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The effect of quitting smoking on chronic periodontitis

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Abstract

Objectives: To evaluate longitudinally the effect of smoking cessation on clinical and radiographic outcomes following non-surgical treatment in smokers with chronic periodontitis.

Material and Methods: Forty-nine smokers with chronic periodontitis who wished to quit smoking were recruited. Full-mouth probing depths, bleeding and plaque data were recorded at baseline, 3, 6 and 12 months. Clinical attachment levels were recorded at target sites and subtraction radiography was used to assess bone density changes. Patients received non-surgical periodontal therapy during the first 3 months and supportive periodontal care over the remainder of the study. Smoking cessation counselling was provided according to individual need.

Results: After 12 months, of patients with complete data, 10 had continuously quit smoking (20% of the original population), 10 continued smoking and six were oscillators (those patients who quit and then relapsed). There were no differences between the groups following treatment with respect to mean clinical or radiographic parameters. Analysis of probing depth reductions between baseline and month 12, however, and comparing quitters with the other two groups combined, demonstrated a significant difference in favour of quitters ($p < 0.05$). Furthermore, quitters were significantly more likely to demonstrate probing depth reductions $\geqslant 2$ and $\geqslant 3$ mm than non-quitters and oscillators ($p < 0.05$).

Conclusion: Quitting smoking has an additional beneficial effect in reducing probing depths following non-surgical treatment over a 12-month period.

Key words: chronic periodontitis; clinical trial; smoking; smoking cessation

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Smoking is one of the most prevalent risk factors for chronic periodontitis and risk calculations suggest that 40% of chronic periodontitis may be attributable to smoking, with an increased odds ratio (OR) of 5.4 (Brothwell 2001). Indeed, Haber (1994) has described a discrete disease entity–smoking-associated periodontitis. In their review, Kinane and Chestnutt (2000) observed that the stronger the association between smoking and periodontal disease, the more likely that smoking is implicated as a risk factor, and the strength of this association can be measured by relative risk expressed in terms of the OR. Indeed, most studies report that smoking increases the risk of periodontal disease between two- and six-fold. For example, Calsina et al. (2002), in a case–control study of 240 private dental patients, showed that smokers had 2.7 times, and former smokers 2.3 times, greater probabilities to have established periodontal disease compared with non-smokers. Linden & Mullally (1994) found the OR to be as high as 14.1 in young subjects whereas Hyman & Reid (2003) examined data from the National Health and Nutrition Examination Survey III and reported an OR of 18.6 for $\geqslant 3$ mm attachment loss among 20–49-year-old smokers compared with non-smokers. Among those over 50 years of age, the OR increased to 25.6 for loss of attachment $\geqslant 4$ mm.

Bergstrom (2003) suggested that smoking, as an associated relative risk factor, is dependent on the definition of disease and prevalence. For a broad definition of disease (1% pockets $\geqslant 5$ mm), the OR was 3.0. When the definition of disease was narrow (15% pockets $\geqslant 5$ mm) the OR was 12.1. Heavy exposure was associated with greater risk and, for the combination of a narrower disease definition and heavy exposure, the risk was defined by an OR of between 9.8 and 20.3.

Numerous authors have reported the potential effects of smoking on the bacterial challenge, the host's periodontal tissues and the immuno-inflammatory response; thus suggesting mechanisms by which patients are at increased risk of periodontitis. For example, the sustained peripheral vasoconstriction of the gingival microvasculature caused by chronic low doses of nicotine may be selective for periodontal anaerobes

(Loesche 1994). This may induce a greater prevalence of the more pathogenic orange and red complex species (Haffajee & Socransky 2001) and the putative pathogens *Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythensis* (i.e. *Bacteroides forsythus*) (Zambon et al. 1996, Kazor et al. 1999). An increase in neutrophil elastase suggests enhancement of degranulation in the neutrophils of smokers (Soder et al. 2002), and increased concentrations of macrophage-derived tumour necrosis factor-α (TNF-α) suggest a more destructive disease process (Bostrom et al. 1998, 1999, Fredriksson et al. 2002). These observations and others suggest that patients with periodontitis who quit smoking may demonstrate significant benefit at the microbiological and immunological levels: more effective phagocytosis and digestion by neutrophils; reduced secretion of TNF-α by macrophages; an increase in oxygen tension within the gingival microvasculature; and a shift towards a less pathogenic subgingival microflora.

At the clinical level, the benefit of quitting smoking for smokers with periodontitis has been inferred from case–control and parallel group studies of smokers, ex-smokers and never smokers following periodontal treatment. For example, non-smokers tend to demonstrate greater resolution of probing depths and improved clinical attachment levels following conventional, non-surgical treatment alone (Preber & Bergstrom 1985, 1990, Ah et al. 1994, Preber et al. 1995, Rosen et al. 1996, Grossi et al. 1997, Kinane & Radvar 1997, Renvert et al. 1998, Palmer et al. 1999, Ryder et al. 1999) and when combined with antimicrobial therapy (Kinane & Radvar 1997, Palmer et al. 1999, Ryder et al. 1999). Bergstrom's group concluded that, as periodontal health appears to improve in former smokers, then smoking cessation is likely to be beneficial (Bergstrom et al. 2000). Currently, however, there are no published, longitudinal studies investigating the benefits of smoking cessation on the periodontium and several authors have expressed concern for this lack of evidence (Qandil et al. 1997, Meinberg et al. 2001, Scott et al. 2001).

The aim of this clinical study was to evaluate the effect of quitting smoking on clinical and radiographic outcomes following the non-surgical treatment of chronic periodontitis.

Materials and Methods

The investigation was a 12-month, longitudinal, non-blinded, clinical trial to investigate the effect of quitting smoking on treatment outcomes following conventional non-surgical management of chronic periodontitis. The Newcastle and North Tyneside Local Research Ethics Committee granted favourable ethical opinion for the study.

Study population

Forty-nine subjects with chronic periodontitis from the Department of Periodontics at Newcastle School of Dental Sciences were recruited. All subjects were smokers who had expressed an interest in quitting the habit. All subjects were given a study information document and consent form at the initial visit.

