Systematic review of the effect of smoking on nonsurgical periodontal therapy

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Periodontitis is the result of complex interrelationships between infectious agents and host factors. Environmental, acquired, and genetic risk factors modify the expression of disease and may therefore affect the onset or progression of periodontitis (27). Among the environmental risk factors, tobacco smoking has been found to be associated with an increased prevalence and severity of periodontal disease (13, 30). It is also apparent that a disproportionately high number of people with severe periodontal disease are smokers (2, 7) and that a strong association exists between smoking and an unusual form of periodontitis that is resistant to treatment (20).

Smoking has also been implicated as a factor that reduces the effectiveness of treatment (17). It appears that smokers may respond to nonsurgical periodontal therapy less favorably than nonsmokers, especially in terms of probing depth and bone level (1, 18, 21). When the effect of the level of cigarette consumption is considered, it seems that the response to periodontal therapy is related to the amount of cigarettes smoked (16), and that previous smokers (quit-smokers) have a similar response to treatment compared to nonsmokers (4, 12, 16). However, the size of the effect on treatment response in these studies is not consistent, making it difficult to draw conclusions about the clinical significance of smoking and the effect of quitting smoking on treatment.

The mechanisms by which smoking could affect the response to periodontal treatment might be related to the altered inflammatory and immune response that has been observed in smokers (17, 19, 22) or to the persistence of pathogenic flora in smokers after treatment (11, 12).

Periodontitis represents an important health issue because it may lead to changes in appearance, impairment in function, significant pain and, finally, tooth loss, all of which may affect the quality of life (25). In addition to the impact on the individual, there is a significant impact on healthcare resources needed to manage the condition. In the USA in 1999, the expenditure on periodontal and preventive care amounted to over $14 billion (5). In England and Wales, £174 million was spent on treatment of periodontal disease by the NHS (National Health Service) in the year 2001–2002 (9).

Therefore, as a public health measure, it is critical to establish the effect of smoking on periodontal therapy. To date there has been no reliable estimate of the impact of smoking on periodontal treatment response. The aim of this systematic review was therefore to investigate the effect of smoking on nonsurgical periodontal therapy in patients with chronic periodontitis. The null hypothesis was that there is no difference between smokers and nonsmokers in their response to nonsurgical periodontal therapy. The focused question was: 'In patients with chronic periodontitis, what is the effect of smoking and smoking cessation on the response to nonsurgical periodontal therapy in terms of clinical and patient-centered outcomes?'.

Methods

Protocol development

We developed the protocol specifying all aspects of the review methods before commencing the review. These included the following: inclusion criteria for studies, search strategy, screening method, data

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abstraction, quality assessment, and data analysis. This aspect of the design was planned to minimize the effect of our possible bias on the review and, in particular, on the potential to alter the methods or analytical techniques based on study findings.

Search strategy

The search strategy involved the use of the following electronic databases: MEDLINE, EMBASE and the Cochrane Central Register of Controlled Clinical Trials (CENTRAL), as well as hand searching of bibliographies of found references, review articles and consensus statements. All databases were searched from their earliest records until March 2003; only English language publications were searched. The full search strategy is included as Appendix 1.

Study selection

The primary study design selected was controlled clinical trials as smoking habit cannot be randomized. In addition, arms of randomized controlled trials investigating the effectiveness of nonsurgical periodontal treatment that reported results separately for smokers and nonsmokers were included. Other inclusion criteria were studies that assessed systemically healthy patients who had been diagnosed with chronic or adult periodontitis and where the patient was the unit of analysis (rather than a tooth- or site-based analysis). Studies were not excluded on the basis of quality, only on whether they fulfilled the inclusion criteria for entry. We planned to investigate the impact of quality on study outcome if there was heterogeneity between studies.

Types of intervention

The intervention of interest was nonsurgical periodontal treatment, including oral hygiene instructions and scaling and root-planing or root debridement. Studies considering nonsurgical periodontal therapy as oral hygiene alone or deliberate curettage were excluded.

Types of outcomes measured

The following outcome measures were reported:
- primary outcomes: tooth loss, changes in probing pocket depth and clinical attachment level (clinical attachment level);
- secondary outcomes: changes in bleeding on probing and complications post-treatment; patient-

centered outcomes, such as quality of life, changes in appearance, and patient experience.

