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A prospective 12-month study of the effect of smoking cessation on periodontal clinical parameters

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Abstract

Aim: The aim of this 12-month prospective study was to assess the adjunctive effect of smoking cessation in non-surgical periodontal therapy of subjects with severe chronic periodontitis.

Materials and methods: Of the 201 subjects enrolled from a smoking cessation clinic, 93 were eligible and received non-surgical periodontal treatment and concurrent smoking cessation treatment. Periodontal maintenance was performed every 3 months. Full-mouth periodontal examination in six sites per tooth was performed by a calibrated examiner, blinded to smoking status, at baseline, 3, 6 and 12 months after non-surgical periodontal treatment. Furthermore, expired air carbon monoxide concentration measurements and interviews based on a structured questionnaire were performed in order to collect demographic and smoking data.

Results: Of the 93 eligible subjects, 52 remained in the study after 1 year. Of these, 17 quit smoking and 35 continued smoking or oscillated. After 1 year, only quitters presented significant clinical attachment gain ($p = 0.04$). However, there were no differences between the groups regarding clinical attachment level, probing depth, bleeding on probing and plaque index after 1 year ($p > 0.05$).

Conclusion: Smoking cessation promoted clinical attachment gain in chronic periodontitis subjects from a smoking cessation clinic after 1 year of follow-up.

Key words: periodontal disease; periodontitis; smoking; smoking cessation; tobacco

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Tobacco use has been one of the public health biggest threats, being considered an epidemic by the World Health Organization since 1990 (WHO 2009). Furthermore, it is estimated that tobacco use kills more than five million people per year, which accounts for every one in 10 adult deaths worldwide. Smoking is a common risk factor for a number of chronic diseases, including cancer, lung diseases and cardiovascular diseases (WHO 2010).

The oral effects of smoking have been studied extensively during the past several years and among several risk factors investigated for periodontitis, the epidemiological evidence for smoking is strong (Hujoel 2003). In summary, cross-sectional studies (Tomar & Asma 2000, Albandar 2002, Susin et al. 2004) have shown that smokers are from two to seven times more likely to present periodontitis as compared with non-smokers. Also, smoking has been associated with tooth loss during periodontal maintenance (Chambrone et al. 2010). Moreover, a few longitudinal studies (Krall et al. 1997, Bergstrom et al. 2000) suggest that quitting smoking represents a lower risk of presenting periodontal conditions and tooth loss. In southern Brazil (Susin et al. 2004), it was estimated that smoking cessation programs could result in an approximate reduction of up to 12% in the number of destructive periodontal disease cases in this population. These statements stress the extreme importance of the implementation of population-based smoking cessation programs both to increase general health (common risk factor approach) (Sheiham & Watt

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2000) and to reduce the prevalence of severe attachment loss in populations with high levels of smoking exposure.

On the other hand, when it comes to the clinical setting, the literature regarding the effects of periodontal therapy seems to be unanimous in showing that smokers have a poorer response to treatment, whether surgical or non-surgical, compared with non-smokers (Kaldahl et al. 1996, Labriola et al. 2005, Wan et al. 2009). These observations have led several authors to conclude that interventions aimed at smoking cessation would result in improvements regarding clinical periodontal parameters.

However, in contrast to the huge concern reported in the literature about all general and oral harms of tobacco smoking and the importance of smoking cessation programs (Marsh & Devine 2011), there is scarce information from prospective interventional studies investigating the benefit of quitting smoking on periodontal conditions.

The only 12-month follow-up study addressing the question (Preshaw et al. 2005) was performed with 49 smokers presenting chronic periodontitis intending to quit smoking. All participants received non-surgical periodontal treatment and smoking cessation therapy according to their individual needs. When only sites with probing depth (PD) $\geqslant$3 mm were considered, participants who successfully stopped smoking had a higher mean PD reduction than the other two groups combined (non-quitters and oscillators). The authors concluded that smoking cessation promoted an additional benefit at reducing PD after non-surgical periodontal treatment.

For the potential benefit of smoking cessation in periodontal treatment to be properly evaluated, it is of paramount importance that more research in this field is conducted. Therefore, the aim of this prospective 12-month follow-up study was to assess the adjunctive effect of smoking cessation in non-surgical therapy of smoking subjects with severe chronic periodontitis.

Material and Methods

Study design and population

This is a 12-month prospective, single-blinded study of the effect of non-surgical periodontal and smoking cessation therapy in smokers.

Participants were recruited for a period of 2 years (May 2007–2009). To be enrolled in the study, they should both be willing to stop smoking and seek also to enroll the service offered at the Smoking Cessation Clinic at the University Hospital (UH) in São Paulo, which is located in southeastern Brazil. UH serves local residents living in the Butantã area of São Paulo. This area consists of seven neighbourhoods comprising inhabitants who present a wide range of cultural and socioeconomic heterogeneity (Instituto Brasileiro de Geografia e Estatística 2000).