Inclusion criteria

The patients had a diagnosis of moderate-to-severe chronic periodontitis and at least six posterior teeth, each with at least one inter-proximal site with probing depth $\geqslant 5$ mm and alveolar bone destruction at least one third of the root length. These sites were designated ''target sites'' for clinical attachment measurements and digital subtraction radiography (DSR).

Exclusion criteria

The following exclusion criteria were implemented: patients who had a recent history of periodontal therapy (within 6 months); chronic users of non-steroidal anti-inflammatory drugs (NSAIDs) or steroid medication; patients who had medical or medication histories that precluded pharmacological smoking cessation intervention, safe completion of the study, or impacted on periodontal disease status; patients who required prophylactic antibiotics prior to dental treatment.

Study design

The clinical trial comprised the following visits:

Screening

The target sites were identified from a full periodontal clinical examination. A smoking history was obtained and personalized risk feedback based on the severity of chronic periodontitis status and the smoking history in pack-years was given. A carbon monoxide (CO) reading was taken at the screening using a CO meter (Micromedical Ltd, Rochester, UK). This reading was repeated at every visit thereafter for the duration of the study.

Baseline

Full-mouth clinical measurements were recorded, and intra-oral vertical bitewing radiographs taken. Salivary cotinine levels were measured using a chair-side colorimetric assay test kit (Mermaid Diagnostics, Birmingham, UK).

Between baseline and the month 3 visit, conventional, non-surgical periodontal treatment was undertaken as an intensive course over several visits. Smoking cessation counselling was also given at each of these visits.

Months 3, 4, 5, 6, 7, 8, 9, 10, 11 and 12

Further smoking cessation counselling and support together with supportive periodontal therapy were provided.

Months 3, 6 and 12

In addition to smoking cessation counselling and support, full-mouth clinical measurements were recorded. Salivary cotinine levels were measured. Intra-oral, vertical bitewing radiographs were taken again only at month 12.

Clinical measurements

Periodontal status was measured using conventional measurements: plaque scores, probing depths, bleeding on probing (BOP) and clinical attachment levels.

Plaque scores

Plaque covered surfaces (six sites per tooth) were identified by disclosing all teeth with a proprietary brand of disclosing solution (Butler, Chicago, IL, USA) and recorded dichotomously. Percent (%) full-mouth scores were then calculated.

Probing depths

Full-mouth probing depths (mm) were recorded using a manual UNC probe at six sites per tooth.

BOP

BOP was recorded dichotomously following probing and calculated as % full-mouth scores.

Clinical attachment levels

Clinical attachment levels were recorded using a Florida disk probe (Florida Probe Corporation, Gainesville, FL, USA). The probe was set at a resolution of 0.1 mm and a standardized probing force of 0.25 N. A validated outlier reduction technique was used. Duplicate measurements were recorded at each site and the mean recorded. When the two measurements differed by >1.0 mm, a third measurement was recorded and the median of the three readings was calculated (Namgung & Yang 1994).

Radiographic examination

Standardized intra-oral vertical bitewing radiographs were taken at target sites using a modified cephalostat at baseline and month 12 to evaluate changes in bone density using DSR. Standard E speed intra-oral films were used to obtain vertical bitewing radiographs with an aluminium step wedge attached to the film holder to act as density reference in the DSR procedure. A cephalostat was used to ensure identical radiographic projection geometry. Exposure conditions and processing were also identical on each occasion (Fidler et al. 2000).

Radiographs obtained at baseline and month 12 were scanned, digitized and subtracted using dedicated DSR software (Compare plug-in) that ran within Image Tool® (University of Texas, San Antonio, TX, USA). The subtraction macro had the following functions: correction for affine differences in perspective between the two images using a patch minimization process; density normalization using first or second order polynomials to match coordinate pixels in the paired images; adjustment of contrast and brightness of the image representing the difference between the two images (Ellwood et al. 1997).

Density changes in subtraction images were assessed using two methods. Firstly, two examiners scored the subtraction images using a five-point subjective ranking system: $+2$ = definitive bone gain; $+1$ = possible bone gain; 0 = no change; -1 = possible bone loss; -2 = definite bone loss. Secondly, quantitative determinations of bone change were made by reference to the aluminium step wedge to generate bone change expressed in aluminium volume equivalents (mm^3Al), with a positive number representing bone gain and a negative number representing bone loss.

Periodontal treatment

Periodontal treatment was undertaken between baseline and month 3. This comprised conventional, non-surgical management using local anaesthetic. The first phase of treatment comprised oral hygiene instruction (OHI) with advice on brushing teeth and the use of adjunctive cleaning aids according to individual patient needs. This was followed by methodical root surface instrumentation using manual and ultrasonic instruments. On an average, patients were seen for between four and six treatment visits, each lasting 45–60 min. Supportive periodontal therapy (reinforcement of OHI, supragingival scaling and prophylaxis) was performed at post-treatment appointments as necessary, according to individual patient needs.

Smoking cessation counselling

Smoking cessation advice was provided at the first treatment appointment as appropriate, based on the smoking history. Smoking cessation counselling was subsequently reinforced at each visit up to, and including month 12. A variety of methods were employed including simple counselling and advice, nicotine replacement therapy (NRT) and the prescription of pharmaceuticals, with each intervention being tailored to individual needs. Those patients who were prescribed buproprion (Zyban, GlaxoSmithKline, Brentford, UK) as part of their smoking cessation regime had their first dose to coincide with the first periodontal treatment visit. All doses of buproprion were prescribed according to the regimen 150 mg once per day for 7 days and then twice daily for 2 months. The ''quit day'' was timed to fall between the first and second periodontal treatment visits. The prescription of Zyban was given only after consultation with the patient's general medical practitioner.

Compliance

Compliance with smoking cessation counselling was assessed by using diaries (self-reporting), CO content in expired air (measured using the CO monitor at each visit), and cotinine concentrations in saliva which were measured using a disposable, near-patient, colorimetric test kit (Mermaid Diagnostics, UK) at 3-monthly intervals (Cope et al. 2000).

Data management

Clinical and demographic data were recorded manually in individual subject case report files. These data were then double-entered by a professional data management team (Tyne Prep UK Ltd. Gateshead, UK).

