥ 4 mm was 12 (0.9) in never-smokers and 13 (1.4) in smokers, and the pockets were predominantly located at proximal tooth surfaces. About 50% of the smokers presented with more than 10 pockets with a depth of ≥ 4 mm, compared with 43% of the never-smokers (Fig 3). No tendency for increased numbers of deepened periodontal pockets was observed with increased cigarette consumption (Fig 4). Independent of smoking habits, the number of sites with PPD ≥ 6 mm was very low and there was no significant difference between the groups (Table 2 and Fig 3). Approximately 80% of the individuals in the group of never-smokers as well as in the group of smokers had no pockets with PPD ≥ 6 mm.

Probing attachment loss and gingival recession

The never-smokers showed a mean PAL of 0.14 mm (0.01), compared with 0.09 (0.01) for the smokers (Mann-Whitney U test p = 0.0037). On average, never-smokers had 0.6 (0.14) sites with a PAL of ≥ 2 mm, whereas the corresponding figure in smokers was 0.3 (0.07) (Table 2). The majority of sites showing PAL were located at facial tooth sites and associated with gingival recession, the prevalence of which did not significantly differ with respect to smoking habits (Table 2 and Fig 6). Three-quarters of both never-smokers and smokers did not have any site with PAL ≥ 2 mm, and only 6 individuals (3%), all never-smokers, showed more than three sites with a PAL of ≥ 2 mm (Fig 5). There was no significant difference in individual mean number of gingival recessions (0.6–0.8; Table 2) between smokers and never-smokers. Neither were any significant differences observed with respect to PAL or gingival recessions between the sub-groups of heavy smokers and never-smokers.

Alveolar bone level and radiographically detectable calculus

The average number of readable tooth sites per subject with respect to ABL was 27 (84%). In both never-smokers and smokers the mean ABL amounted to 1.3 mm (Table 3). Approximately 70% of the never-smokers and 80% of the smokers did not show any sites with an ABL >2 mm (Fig 7). More than five sites with an ABL >2 mm was only found in 4% of the never-smokers and in 1% of the smokers.

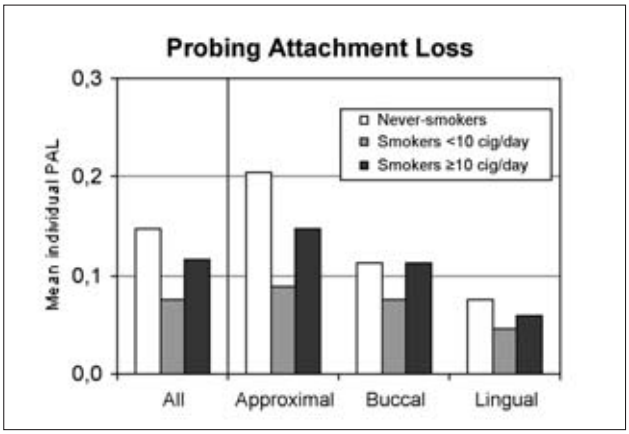

Fig 6 Mean probing attachment loss in each smoking category and according to tooth sites.

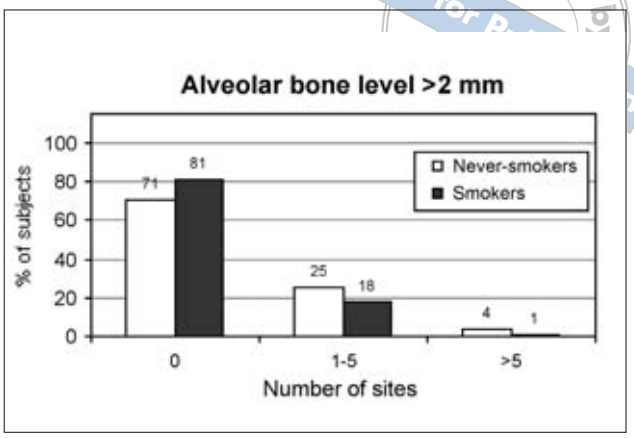

Fig 7 Percentage distribution of never-smokers and smokers according to number of tooth sites displaying a radiographic alveolar bone level (ABL) of >2 mm.