Subjects enrolled at the Smoking Cessation Clinic received a multi-disciplinary therapy and were given lectures by one of the investigators (G. I.) at the UH Smoking Cessation Clinic. These lectures were aimed at describing the harms of smoking on oral health and introducing the aims of the study. Following the lecture, subjects were invited to participate in the screening examination. Subjects were eligible to the study and invited to participate if they were smokers, $\geqslant$18 years old, willing to stop smoking, presented at least 10 teeth in the oral cavity and destructive periodontal disease, defined as 30% or more of their teeth with proximal clinical attachment level (CAL) $\geqslant$5 mm (Tonetti & Claffey 2005); and they were not eligible if they presented: (1) any systemic condition considered as a risk factor for periodontal disease (e.g. diabetes, HIV infection), (2) undergone periodontal therapy in the last 6 months or (3) continuous systemic use of steroidal or non-steroidal anti-inflammatory drugs. This study was approved by the School of Dentistry of University of São Paulo Ethics Committee (protocol 29/07). All subjects were given information about the study and provided written informed consent before the start of the study.

Interview and clinical examinations

After inclusion, subjects were interviewed and received oral clinical examination at baseline, 3, 6 and 12 months after non-surgical periodontal treatment. Examinations and periodontal treatment were carried out at the post-graduate clinic of the Periodontology Department, School of Dentistry, University of São Paulo.

A single-trained and calibrated examiner (P. C.) interviewed all enrolled subjects using a structured written questionnaire. The baseline interview questions comprised information about demographic data such as age (in years); sex, marital and socioeconomic status; oral and general health data; oral hygiene habits; and smoking-related data. Smoking-related data were collected at baseline, 3, 6 and 12 months of follow-up. At baseline, the questions pertaining to smoking habits included current smoking status (yes/no), the duration of current smoking (in years), the type of tobacco-containing items used, the number of items smoked on a daily basis, as well as the number of previous attempts to quit smoking and which kind of attempts were done. Pack/years were calculated by the following formula: number of packs smoked per day $\times$ smoking years. In all the remaining follow-up sessions, subjects were further interviewed with questions related to their smoking habits in the last 3 months, whether or not they succeeded at quitting smoking; and, if not, their reasons, the number of cigarettes smoked per day, and if they had noticed any changes in their general health since then.

If positive at the follow-up clinical examinations, questions related to reasons for tooth loss in the previous 3 months were also performed.

Clinical examinations

After the interview, subjects had their expired air carbon monoxide (CO) concentrations measured with the help of a CO monitor (Micromedical Ltd., Kent, UK) by the same examiner that performed all the interviews (P. C.). Measurements of exhaled CO concentrations were performed in order to correlate the smoking status interview data with clinical assessment of smoking status. The cut-off point of 10 ppm was considered to distinguish smokers from non-smokers (Subcommittee on Biochemical Verification 2002).

Following that procedure, the clinical dental status and a full-mouth periodontal examination of all present teeth, excluding third molars, were performed by a single-trained and calibrated examiner (E. F. R.), blinded for smoking status, assisted by a recorder. The following periodontal clinical parameters were assessed at six sites per tooth (mesio-buccal, buccal, disto-buccal, mesio-lingual, lingual and disto-lingual), using a manual probe (Trinity®, São Paulo, Brazil): PD, gingival recession (GR) and bleeding on probing (BoP), recorded as present if bleeding occurred within 15 s after the assessment of PD. CAL was obtained as the

sum of GR and PD measurements for each site. The measurements were rounded to the lowest whole millimetre. Two sites per tooth (mid-buccal and mid-lingual) were assessed for the presence of visible plaque (VP) (yes/no). Excessive amounts of supragingival calculus compromising assessment of the periodontal conditions were removed with periodontal curettes (Gracey 5–6, Trinity®) before probing.

Presence (yes/no) of tooth decay, multiple or single bridges or restorations nearby or below the free gingival margin, or presenting defective margins were also assessed with the help of an explorer (Duflex, Rio de Janeiro, Brazil).

Blinding

The examiner was blind to smoking status. Before each follow-up periodontal examination (3, 6 and 12 months), the following procedures were performed by a periodontist: (1) removal of all tobacco stains from subject's teeth and/or dental polishing, with a standardized duration of up to 30 min., (2) 0.12% chlorohexidine rinse (1 min.) aimed to hide any possible cigarette odour exhaled from the oral cavity. After these procedures, the examiner entered the examination room fully equipped (including a mask), in order to avoid noticing the smoking status by odour.

Measurement reproducibility

The interview questions were tested before the study through a pre-test conducted on seven smokers and former smokers ($\sim$ 10% of the planned sampling size) who attended the graduate clinic of the Periodontology Department, School of Dentistry, University of São Paulo. After pre-test, one question was eliminated, two questions were included and three were modified for a better understanding.

Before baseline and 1 year after the start of the study, intra-examiner reproducibility for GR and PD was assessed by double recordings on randomly chosen quadrants in eight subjects. Two examinations, with 7 days of interval, were conducted in these subjects, and the agreement between them was calculated with the intra-class correlation (ICC) coefficient. The ICC coefficient ranged from 0.85 [confidence intervals (CI) 95% 0.81–0.88] to 0.87 (CI 95% 0.82–0.91) for PD measurements and 0.79 (CI 95% 0.71–0.85) to 0.87 (CI 95% 0.84–0.90) for mean GR.

Smoking cessation therapy

The smoking cessation therapy was performed at the Smoking Cessation Ambulatory Clinic at UH. It consisted of four consecutive lectures, one per week with a mean duration of 1 h each, provided by a multi-disciplinary team comprising doctors, psychologists and a dentist, and was intended to counsel subjects about the harms of smoking and benefits of quitting the habit. Furthermore, they also received a psychologist-assisted cognitive behavioural therapy (Webb et al. 2010). According to their individual needs, nicotine replacement therapy (Wu et al. 2006) and/or bupropion (Hughes et al. 2007) were also employed as adjunctive therapy.