Statistical analyses

For the analysis, each subject was categorized (based on continuous quit status throughout the study) as either having: not attempted to quit smoking (non-quitter); made an unsuccessful attempt to give up smoking (oscillator); quit smoking for at least the entire 12-month duration of the study (quitter).

The data were analysed in SPSS version 11. Repeated measures analysis of variance (ANOVA) was used to test simultaneously: whether there were significant differences between time points (baseline, 3 , 6 and 12 months); whether there were differences between the three groups (non-quitters, oscillators and quitters); and whether there were time point by quit status interactions. Differences between specific time points or groups were only investigated when one of the above effects was significant at the 5% level.

The primary level of analysis was the subject. For each outcome, a single summary measure was derived for each subject by taking the mean value across all the sites for which the outcome was assessed. The change in probing depths from baseline was also analysed as a pre-specified comparison of direct interest. The group variable was replaced with two indicator variables, one for quitters and one for the other subjects; that is, effectively pooling oscillators and non-quitters. Bone changes, using aluminium equivalents data (volume in mm^3Al) were analysed using parametric tests (ANOVA) and categorical changes were analysed using nonparametric tests (Kruskal–Wallis statistics).

The number of sites at which there were reductions in probing depth $\geqslant 2$ or $\geqslant 3$ mm (i.e. clinical improvements) from baseline to month 12 was calculated for each subject, and analysed using negative binomial regression. For each subject, the log of the total number of sites was included in the model as an offset, and significant variation between the smoking groups was assessed by comparing the change in -2 loglikeli-

hood observed when fitting this with the percentage points of a chi-squared distribution with two degrees of freedom. Data were expressed as relative risks (RR) and 95% confidence intervals (CIs).

Results

Study population

Forty-nine subjects were recruited, with a mean (SD) age of 42.0 (8.7) years. The age range was 23–61 years. Eighteen subjects were male and 31 were female. All subjects satisfied the inclusion criteria and suffered from moderate to advanced chronic periodontitis.

Smoking status at month 12

All subjects were confirmed smokers at baseline. At month 12, there were 11 continuous quitters (22.4% of the original cohort) and 11 continuous non-quitters. Fifteen subjects had withdrawn from the study. Twelve subjects were classified as oscillators who had either quit smoking but relapsed, or had only quit at certain time points throughout the study.

For each of the periodontal outcomes, statistical analyses were only undertaken for subjects for whom there were complete data sets. For example, for attachment loss there were: 10 quitters (20.4% of the original cohort), 10 non-quitters and six oscillators. The age, gender, smoking exposure status and numbers of remaining teeth at baseline for the subjects with complete data sets are presented by subsequent quit status in Table 1. The remaining subjects who had either withdrawn early (thereby preventing a determination of quit status) or for whom there were incomplete data (because of their failing to attend some of the follow-up appointments) are indicated in Table 1 as ''Quit status not known/incomplete data''. No significant differences between these groups at baseline were identified with respect to the parameters recorded in Table 1 ($p > 0.05$).

Compliance

Each subject was asked about his or her smoking status at each time point. The CO reading was used to confirm the self-reported status. All quitters had CO readings of 10 p.p.m. or below. Eighty-two percent of non-quitters (nine out of 11) had readings above 10 p.p.m. One non-quitter had a CO reading of 10 at month 3 and another had a CO reading of 7 at month 6 and a reading of 8 at month 12, however, both of these subjects reported that they were still smoking. In addition, the salivary cotinine test was used as a marker for compliance, with all quitters scoring 0, indicating no nicotine metabolites present. A score of 1–6 (indicating a detection of cotinine) confirmed the smoking status of the non-quitters. This test was also of value as a motivational tool with regard to smoking cessation.

Data presentation

The clinical data are presented diagrammatically as box and whisker plots showing the median values, inter-quartile and full ranges of values. Outliers were defined as data points greater than twice the inter-quartile range from the median value. The mean (SD) clinical data for probing depths, attachment levels, bleeding scores and plaque scores are presented for all groups and at all time points in Table 2.

Probing depths

Box and whisker plots for probing depths are shown for all sites and for all subjects in Fig. 1a and for all sites in the defined quitters, oscillators and non-quitters in Fig. 1b. The differences between baseline and each post-baseline time point were significant ($F_{3,66} = 27.5$; $p < 0.001$). The decrease in probing depths observed in Fig. 1a, however, was not entirely linear across the four time points, with the test for a quadratic effect being significant at the 5% level ($F_{1,22} = 5.66$; $p = 0.03$). Overall, across the four time points, the differences between groups were not significant ($F_{2,22} = 0.89$; $p = 0.42$) (Fig. 1b), nor was there any evidence that the pattern of the reduction in probing depths differed between the groups ($F_{6,66} = 1.23$; $p = 0.30$) (Fig. 1b, Table 2).

When the analysis of probing depths involved only those sites that were considered to be diseased at baseline (> 3 mm), the outcomes of the repeated measures ANOVA were similar to those seen for all sites, with a highly significant reduction over time ($F_{3,66} = 62.4$; $p < 0.001$). Across the four time points, the differences between groups were not significant ($F_{2,22} = 0.46$; $p = 0.64$), nor was there any evidence that the pattern of the reduction in probing depths differed between the groups ($F_{6,66} = 1.07$; $p = 0.39$) (Fig. 2, Table 2).

Mean (SD) changes in probing depth from baseline at diseased sites in the three groups are shown in Fig. 3. For the ANOVA, the group variable was replaced with two indicator variables: one for quitters and one for all other subjects. The difference between quitters and the other groups was significant ($p < 0.05$) with an additional reduction in probing depth of 0.32 mm (95% CI: 0.07, 0.52) for quitters over the other two groups at month 12.