Radiographically detectable calculus was found in seven subjects (3%); four never-smokers and three smokers. Two individuals presented calculus both in the maxilla and the mandible (never-smokers), two only in the maxilla (smokers) and three only in the mandible (two never-smokers and one smoker).

Logistic regression analysis

The results of the logistic regression analysis are presented in Table 4. The independent variable 'smoking habits' did not reach statistical significance for any of the defined criteria of periodontal destruction. The odds ratios varied between 0.62 and 1.33, indicating negligible impact of smoking on the periodontal conditions. In fact, the data indicated a lower rather than a higher risk, although not statistically significant, of finding a subject with sites showing PAL of ≥ 2 mm or ABL of >2 mm in smokers compared with never-smokers (OR 0.62–0.69).

DISCUSSION

The results of the present study of 19-year-old individuals revealed no significant differences in periodontal conditions between smokers and never-smokers, either with respect to oral hygiene, gingivitis, deepened periodontal pockets, PAL, gingival recession or ABL.

A number of previous studies have addressed the question regarding potential effects of tobacco smoking on the periodontal conditions in young individuals. Preber and Kant (1973) examined 15-year-old schoolchildren and reported no differences in gingival status and ABL between smokers and non-smokers with comparable oral hygiene standards. In a subsequent study (Preber et al, 1980), involving 134 male army conscripts with an average age of 22 years (range 19–27), the authors found significantly higher prevalence of plaque and gingivitis in smokers compared with non-smokers, but no differences with regard to PPD or ABL at lower incisors. A lack of association between smoking and clinical attachment loss was also shown in a study of a sample of schoolchildren aged 12–21 in Chile (Lopez et al, 2001), in which 25% of the subjects were smokers with an average daily consumption of 5.4 cigarettes for a mean of 3 years. In contrast, Machuca et al (2000) reported a significant impact of smoking habits on the periodontal support from a study involving army recruits (mean age 19.4 years). In that study, smokers (5–20 cig/day for <5 years) presented significantly lower prevalence of plaque and gingival bleeding but significantly higher mean PPD and attachment loss than non-smokers. The latter observation is in accordance with results from a study of a population sample of 26-year-old individuals in New Zealand (Hashim et al, 2001) showing that cigarette smoking was a significant predictor for loss of attachment. However, in contrast to the previous study, smokers showed also higher prevalence of plaque and gingival bleeding than non-smokers. In a case-control study, Al-Wahadni and Linden (2003) reported a significant impact of smoking on alveolar bone loss in molars (OR 4.95, 95%CI 2.48–9.88) in a patient sample of the age 20–35 years at a dental hospital clinic. The smokers in this study (average 25 cig/day for 5.5 years) presented, compared with non-smokers, a higher prevalence of plaque (92% versus 70%) and gingivitis (52% versus 25%) as well as higher mean

Table 3 Individual mean alveolar bone level (ABL) and number of proximal sites with an ABL of >2 mm. Mean (SE) and unpaired *t*-test

	Never-smokers	Smokers	p-value
ABL	1.3 (0.18)	1.3 (0.02)	0.797
No. of sites with ABL >2mm	0.9 (0.15)	0.5 (0.14)	0.178

Table 4 Results of logistic regression analyses with smoking as independent variable

Dependent variables	Coefficient	SE	p-value	OR	95% CI
PPD ≥4 mm	0.286	0.270	0.290	1.33	0.78–2.26
PPD ≥6 mm	-0.081	0.347	0.815	0.92	0.47–1.82
PAL ≥2 mm	-0.485	0.479	0.312	0.62	0.24–1.58
Gingival recession	0.173	0.356	0.627	1.29	0.59–2.39
ABL >2 mm	-0.374	0.388	0.334	0.69	0.32–1.47