Further, smoking cessation counselling and support were also provided by the interviewer at baseline, 3, 6 and 12 months of follow-up after the performance of smoking status questions. Counselling during these interviews was based on motivational interviewing techniques (Ramseier et al. 2010).

Non-surgical periodontal therapy

After the initial examination, all subjects received non-surgical periodontal therapy by two periodontists (V. F. C. and E. F. G.). Periodontal treatment consisted of supra and subgingival scaling of all teeth using manual (curettes, Trinity®) and ultrasonic instruments (MiniPiezon®, EMS, Nyon, Switzerland) (Cobb 2002), oral hygiene instructions (OHI), and removal of all intra-oral biofilm retentive factors. After the active phase of periodontal treatment, which was held for four and six sessions with a 7-day interval between each one, the subjects entered into a maintenance programme of 3-month intervals. Patients reporting hypersensitive teeth received fluoride varnish (Duraphat, Colgate, São Paulo, Brazil) applications in a routine follow-up basis on dentin-exposed surfaces.

Data analyses

All interview and clinical written data were converted into an electronic form by means of data entry software (Epidata 3.1, Odense, Denmark). Statistical analysis was performed using Stata 10.1 (Stata version 10.1 for Windows, Stata Corporation, College Station, TX, USA).

Mean values, standard deviations, CI and frequency distributions are given for CAL, PD, percentage sites with VP and BoP for quitters, non-quitters and oscillators (subjects who stopped smoking more than once during the study or started smoked again) and for the last two groups combined.

The main purpose of the statistical analysis was to compare subjects who stopped smoking among those who did not stop (non-quitters and oscillators together). The two groups were compared with baseline, 3, 6 and 12 months regarding overall means of percentage sites with VP, BoP, PD and CAL using repeated ANOVA measures. Multiple comparisons were conducted with the post hoc Newman–Keuls test. Furthermore, differences between groups at baseline were assessed by means of Student's *t*-test. The groups were compared with respect to categorical variables using the χ^2-test. A significance level of $\alpha = 5\%$ was used in all statistical tests. Statistical analyses were applied for CAL and PD only for missing data at 3 and/or 6 months for subjects with 1 year of follow-up. Data were imputed in Stata through the last value carried forward method (Twisk & de Vente 2002).

Moreover, to analyse whether the intervention had a clinically significant effect, i.e. reached treatment success at 12 months, the data were presented in several success criteria outcomes for CAL and PD, which included the following: differences in overall mean and exclusively in sites presenting baseline PD $\geqslant$ 4 mm; overall mean CAL gain and PD reduction and exclusively in sites presenting baseline PD $\geqslant$ 4 mm; percentage of sites per subject presenting CAL and PD $\geqslant$ 4 mm and 4–6 mm; and prevalence and extent (absolute number and percentage of sites per subject) of CAL gain or loss and PD reduction or increase of 0, 1 and 2 mm. In all instances, the subject was the statistical unit of the analysis.

Results

Between May 2007 and 2009, 877 subjects applied for the UH Smoking Cessation Clinic. Out of them, 201 were screened and 93 met the eligibility criteria and were included in the study. They received non-surgical periodontal

treatment and concurrent smoking cessation therapy. Forty-one subjects were lost to follow-up, 22 until the third month, 8 up to the sixth month and 11 up to 1 year (Fig. 1).

Of the 52 subjects who remained in the study after 1 year, 42.31%, 32.69% and 32.69% were not smoking at 3, 6 and 12 months, respectively. From the remaining 67.3% smokers at 12 months of follow-up, 50% (26) failed to quit smoking and 17.3% (nine) oscillated. None of the participants who reported having quit smoking showed a CO expired value greater than 6 ppm. Regarding the subjects, which successfully stopped smoking after 12 months, 20% were not users of any nicotine replacement, 33.3% used nicotine patches and gums daily for up to 2 months, 33.3% up to 6 months and 13.3% over 6 months.

Table 1 describes the demographical, behavioural and clinical baseline characteristics of the subjects who quit [(quitters (Q)], $n = 17$; continued smoking or oscillated [(non-quitters (NQ)], $n = 35$; and those also lost to follow-up, $n = 41$. There was no significant difference between Q and NQ regarding all baseline characteristics ($p > 0.05$), except for baseline levels of CO ($p = 0.03$). When subjects who completed the entire sequence of events comprising the study were compared with those lost at follow-up, it was observed that the former presented higher age ($p = 0.03$) and lower PD, BoP ($p < 0.01$) and CO ($p = 0.03$).

Subjects did not present significant difference in the number of present teeth during the 1 year period (20.6 ± 4.8 *versus* 20.2 ± 5.1 at follow-up), and although non-quitters have lost more teeth than quitters (0.51 ± 1.0 *versus* 0.12 ± 0.6) at follow-up, the difference was non-significant ($p = 0.15$).

Both groups (Q and NQ) revealed a significant reduction in the percentage sites presenting VP ($p < 0.05$) after 1 year, although no difference between groups was detected at any time during the study. As regards BoP, there was no significant change over time for both groups, and no difference between groups could be observed (Table 2). A significant CAL gain of 0.21 mm was observed in individuals who quit smoking ($p = 0.04$) *versus* and a non-significant gain of 0.13 mm in the NQ group. Q and NQ groups presented a significant PD reduction of 0.29 mm ($p = 0.002$) and 0.30 mm ($p = 0.007$), respectively.