The numbers of sites demonstrating probing depth reductions $\geqslant 2$ and $\geqslant 3$ mm in the smoking sub-groups are presented in Table 3. When considering the numbers of sites demonstrating probing depth reductions $\geqslant 2$ mm from baseline to month 12, negative binomial regression indicated that quitters were significantly more likely to demonstrate

Table 1. Baseline characteristics of patients by subsequent quit status

	Non-quitters ($n = 10$)	Oscillators ($n = 6$)	Quitters ($n = 10$)	Not known/incomplete data ($n = 23$)
Gender (M:F)	5:5	1:5	4:6	8:15
Mean (SD) age (years)	41.4 (4.9)	41.2 (8.0)	45.3 (9.9)	41.0 (9.8)
Mean (SD) pack years of smoking	22.7 (5.3)	24.8 (11.5)	25.0 (18.0)	24.9 (15.6)
Number of teeth present	25.7 (1.9)	24.2 (1.3)	23.7 (2.9)	24.5 (3.0)
Salivary cotinine score	2.4 (1.3)	2.5 (1.2)	2.3 (1.2)	3.6 (1.5)
CO reading (p.p.m.)	34.1 (15.6)	24.4 (22.8)	18.7 (13.5)	27.8 (13.6)

CO, carbon monoxide.

Table 2. Mean values for clinical outcomes: full-mouth and diseased sites (>3 mm) probing depths (mm), clinical attachment levels (mm), bleeding on probing (%) and plaque scores (%)

Outcome	Group (n)	Mean (SD) scores				Repeated measures ANOVA		
		baseline	3 months	6 months	12 months	time point	smoking status	time point by smoking interaction
Full-mouth mean probing depth (mm)	Non-quitters (10)	3.44 (0.62)	3.10 (0.52)	2.96 (0.38)	2.83 (0.23)	$F_{3,66} = 27.5$; $p<0.001$	$F_{2,22} = 0.89$; $p = 0.42$	$F_{6,66} = 1.23$; $p = 0.30$
	Oscillators (6)	3.71 (0.80)	3.47 (0.92)	3.31 (1.09)	3.21 (1.05)			
	Quitters (9)	3.98 (0.68)	3.32 (0.41)	3.23 (0.43)	3.02 (0.52)			
Diseased sites (>3 mm) mean probing depth (mm)	Non-quitters (10)	4.84 (0.33)	4.10 (0.53)	3.93 (0.53)	3.69 (0.38)	$F_{3,66} = 62.4$; $p<0.001$	$F_{2,22} = 0.46$; $p = 0.64$	$F_{6,66} = 1.07$; $p = 0.39$
	Oscillators (6)	5.03 (0.54)	4.36 (0.92)	4.18 (1.14)	3.95 (1.09)			
	Quitters (9)	5.31 (0.43)	4.22 (0.46)	4.08 (0.60)	3.71 (0.54)			
Attachment level (mm)	Non-quitters (10)	10.7 (0.8)	10.8 (1.0)	10.8 (1.4)	10.5 (1.4)	$F_{3,66} = 0.93$; $p = 0.43$	$F_{2,23} = 3.84$; $p = 0.04$	$F_{6,69} = 1.56$; $p = 0.17$
	Oscillators (6)	10.5 (1.9)	10.3 (2.1)	10.4 (1.9)	10.3 (1.8)			
	Quitters (10)	12.2 (1.4)	11.9 (1.6)	12.2 (1.4)	12.3 (1.3)			
Bleeding on probing (%)	Non-quitters (10)	68.8 (23.0)	55.6 (13.1)	60.8 (9.0)	54.6 (7.7)	$F_{3,66} = 7.94$; $p<0.001$	$F_{2,22} = 0.24$; $p = 0.78$	$F_{6,66} = 0.34$; $p = 0.92$
	Oscillators (6)	67.2 (10.2)	60.5 (18.2)	57.4 (27.8)	49.3 (27.4)			
	Quitters (9)	65.1 (24.6)	55.3 (15.5)	54.5 (16.8)	45.8 (20.7)			
Plaque score (%)	Non-quitters (10)	86.8 (10.7)	70.0 (26.7)	71.6 (25.0)	69.7 (30.8)	$F_{3,66} = 9.75$; $p<0.001$	$F_{2,22} = 1.07$; $p = 0.36$	$F_{6,66} = 1.65$; $p = 0.15$
	Oscillators (6)	75.7 (15.9)	75.2 (9.4)	43.3 (24.2)	60.5 (27.8)			
	Quitters (9)	87.8 (10.0)	72.7 (19.0)	66.9 (19.5)	73.4 (14.4)			

The mean scores at baseline, 3, 6 and 12 months are given for non-quitters, oscillators and quitters. The summary data for ANOVA (F, P values) are presented by occasions (time points), groups (non-quitters, oscillators, quitters) and for the interaction of time points by smoking status.
ANOVA, analysis of variance.

an improvement of at least 2 mm compared with non-quitters and oscillators (RR = 1.67 with 95% CI: 1.03, 2.70, $p<0.05$). When the threshold of clinical improvement was raised to a probing depth reduction of $\geqslant 3$ mm, again, quitters were significantly more likely to demonstrate this level of improvement compared with the other subjects (RR = 2.36 with 95% CI: 1.26, 4.42, $p<0.01$).

Clinical attachment level

Mean attachment level data for the three groups at all time points are presented in Table 2. There were no significant differences at the 5% level between time points ($F_{3,66} = 0.93$; $p = 0.43$). There was some evidence to suggest that, overall, the quitters had greater loss of attachment than the other groups ($F_{2,23} = 3.84$; $p = 0.04$) although there was no evidence that this difference varied between time points ($F_{6,69} = 1.56$; $p = 0.17$).

BOP

For BOP, decreases in %BOP between baseline and each post-baseline time point were significant ($F_{3,66} = 7.94$; $p<0.001$). Across the four time points, the differences between groups were not significant ($F_{2,22} = 0.24$; $p = 0.78$) (Table 2), and there was no evidence that the pattern of change was different between the groups ($F_{6,66} = 0.34$; $p = 0.92$).

Plaque

Decreases in plaque scores between baseline and each post-baseline time point were significant ($F_{3,66} = 9.75$; $p<0.001$). Across the four time points, the differences between groups were not significant ($F_{2,22} = 1.07$; $p = 0.36$), and there was no evidence that the pattern of change was different between the groups ($F_{6,66} = 1.65$; $p = 0.15$) (Table 2).