PPD (2.5 mm versus 1.9 mm) and mean bone level (2.2 mm versus 1.6 mm). Taken together, the data presented in the aforementioned studies are inconsistent with regard to a potential association between smoking and impaired periodontal health in young individuals. One explanation for this could be that the majority of the studies used selected or convenient subject samples with a wide range in age instead of a randomised sample of the target population. Furthermore, the magnitude of exposure to tobacco smoking, both in terms of amount and years, varied between the studies.

The data analysed in present study were generated from an epidemiological survey of 19-year-old individuals under regular dental care, randomly selected among those living in one of the major cities in Sweden. Since the dental care is provided by the Dental Public Health service and free of charge for all children and adolescents until the age of 20 years, the 19-year age group was selected in order to minimise the potential influence of variation in dental care on outcome variables, while at the same time maximising the potential exposure to tobacco smoking. The sample generated had an even distribution with respect to gender and a proportion of smokers of 29%, with a mean consumption of 7 cig/day and a mean smoking duration of 4.1 years. Furthermore, as indicated by the plaque score data (Table 1), there was no significant difference between smokers and never-smokers with regard to the oral hygiene standard, although this was poorer than that reported in other cross-sectional studies performed in Sweden on population samples of individuals of a similar age (Hugoson and Rylander, 1982; Hugoson et al, 1998). Although the prevalence of smokers in the present study (29%) was higher than that of the comparative studies, one has to consider the potential risk of misclassification bias of the subjects since the information on smoking habits was self-reported. By assessing cotinine levels in self-reported non-smokers, Wells et al (1998) calculated the misclassification bias to be 0.8% and 6.0% for female regular and occasional smokers, respectively, and somewhat higher for males.

The descriptive statistics revealed similar periodontal conditions for smokers and never-smokers in the present study, considering the proportion of bleeding gingival sites and the number of sites with deepened pockets, PAL of ≥2 mm or ABL of >2 mm. In fact, the odds ratios for smoking were low and non-significant for all the criteria of periodontal destruction (OR 0.62–1.33; Table 4), indicating negligible impact of smoking on the periodontal conditions of the adolescents. These findings support observations made by Lopez et al (2001) in young subjects with comparable duration and amount of smoking exposure, but differ from those of studies involving somewhat older subjects with longer and heavier experience of smoking (Hashim et al, 2001; Al-Wahadni and Linden, 2003).

It should be noted that in the present study the majority of the sites showing loss of attachment were associated with facial gingival recessions, and independent of self-reported smoking habits. Gunsolley et al (1998) suggested, based on observations made in a clinical study, that gingival recession is a more prevalent finding in smokers than in non-smokers. However,

findings reported in a study involving heavy smokers (Müller et al, 2002), as well as the results of the present study, did not support the hypothesis that young smokers are at greater risk for the development of gingival recession.

In summary, in the present population sample of adolescents, characterised by high plaque scores, smoking habits did not contribute to a higher prevalence of signs of periodontal destruction, i.e. deepened PPD, attachment loss, gingival recession or ABL. It may be necessary to have longer and heavier exposure to smoking, and consequently older individuals than in the present study, in order to be able to detect detrimental effects of smoking on the periodontal tissues. In fact, Bergström et al (2000) suggested, based on data obtained from a cross-sectional study of 257 dentally aware adults, that a rather long incubation time is required until symptoms of disease become clinically or radiographically apparent to such an extent that the effects of smoking can be discerned. Hence the authors reported that among individuals aged below 40 years and with a smoking duration of approximately 10 years, no major differences in periodontal conditions were observed in comparison with non-smokers, whereas between 40 and 69 years of age, with an average exposure time of 30 years, the negative effects of smoking were marked.

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