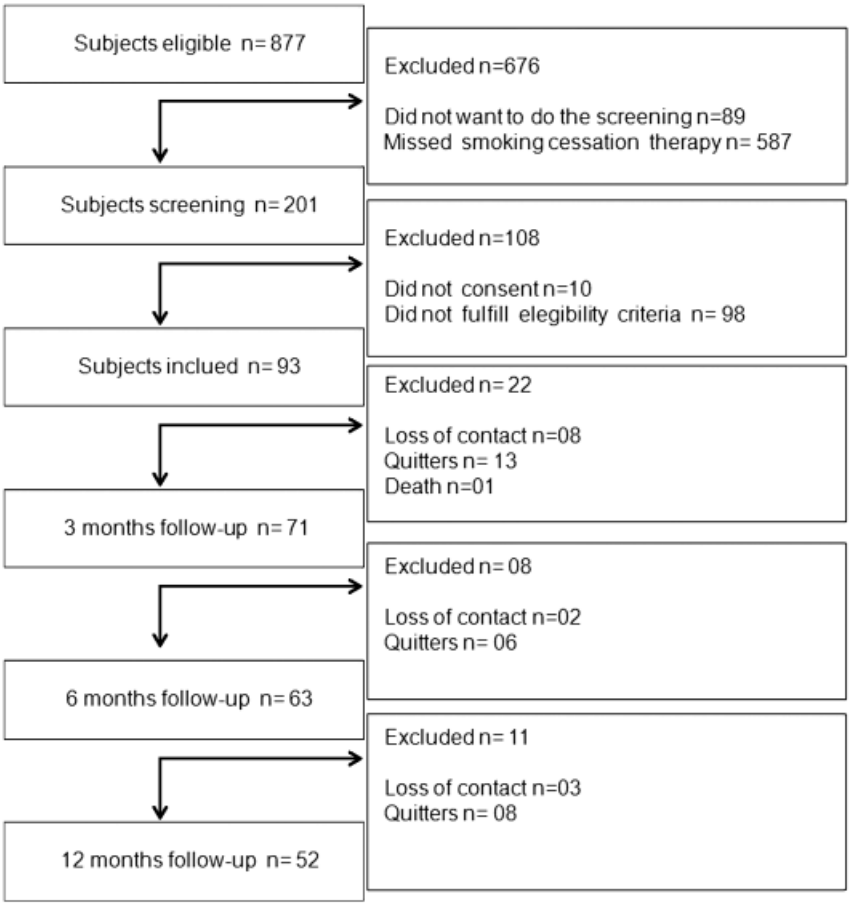

Fig. 1. Flow chart of participation in the study.

However, there was no significant difference between groups at any time during the study by means of repeated measures ANOVA for CAL and PD (Table 2). When only sites presenting baseline PD $\geqslant$ 4 mm were analysed, both groups presented significant PD reduction ($p < 0.001$), but there was no difference between groups at any time of the study. Quitters presented a significant 1.32 mm *versus* a significant 0.85 mm CAL gain for non-quitters ($p < 0.001$). Although there was no difference between groups as regards CAL, there was a significant difference favouring the Q group regarding the magnitude of CAL gain (1.32 mm gain for Q and 0.85 mm gain for NQ, $p = 0.02$) (Table 2).

Figure 2 shows the mean distribution of the CAL and PD reduction over time for quitters, non-quitters and oscillators. Quitters presented a trend for a greater initial PD and CAL reduction at 3 months when compared with the other two groups, followed by a slight decrease in the reduction after 6 months. This pattern was similar to the one found by the oscillator group. However, the NQ group presented different trend pictures for CAL and PD. For CAL, they were featured by initial attachment loss at 3 months, followed by CAL reduction at 6 and 12 months. That reduction was inferior to the one presented by the Q group at 12 months. As regards to PD, a smaller initial reduction was observed at 3 months, reaching to a similar reduction to the Q group at 12 months.

Table 3, describes the prevalence (percentage of subjects) and extent (percentage of sites per subject) of CAL and PD $\geqslant$ 4 mm and 4–6 mm, respectively. Both groups presented similar extent and prevalence of CAL at baseline and after 12 months. For CAL $\geqslant$ 4 mm there was a significant reduction on the extent of sites for both groups. Regarding PD measurements, no differences in prevalence could be observed in the groups at baseline and after 1 year. However, quitters presented significant lower extent than NQ at baseline for PD $\geqslant$ 4 mm ($p = 0.04$) and 4–6 mm ($p = 0.04$). After 12 months, there was a significant reduction on the extent for both groups for PD $\geqslant$ 4 mm and 4–6 mm.