Radiographic bone change

Mean bone density changes together with the 95% CIs for the aluminium equivalents data are presented for non-quitters, quitters and oscillators in Table 4. When considering bone changes using either categorical or aluminium equivalents data, the mean changes identified over the 12-month period

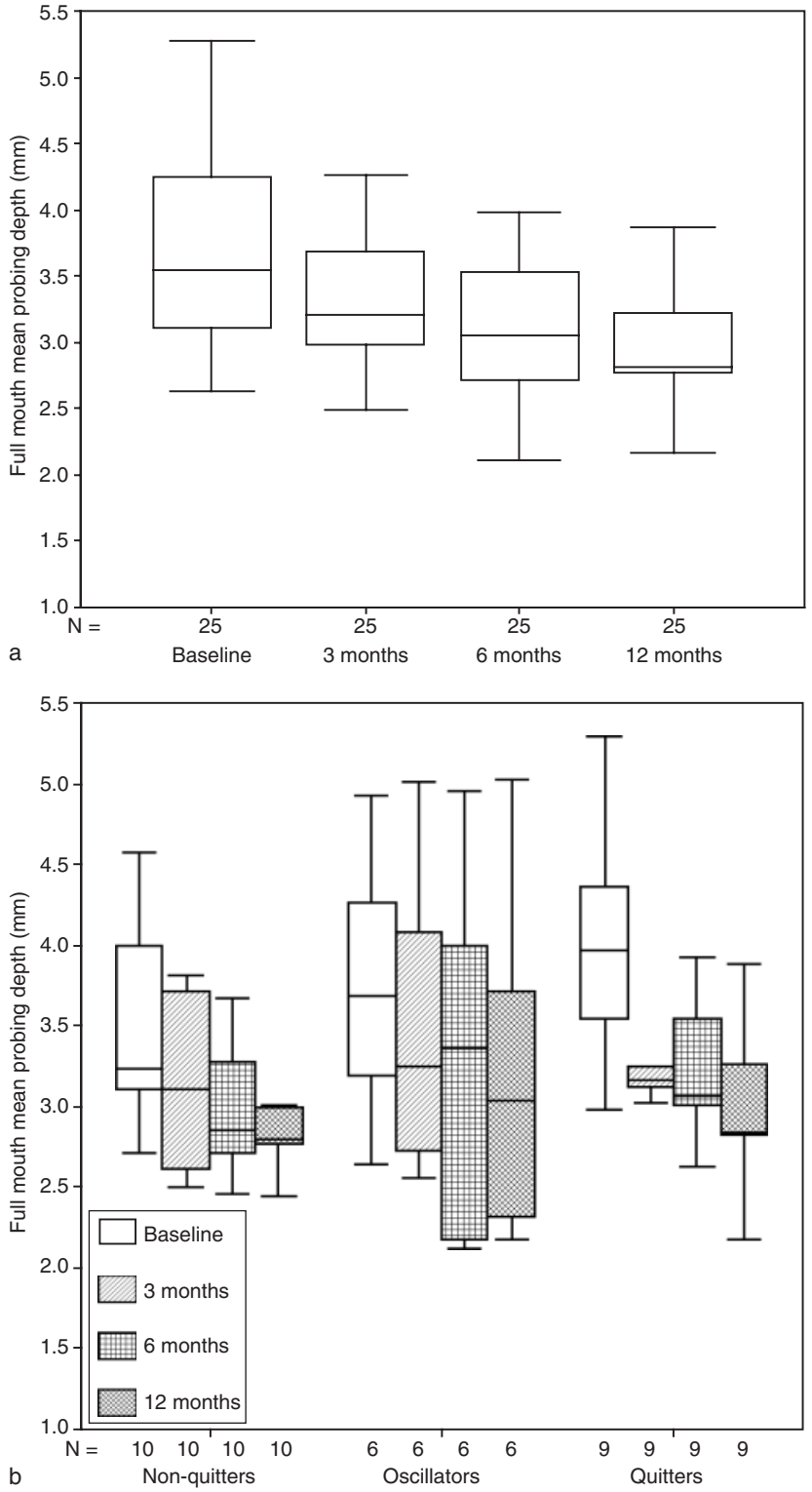

Fig. 1. Box and whisker plots for full-mouth mean probing depths (mm) at baseline, 3, 6 and 12 months for (a) all subjects with complete data ($n = 25$), (b) non-quitters ($n = 10$), oscillators ($n = 6$) and quitters ($n = 9$). The median, inter-quartile and full range of values are shown.

were small and representative of bone loss in all groups. There were no significant differences in bone changes between non-quitters, quitters and oscillators, whether analysed using categorical changes ($p > 0.05$) or analysed using aluminium equivalents data ($p > 0.05$).

Discussion

The principal aim of this project was to evaluate the effect of smoking cessation on clinical and radiographic outcomes following the non-surgical treatment of periodontal disease. This study is the first periodontal clinical trial that has followed longitudinally the clinical periodontal outcomes which occur in smokers when they quit the habit. Previously, clinical trials have only reported outcomes in separate cohorts of smokers, ex-smokers and non-smokers following non-surgical treatment.

For the purpose of the study, 49 smokers were enrolled, all of whom had expressed a desire to quit smoking. An alternative would have been to design the study with two parallel groups: one containing smokers attempting to quit and the other containing smokers with no intention of quitting. This, however, would have resulted in an even smaller number of quitters after 12 months as a significant proportion of those in the attempting-to-quit group would have failed to quit the habit. The intention was therefore to encourage all subjects to quit, assuming that some would be successful and others unsuccessful. The subjects would, therefore, effectively determine themselves the group (non-quitters or quitters) to which they would be allocated (rather than randomized) at the 12 month time point.

Inevitably, because of the unpredictable and individual nature of smokers, the study concluded with three groups (not including those who dropped out of the study): quitters, non-quitters and oscillators. The oscillators were those subjects who quit the habit and relapsed, together with those who may have quit on more than one occasion within the time frame of the study. There was a disappointing number of subjects who withdrew from the trial or who failed to attend appointments for data collection. The multiple, time-consuming appointments and long-term commitment to the study were considered to be contributing factors for those subjects who chose to withdraw, although the deciding factor for most may well have been their failure to quit smoking. The confirmed continuous quit rate that was recorded after 12 months was approximately 20%, which compares favourably with quit rates reported in other studies (Johnson 2004).

It is important to note that the quit groups in this study were self-selected

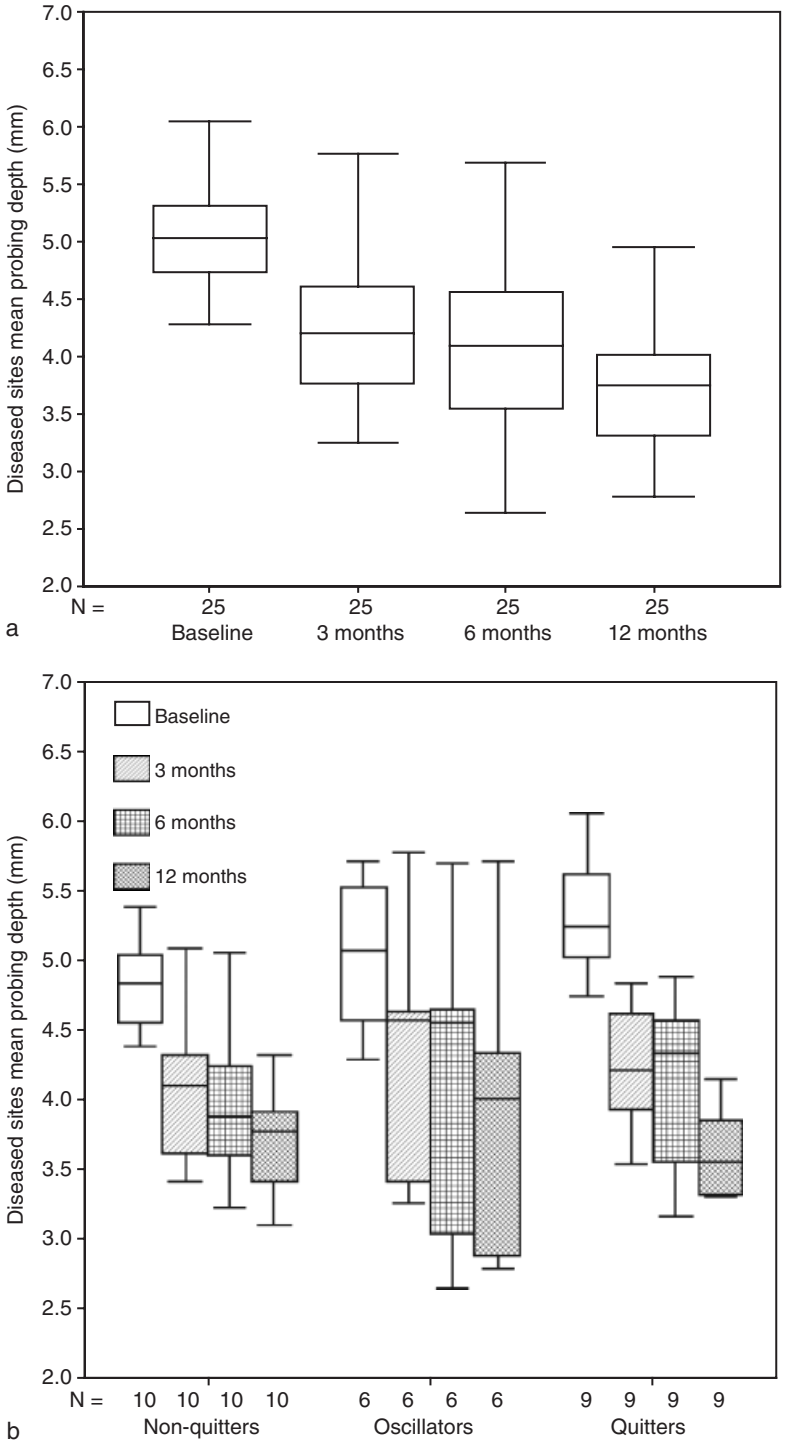

Fig. 2. Box and whisker plots for mean probing depths of diseased sites (>3 mm) at baseline, 3, 6 and 12 months for (a) all subjects ($n = 25$), (b) non-quitters ($n = 10$), oscillators ($n = 6$) and quitters ($n = 9$). The median, inter-quartile and full range of values are shown.

and were not randomly assigned. Table 1 shows that the quit groups (and the group of subjects who withdrew from the study) were very similar in terms of age, gender distribution, tobacco exposure (measured in pack-years), numbers of remaining teeth, and salivary cotinine and CO levels at baseline ($p > 0.05$). In other words, it does not seem likely that there were any pre-existing differences between groups with respect to these variables that could have influenced subsequent success or failure in quitting smoking.

The difficulty that some subjects had in quitting smoking may also explain the relatively large number of oscillators. Furthermore, some ''quitters'' for whom there were missing data because of their failure to attend interim appointments may have failed to attend because they lapsed to being a smoker. For these reasons, the analysis was only undertaken on complete, 12 month data sets for quitters, non-quitters and oscillators.

Generally, the data from this study show that there were no differences in mean probing depths, attachment levels, %BOP and plaque scores between the three groups at any time during the 12 months. This failure to identify differences in clinical measures between the groups may be related to the small numbers of subjects who completed the study, which although disappointing, is, in itself, a relevant finding.

Analysis of the overall probing data revealed that, in all groups and in the cohort as a whole, probing depths reduced significantly over the 12 months of the study, although the change was not linear. For example, there were greater reductions of probing depths over the first 3 months; an observation that is consistent with those made in the classic, definitive studies of patients following non-surgical periodontal treatment (Badersten et al. 1981, 1984). It is worth noting that the results of these and other classic studies (Lindhe et al. 1984, Kaldahl et al. 1988) are now viewed with some caution because there was no consideration given to smoking as a risk factor and the sole unit of analysis of data was the site rather than the subject.