Table 1. Baseline demographical, behavioural and clinical characteristics of the subjects who quit ($N = 17$) or not (non-smokers and oscillators combined) ($N = 35$) smoking after 1 year follow-up, and comparison among those lost at follow-up ($N = 41$)

Parameters	Quitters ($N = 17$)	Non-quitters ($N = 35$)	p value	Total ($N = 52$)	Lost follow-up ($N = 41$)	p value
Mean (SD) age (years)	48.9 (6.1)	49.5 (7.0)	0.79	49.3 (6.7)	45.8 (8.7)*	0.03†
Gender (%) male	7 (41.2%)	13 (37.1%)	0.78	20 (38.5%)	11 (27.6%)	0.27
Education, N (%) subjects $\leqslant 10$ years education	9 (52.9%)	14 (42.4%)	0.48	23 (46%)	21 (51.9%)	0.57
Income, N (%) monthly income $\leqslant 1000$ BZR	7 (43.8%)*	14 (46.7%)‡	0.85	21 (45.7%)§	15 (41.7%)‡	0.72
Smoking, Mean N pack/years	35 (18.5)	42.2 (64.9)	0.66	39.8 (54.1)	35.1 (22.6)	0.60
Mean (SD) exhaled CO reading (ppm)	14.94 (10.39)	23.20 (13.48)	0.03†	15.81 (13.19)	21.53 (15.9)	0.03†
Mean (SD) number of teeth present	20.5 (4.5)	20.6 (5.0)	0.91	20.6 (4.8)	18.8 (4.8)	0.08
Daily brushing frequency, N (%) subjects $\geqslant 2$	16 (94.1%)	35 (100%)	0.15	51 (98.1%)	36 (89.3%)	0.07
Use of inter-dental devices, N (%)	8 (47.1%)	18 (51.4%)	0.77	26 (50.0%)	16 (39.3)	0.30
Mean (SD) % sites visible plaque	84.3% (23.7)	81.1 (19.8)*	0.61	82.2 (21.0)*	83.0 (26.0)*	0.87
Mean (SD) CAL (mm)	3.74 (0.68)	4.19 (1.39)	0.21	4.01 (1.19)	4.06 (0.95)	0.83
Mean (SD) PD (mm)	2.76 (0.51)	3.07 (0.74)	0.12	2.92 (0.65)	3.34 (0.67)	<0.01†
Mean (SD) % sites BoP	25.6% (17.6)	19.8 (13.8)	0.20	21.7% (15.3)	31.5 (20.0)	<0.01†

*One missing value.
†Significant at $\alpha = 5\%$.
‡Five missing values.
§Six missing values.
SD, standard deviation; CO, carbon monoxide; CAL, clinical attachment level; PD, probing depth.

Table 2. Repeated measurement ANOVA for mean values of percentage of plaque, percentage of BOP, PD and CAL (mm) and for sites presenting PD $\geqslant 4$ mm at baseline

Outcome	Group (n)	Mean (SD) scores				Baseline × 12 months
		baseline	3 months	6 months	12 months	p*
All sites						
% sites	Q (17)	85.08 (20.86)	61.11 (32.58)†	62.06 (26.68)†	58.62 (28.97)†	<0.001†
Visible plaque	NQ (35)	81.62 (19.66)	66.49 (29.21)†	64.59 (27.97)†	72.09 (20.88)†	0.04†
	P*	0.65	0.89	0.74	0.48	–
% BOP	Q (17)	25.61 (17.62)	19.57 (11.09)	24.05 (14.54)	20.41 (11.63)	0.16
	NQ (35)	19.75 (13.83)	17.98 (11.40)	20.93 (13.96)	20.04 (13.15)	0.90
	P*	0.67	0.68	0.43	0.92	–
CAL	Q (17)	3.73 (0.68)	3.42 (0.70)	3.49 (0.69)	3.52 (0.55)†	0.04†
	NQ (35)	4.18 (1.39)	4.23 (1.53)	4.18 (1.46)	4.05 (1.21)	0.41
	p*	0.59	0.34	0.31	0.31	–
PD	Q (17)	2.76 (0.50)	2.39 (0.33)†	2.45 (0.42)†	2.47 (0.34)†	0.002†
	NQ (35)	3.07(0.73)	2.89 (0.69)	2.85(0.67)	2.77 (0.65)†	0.007†
	p*	0.43	0.09	0.18	0.23	
Sites presenting baseline PD $\geqslant 4$ mm						
CAL	Q (17)	5.71 (1.28)	4.59 (1.45)†	4.37 (1.35)†	4.39 (1.18)†	<0.001†
	NQ (35)	5.76 (1.21)	5.31 (1.44)†	5.20 (1.47)†	4.91 (1.39)†	<0.001†
	p*	0.89	0.29	0.25	0.39	–
PD	Q (17)	4.66 (0.49)	3.54 (0.85)†	3.41 (0.82)†	3.42 (0.73)†	<0.001†
	NQ (35)	4.72 (0.50)	3.96 (0.58)†	3.86 (0.68)†	3.63 (0.76)†	<0.001†
	p*	0.75	0.16	0.16	0.52	

*Newman–Keuls test.
†Significant difference in relation to baseline ($p < 0.05$).
BOP, bleeding on probing; CAL, clinical attachment level; PD, probing depth; Q, quitters; NQ, non-quitters.

In Table 4, the sites were analysed regarding the prevalence (percentage of subjects) and extent (percentage of sites per subject), for CAL gain/loss and PD reduction/increase in five categories: -2 (attachment loss of 2 mm or increase of 2 mm PD), -1(attachment loss of 1 mm or increase of 1 mm PD), no changed in CAL and PD, 2 (2 mm attachment gain or decrease of 2 mm PD) and 1 (1 mm attachment gain or decrease of 1 mm PD). The Q group presented a numerically smaller extent of sites per subject presenting CAL loss and PD increase after treatment. However, no significant difference was observed between the two groups. As regards oscillators, the group presented a similar distribution pattern when compared with Q (Fig. 3).