When only the ''diseased'' sites (probing depths >3 mm at baseline) were analysed, again, it was found that there were highly significant reductions in probing depths after baseline, but there were no differences between the groups at any time point, nor was there evidence that the pattern of reduction differed between groups. However, when the mean *reductions* in probing depths between baseline and month 12 were analysed, the continuous quitters had a significantly greater reduction, approximately 0.3 mm, after 12 months compared with the other subjects (Fig. 3). This difference in magnitude of probing depth reduction between (essentially) non-smokers and smokers is consistent with the magnitude of difference seen between the two groups following non-surgical treatment in other reported

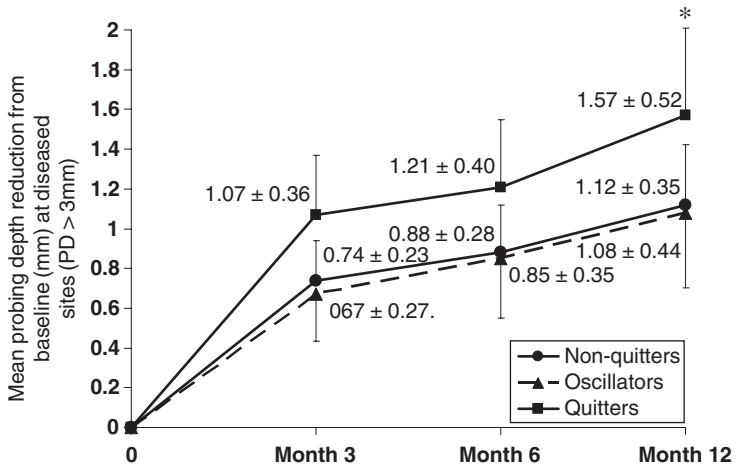

Fig. 3. Means (SD) for the reduction in probing depths at initially diseased sites (>3 mm) between baseline and 3, 6 and 12 months. Changes within each of the three groups are shown separately: quitters, oscillators and non-quitters (*$p<0.05$ compared with oscillators and non-quitters combined).

Table 3. Number (%) of periodontal sites demonstrating probing depth reductions ⩾2 and ⩾3 mm from baseline to month 12

	Non-quitters	Oscillators	Quitters
⩾2 mm improvement *N* (%) of sites	276 (18.0)	146 (16.8)	351 (28.5)
⩾3 mm improvement *N* (%) of sites	79 (5.2)	42 (4.8)	141 (11.5)
Total *N* (%) of sites in each category	1536 (100)	864 (100)	1232 (100)

Table 4. Bone changes at month 12 compared with baseline for non-quitters, quitters and oscillators.

Outcome	Status	*N*	Mean (SD)	95% CI	ANOVA	
					F	*p*
Quantitative bone change (mm^3Al)	Non-quitters	10	−0.08 (0.86)	(−0.12, 0.36)		
	Oscillators	6	−0.08 (0.74)	(−0.18, 0.34)	0.128	0.88
	Quitters	10	0.00 (0.64)	(−0.12, 0.21)		

ANOVA, analysis of variance; CI, confidence interval. Changes evaluated using aluminium equivalents data (mm^3Al).

studies (Preber & Bergstrom 1985, Kaldahl et al. 1996, Grossi et al. 1997, Kinane & Radvar 1997, Pucher et al. 1997, Preshaw et al. 1999, Zuabi et al. 1999).

Not only did the quitters have a greater probing depth reduction, they also had (non-significantly) higher probing depths at baseline. There is no obvious reason or explanation for this observation; indeed, it may just be an anomaly because of the small number of subjects in the study. It is possible that the subjects who quit smoking did indeed benefit from the elimination of this potent risk factor for the disease and that this benefit was manifest as a greater reduction in probing depths over time. It is also possible, however, that following conventional periodontal treatment, all probing depths showed a tendency to regress to the mean and this in itself may have, at least in part, been responsible for the difference in the probing depth reductions between the groups. In other words, because of measurement error or perhaps biological variation, greater or smaller values at first measurement (baseline), will on average have tended to become closer to the group mean on the second measurement irrespective of any intervention made between the measurement occasions (Tu 2004).

The same observations were made for clinical attachment levels; there was no evidence of change of attachment with time, irrespective of analysing the data as a single cohort or in separate groups of non-quitters, quitters and oscillators. The quitters, at all time points, had greater loss of attachment. Again there is no obvious explanation for this, although the greater loss of attachment at baseline may have had a direct association with the greater probing depths at this time point.

An important consideration when measuring changes in probing depths in patients following a treatment intervention is the choice of outcome measure that is employed. Typically, mean probing depth changes are reported in clinical studies, and although these are a useful summary statistic of changes occurring in the periodontal tissues, they are often small changes, and population means are difficult to relate to individual patients or what might be happening at individual periodontal sites. Furthermore, the issue of whether a statistically significant change is also clinically significant is often raised. We would suggest that a clinically significant change is one that can be readily detected at the chair side using conventional techniques and instruments, e.g. a reduction in probing depth of ⩾2 mm. When site-specific changes were analysed in this way, it can be seen from Table 3 that many more sites in the quitters group achieved such thresholds of change, compared with the non-quitters and oscillators. For example, 29% of sites in quitters reduced by ⩾2 mm compared with 16–18% in the other two groups, and more than twice as many sites in quitters reduced by ⩾3 mm compared with the non-quitters and oscillators. Statistical analyses confirmed the increased likelihood of quitters demonstrating these thresholds of improvement compared with the non-quitters and oscillators ($p<0.05$). This is an important clinical finding and confirms the benefit of quitting smoking when combined with non-surgical periodontal therapy.

A complicating factor when assessing the response to treatment by measuring probing depths in patients who quit smoking is that changes in the tissue inflammatory response occurring as a result of quitting smoking may impact on the probing depth measurements directly. For example, there is evidence that there is less penetration of the probe tip into the periodontal tissues in smokers when the tissues are less inflamed (Biddle et al. 2001). Thus, it is possible

that the probing depths recorded in the quitters at post-baseline time-points are over-estimations of pocket depth (because of increased probe tip penetration as a result of changes in the inflammatory response after quitting smoking), an effect that would result in an apparent masking of true probing depth reductions in this group. However, without histological examination, there is no option for confirming or refuting this hypothesis.

There were no significant bone density changes between baseline and month 12, nor between any of the groups of patients. Non-quitters, quitters and oscillators all demonstrated a very small amount of bone density reduction whether calculated using the qualitative or quantitative assessment criteria. This observation is consistent with the very slight bone change reported in a similar cohort of patients with chronic periodontitis following treatment (Preshaw et al. 1999). The mean bone changes seen in both studies were extremely small, however, and, given the small number of patients recruited, should be viewed with some caution.

The results show that although there were no differences between the groups for bleeding, %BOP did reduce significantly at post-baseline time points, as would be expected after periodontal treatment and instruction in oral hygiene. There was no evidence of an increase in %BOP (''rebound BOP'') in those patients who managed to quit compared with those who did not, a finding that has been reported in periodontally healthy patients who quit smoking but who did not receive periodontal treatment or instruction in plaque control (Nair et al. 2003).

There was no evidence of differences between the groups when analysing the plaque scores, and no difference in the pattern of change in plaque levels between the groups with time. Once again, the reduction in plaque scores post-baseline for all groups was statistically significant, which was expected. One possible disadvantage with the design of the study is that the quitters might have been more motivated and compliant with both smoking cessation advice and OHI. This may have shown in their ability to both quit smoking and improve plaque control. A greater reduction in probing depths observed between baseline and month 12 in this group may, therefore, have been associated with greater motivation to improve plaque control rather than an effect of smoking cessation. However, the plaque score data tend to suggest that this was not the case. One additional problem was that all subjects, regardless of group, demonstrated relatively high plaque scores at baseline, and then throughout the 12 months. This might be attributed to the dichotomous (present/absent) system used for scoring plaque and the fact that even minimal, isolated deposits of plaque were scored positively. The low reductions in plaque scores that were observed may also have had an impact on reductions of other clinical measures, such as probing depths and BOP. Thus, the relatively high plaque levels throughout the study may have further compromised the likelihood of identifying differences between the smoking sub-groups in addition to the effect of the high withdrawal rate.

The two main methods for determining smoking status were self-reporting and expired CO levels. Jarvis et al. (1987) suggested that for most clinical applications, expired air CO monitoring provides an acceptable degree of discrimination between smokers and non-smokers. It is a simple and inexpensive method, but is only reliable if the subjects have recently smoked, or if they are heavy smokers. They concluded that cotinine measurement should be considered the optimal method of measuring recent tobacco smoke exposure, because of its sensitivity and stability. Gonzalez et al. (1996) reported data to indicate that serum cotinine levels used as a biochemical marker of smoking status correlated with severity of attachment loss. Both CO monitoring and cotinine measurements are a measure of current smoking status and do not differentiate between never and former smokers. For the purposes of this study, however, the essential requirement was to determine current smoking status. The cotinine test of choice for this study was Smokescreen (Cope et al. 2000). This test was used in part to confirm smoking status, but it was also helpful as a motivational tool to complement smoking cessation intervention. The threshold for defining non-smoking status was less than 10 p.p.m. CO in expired air and a reading of 0 in the salivary cotinine test (confirming the absence of detectable nicotine).

Several subjects used NRT throughout the study, which is a useful adjunct to smoking cessation as it alleviates withdrawal symptoms. Continine has been detected in the saliva of non-smokers following the ingestion of nicotine, which suggests that the cotinine test results of subjects using NRT may have been compromised and shown false positives (Jarvis et al. 1988). The question of whether the nicotine in NRT can adversely affect the periodontal tissues needs to be the focus of further research.

There are a number of well-recognized risk factors for periodontal disease, of which smoking, the focus of this study, is just one. Other potential risk factors include: previous periodontal disease; increased probing depths; infrequent dental attendance; stress; poor oral health; specific bacterial pathogens; other systemic/environmental host factors; diabetes; and inherited risk (Darby 2003). Patients with diabetes were excluded because, with small numbers of patients, it would have been impossible to stratify the groups to account for this additional risk factor. A diabetic smoker over 45, for example, has an OR for attachment loss of 30 times compared with a non-smoker without diabetes (Grossi et al. 1994). It is possible that other unidentified risk factors (such as non-diagnosed diabetes) may have confounded outcomes in this study.

In conclusion:

- The sequential type of design for longitudinal clinical trials involving patients who propose to quit smoking is a viable alternative to the parallel group design;
- clinical outcomes (probing depths, bleeding, plaque scores) improved as expected, following conventional non-surgical treatment, an observation that is consistent with previous studies;
- over the 12-month period, there were no significant differences in mean outcomes between the non-quitters, quitters and oscillators who had complete data sets at 3, 6 and 12 months;
- analysis of change in probing depths between baseline and 12 months indicated a significant reduction in favour of the quitters compared with the rest of the cohort. This may in part, however, be attributed to statistical or biological regression, but may also reflect greater clinical resolution of disease for those who quit smoking;

- furthermore, quitters had a statistically significantly increased likelihood of demonstrating clinically significant probing depth reductions $\geqslant 2$ and $\geqslant 3$ mm compared with the non-quitters and oscillators over the 12 months of the study;
- the principal recommendation for future research is for a multi-centre clinical trial with a view to significantly increasing sample size and to extend the period of observation beyond 12 months. It should be possible to substantiate or refute the observation from this study that, following non-surgical periodontal treatment, probing depths show greater resolution in smokers who are able to quit the habit.

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Clinical Relevance

Scientific rationale for the study: The literature supports that smoking is a key risk factor for periodontitis, based on case–control studies. The rationale for conducting this study was to investigate longitudinally (12 months) the effect of quitting smoking on periodontal status when combined with non-surgical periodontal therapy.

Principal findings: A 20% quit rate was achieved in 49 smokers with periodontitis. A high number of subjects did not comply with the study protocol. No statistically significant differences were identified in mean clinical parameters between quitters, non-quitters, and oscillators (those subjects who quit and resumed smoking again). However, significantly greater probing depth reductions were observed in quitters compared with non-quitters and oscillators combined ($p < 0.05$), and quitters were more likely to demonstrate clinically relevant probing depth reductions $\geqslant 2$ and $\geqslant 3$ mm than the other groups ($p < 0.05$).

Practical implications: Smoking cessation has an additional beneficial effect in reducing probing depths over 12 months.