Discussion

The aim of this study was to assess the effect of smoking cessation on the clinical periodontal parameters after non-surgical treatment of chronic periodontitis. So far, only one interventional study addressing this issue (Preshaw et al. 2005) was found in the literature. In summary, periodontal treatment was

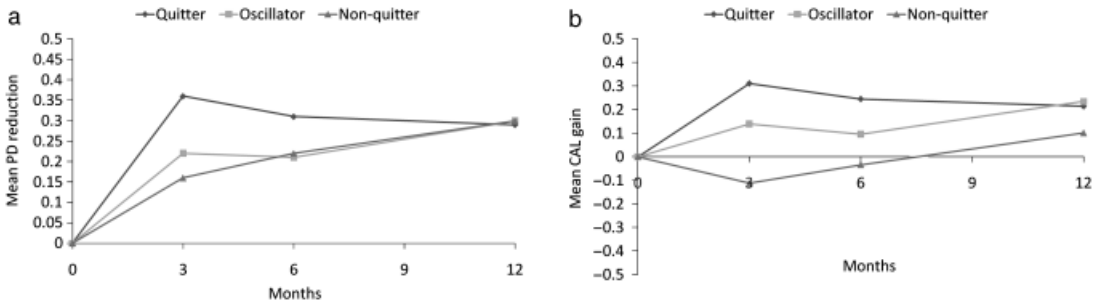

Fig. 2. Mean probing (PD) reduction (a) and clinical attachment level (CAL) gain (b) by smoking status group after 1 year (quitter, oscillator, non-quitter).

Table 3. Prevalence and extent [mean percentage (*N*)] of sites per subject presenting CAL and PD ⩾4 mm and 4–6 mm at baseline and 12-month follow-up for quitters ($N = 17$) and non-quitters ($N = 35$)

		Baseline		12 months	
Categories	Group	%	Extent (SD)	%	Extent (SD)
Clinical attachment level					
⩾4 mm	Quitters ($N = 17$)	100	47.6 (16.9)	100	41.2 (13.8)†
	Non-quitters ($N = 35$)	100	54.9 (25.3)	100	52.3 (22.9)†
	P	1	0.28	1	0.07
4–6 mm	Quitters ($n = 17$)	100	40.7 (12.5)	100	41.2 (13.3)
	Non-quitters ($n = 35$)	100	41.4 (16.5)	100	42.9 (17.0)
	P	1	0.88	1	0.70
Probing depth					
⩾4 mm	Quitters ($N = 17$)	100	21.3 (15.3)	94.1	10.2 (7.4)†
	Non-quitters ($N = 35$)	100	32.6 (19.9)	97.1	21.5 (16.1)†
	P	1	0.04*	0.60	0.01*
4–6 mm	Quitters ($N = 17$)	100	19.9 (13.9)	94.1	9.2 (6.5)†
	Non-quitters ($N = 35$)	100	29.6 (16.7)	97.1	19.7 (13.7)†
	P	1	0.04*	0.60	<0.01*

*$p <= 0.05$, paired data.
†$p <= 0.001$, paired data.

effective for the NQ and Q groups, and there were no significant differences between groups at any time for the variables PD and BoP. CAL gain was significant for Q group only, and the magnitude of gain was significantly greater in Q group for sites ⩾4 mm.

Ninety-three subjects wishing to quit smoking who applied for a smoking cessation program in the UH were included. For ethical reasons, no randomization regarding smoking cessation intervention was conducted; thus, subjects were analysed according to their response to anti-smoking intervention, i.e those who quit smoking up to 12 months and those who never quit or oscillated. Even so, NQ and Q groups were comparable as regards to age, gender, income, years of study, packs per year of life, number of teeth, CAL, PD, BoP and VP.

Initial levels of CO were significantly lower in individuals who stopped smoking after 1 year ($p = 0.03$), although no significant difference was found regarding mean pack years. These results are consistent with the study of Nasry et al. (2006), which have further observed that initial levels of CO were predictors of smoking cessation success.

The methods used to assess baseline smoking detection and compliance to the smoking cessation program comprised an interview (self-reporting) and monitoring of expired air CO. These methods are simple, inexpensive, reliable and widely described in the literature (Schuurmans et al. 2004, Nasry et al. 2006, Bouloukaki et al. 2009). Although cotinine detection is the optimal method to detect recent tobacco smoking exposure, this method is considered to be inappropriate in smoking cessation programs, as nicotine replacement use by the participants may result in false positive results (Scott et al. 2001). In the present study, all patients who reported not smoking had their level of CO <6 ppm, therefore consistent with self-reporting through interviews.

The loss to follow-up in this study was similar and as disappointing as in the Preshaw et al. (2005) study, over 50%. The smoker profile during smoking cessation treatment is unstable; i.e. a high probability of low morale is found throughout the study, which makes the research work extremely difficult. The relationship between smoking and depression is well established in the literature (Berlin et al. 2009), with studies ranging from reporting bouts of depression or exacerbated depression in smoking cessation program participants (Glassman et al. 2001, Thorndike et al. 2008), to an increased risk for suicide during this phase (Hughes 2008, Riala et al. 2009). All those factors may have accounted for the low compliance observed in the present study. Considering a 3.5 mean of CAL in the Q group and a 4.0 CAL mean in the NQ group after 1 year, and a sample size of 17 and 35 in each group, respectively, the statistical power was 0.68.

On the other hand, the smoking cessation rate was up to 30%, superior to the 20% rate observed in previous periodontal literature (Preshaw et al. 2005), and to the mean 10% found in studies involving dentists (Johnson 2004). This successful rate could be influenced by the multi-disciplinary approach employed in this study (Sales et al. 2006, Binnie et al. 2007, Cofta-Woerpel et al. 2007, Webb et al. 2010), and also by the reinforcement every 3 months with the help of the dental team through motivational interviewing techniques.

Overall, both groups attained significant reductions in PD, and only the Q group presented a significant mean intra-group CAL gain. However, no differences between groups could be detected in overall mean of both CAL and PD at any time during the study. When subgroup analysis was performed only for sites presenting baseline PD⩾4 mm, in order to compare with the results and conclusions stated by

Table 4. Prevalence (%) and extent (% sites) (95% CI) of CAL and PD change by smoking cessation group [quitters (Q) and non-quitters (NQ)] and thresholds of changes in PD and CAL during the 1-year study period

Group	CAL loss <2 mm		CAL loss <1 mm		No change CAL		CAL gain $\geqslant 1$ mm		CAL gain $\geqslant 2$ mm	
	%	Extent (SD)	%	Extent (SD)	%	Extent (SD)	%	Extent (SD)	%	Extent (SD)
Q ($N = 17$)	100	9.8 (5.7)	100	27.9 (10.5)	100	72.1 (10.5)	100	39.3 (12.3)	100	15.8 (7.8)
NQ ($N = 35$)	100	12.7 (8.1)	100	32.7 (13.1)	100	67.3 (13.1)	100	34.4 (14.8)	100	14.7 (11.2)
P	1	0.20	1	0.19	1	0.19	1	0.24	1	0.72
Group	PD increase $\geqslant 2$ mm		PD increase $\geqslant 1$ mm		No change PD		PD reduction $\geqslant 1$ mm		PD reduction $\geqslant 2$ mm	
	%	Extent (SD)	%	Extent (SD)	%	Extent (SD)	%	Extent (SD)	%	Extent (SD)
Q ($N = 17$)	94.1	3.9 (4.0)	100	19.5 (8.6)	100	60.7 (15.2)	100	39.3 (15.2)	94.1	10.4 (9.4)
NQ ($N = 35$)	88.6	5.6 (4.8)	100	22.7 (12.8)	100	63.5 (16.9)	100	36.5 (16.9)	94.3	14.0 (12.8)
P	0.53	0.23	1	0.35	1	0.57	1	0.57	0.98	0.33

SD, standard deviation; CAL, clinical attachment level; PD, probing depth; CI, confidence interval.

Preshaw et al. (2005), both groups presented significant PD reduction and CAL gain. Quitters presented a 1.32 mm CAL gain *versus* 0.85 mm for NQ for sites presenting baseline PD $\geqslant$ 4 mm, this difference being significant ($p = 0.02$). However, the results of this last analysis should be interpreted with caution. Even so, it seems that quit smoking can promote an additional benefit to the non-surgical periodontal treatment, especially with regards to CAL gain.

Furthermore, when data were analysed regarding CAL and PD change estimates, it was observed, a non-significant positive trend favouring quitters towards a higher extent of stable sites (that is, no CAL loss or no PD increase), fact that could not be observed in non-quitters, who also presented overall mean CAL loss at the first 3 months of follow-up. This is an important observation, meaning that periodontal treatment associated with smoking cessation at least reduces the probability of CAL loss, fact that might be more significant after a long-term observation and control period. On the other hand, oscillators presented a similar feature pattern when compared with the quitter group.

Measurement bias in PD recording could be more prone to happen in this study, because smoking subjects with decreased inflammatory process present resistance to penetration of the probe, due to the presence of a fibrotic tissue (Biddle et al. 2001), while patients who quit smoking may also have a false reading through less resistance to penetration of the probe, depending on the immunoinflammatory changes caused by smoking cessation. This may have influenced the results observed between the groups regarding PD and CAL.

No significant change over time was observed in BoP in both groups. Morozumi et al. (2004a, b) reported an increase in gingival blood flow after smoking cessation, as observed with Laser Doppler flowmetry, supporting the theory of vasoconstriction by nicotine in human periodontal tissues (Clarke et al. 1981). Nevertheless, Nair et al. (2003) reported a significant BoP increase in patients with gingivitis and moderate periodontal disease after quitting smoking and concluded that smoking masks the clinical signs of inflammation. This increase found was not observed in the Q group in the present study. This may be explained by the fact that 80% of the subjects used nicotine patches and/or gum, which may have directly influenced BoP.

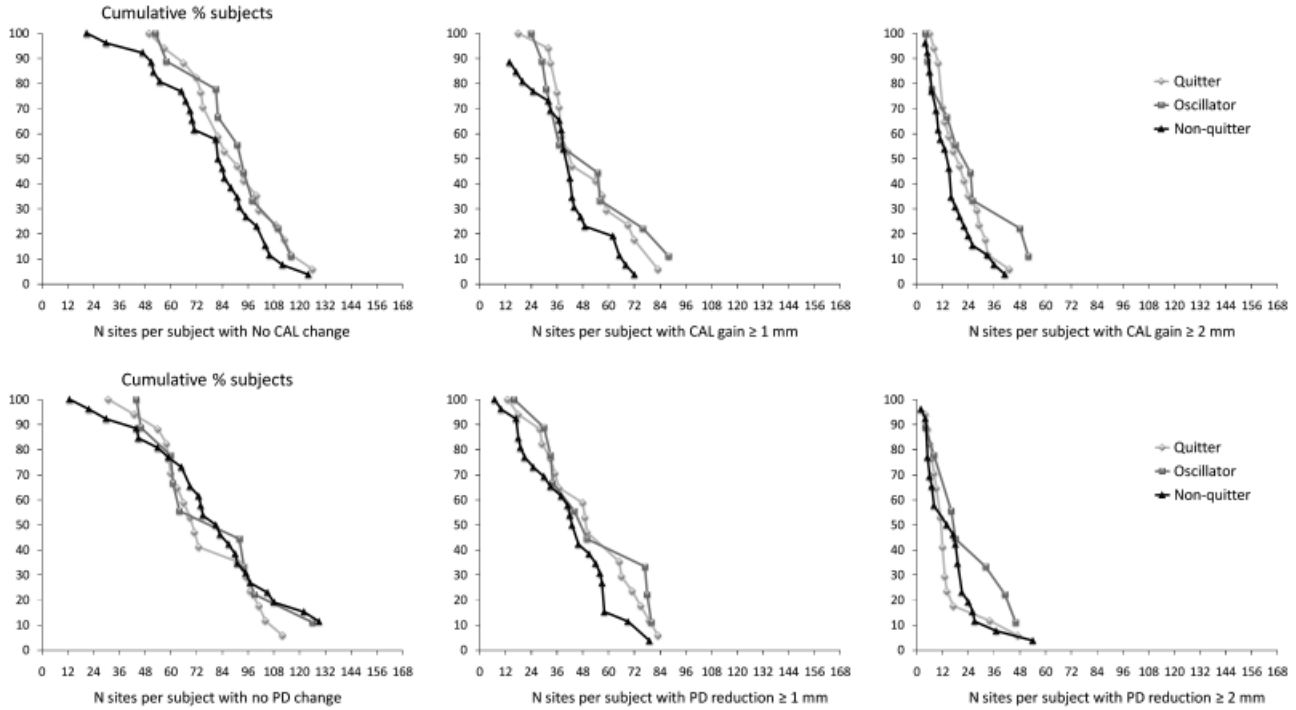

Fig. 3. Prevalence (percentage) and extent (percentage sites) by smoking cessation group and thresholds of changes in probing depth (PD) and clinical attachment level (CAL) during the 1-year study period.

The specific mechanisms by which smoking increases the risk of periodontal diseases are yet to be fully understood. Probably they are multi-factorial by nature and interactive in their effects. They include structural and immunologic mechanisms (Bagaitkar et al. 2008). As regards to the immunological mechanisms, tobacco components may impair the chemotaxis and phagocytosis of neutrophils (Matthews et al. 2011), modifying the production of cytokines and inflammatory mediators (Tymkiw et al. 2011). Also, smoking decrease blood flow and impairs revascularization of the periodontal tissues, thereby causing delayed wound healing (Ojima & Hanioka 2010). It is also unclear how long after quitting smoking, it takes the body to return to its normal inflammatory conditions, and this is another very important factor when interpreting the results. Domagala-Kulawik (2008) reports that, after smoking cessation many changes in the immune system are permanent. In turn, Bouloukaki et al. (2009), describes a return in the balance of inflammatory cells 6 months after smoking cessation. Morozumi et al. (2004a, b) states that it takes more than 8 weeks for levels of IL-1β, IL-8, TNF-α and VEGF to return to its normal values, and that the role (or function) of neutrophils is still not completely recovered after this period, compared with non-smokers. Thus, the slight effect of smoking cessation on CAL gain in the Q group, and the absence of effect on PD may be due to an insufficient follow-up time for the effects of smoking cessation on periodontal status to be observed.

Although a significant decrease in the percentage of sites with VP was observed after 12 months, plaque levels were still elevated in both groups (58.6% Q and 72.0% NQ). These means were similar to those found after 1 year in the Preshaw et al. (2005) study (69.7% NQ and 73.4% Q). However, they are still incompatible with the expected after periodontal treatment and OHI. One possible reason may be the dichotomous system (present/absent) used in this study, which does not take into account the amount of plaque built up. Another possibility may be the depressive profile of participants of this study, as mentioned before. Depressed patients may neglect oral hygiene as a result of reduced mood and interest (Monteiro da Silva et al. 1996, Saletu et al. 2005). This means that much more effort must be done in following studies aiming to perform smoking cessation interventions in periodontology in order to provide an alternative to the traditional OHI program to attain better compliance and reduction of plaque scores compatible with health in these subjects. These can also include individually tailored oral hygiene procedures, which also employ cognitive and motivational interviewing techniques, recently shown to be a successful alternative to the general OHI (Jönsson et al. 2009, 2010).

It was concluded that, in subjects from a Smoking Cessation Clinic, smoking cessation promoted clinical attachment gain, after 1 year of follow-up, for Q group only, and the magnitude of gain was significantly greater in Q group for sites $\geqslant$4 mm. More interventional studies with longer observation period are needed, in order to establish the effect of smoking cessation on periodontal status.

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Clinical Relevance

Scientific rationale for the study: Studies have shown a higher prevalence, extent and severity of periodontal disease in smokers than in non-smokers. However, there is little data about the effect of quitting smoking on periodontal conditions.
Principal findings: Although quitters presented significant clinical attachment gain, there were no differences between groups after 1 year of follow-up regarding periodontal parameters.
Practical implications: Smoking cessation may be an adjunct to periodontal treatment.