Validity assessment

The lead investigator (A.L.) was initially calibrated for screening against another investigator with experience of several systematic reviews (I.N.). Sixty records in batches of 20 were screened in this manner until a kappa (K) score of > 0.80 was achieved. Titles and abstracts were then screened for possible relevance by one investigator (A.L.). For all studies of possible relevance, the full text was retrieved. This was examined independently and in duplicate with a second investigator. Disagreement was resolved in all cases by discussion; the K-score for agreement was 0.75. Evaluation of studies was not masked in relation to study authors or affiliations as this has not been shown to significantly alter outcomes (23).

Study quality was assessed for the similarity between groups at baseline, the report of adjustment for confounding factors, blindness of examiner to smoking status, proportion of cohort followed up, and presence of specified eligibility criteria. The criteria were modified from a guideline for quality assessment of follow-up studies (26).

Data abstraction

The data abstraction form was piloted over 20 studies and used to abstract general information about the paper, study characteristics, outcome measures, treatment characteristics and quality assessment data. Abstraction was performed in duplicate independently. Where disagreement arose, this was resolved by discussion.

Study characteristics and quantitative data synthesis

From evidence tables, studies were analyzed for similarity in key components and suitability for meta-analysis. For the studies that could be included in the meta-analysis, the weighted mean difference was used for continuous outcomes comparing nonsmokers, smokers and quit smokers (STATA version 7). Where heterogeneity between studies existed, it was investigated using a limited number of factors thought most likely to generate differences in outcomes, including clinical and methodologic variables. These were defined *a priori*. Some studies reported mean values calculated from all sites in the mouth (full-mouth studies) including both diseased

and nondiseased sites. Other studies calculated mean values only for sites above a defined disease threshold (threshold studies); for instance, initial pocket depth ≥5 mm. Data from these two sets of studies were analyzed separately.

Results

Search (Fig. 1)

From the 330 studies initially obtained from the search, 80 full text articles were independently screened by two reviewers and the level of agreement was determined by Kappa score (K-score for full text screening: 0.57). Of the 80 full text articles screened, 67 were not relevant and were excluded and 13 were considered eligible for inclusion in the review. The most common reason for study exclusion was the lack of a control group receiving the same treatment but not exposed to smoking (58 studies). Other reasons were site-based analysis (2 studies), data for nonsurgical and surgical therapy combined (1 study), selected patients for subgroup not representative of initial sample (1 study), duplicate data (1 study) and not a clinical trail (1 paper). The characteristics of the included studies are shown in Tables 1 and 2.
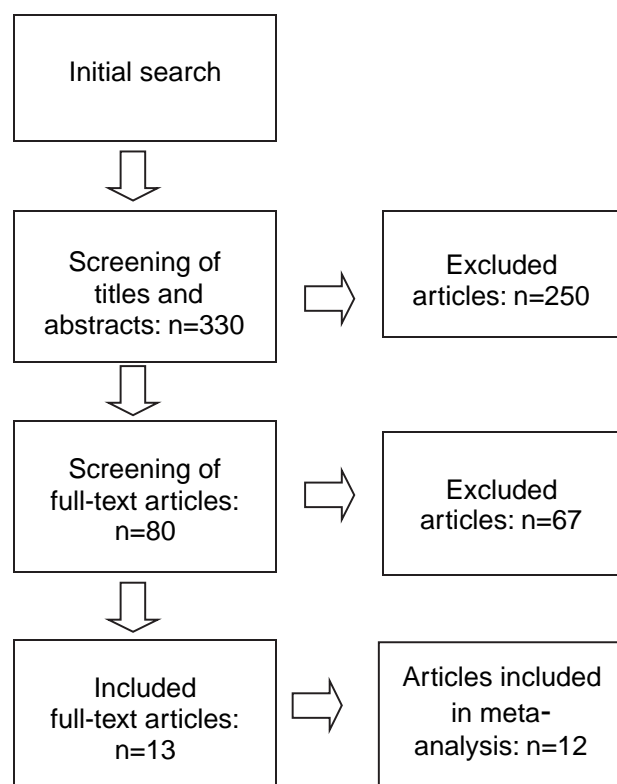

Fig. 1. Flow of articles through the review.

The quality assessment revealed that of the 13 eligible studies, seven showed a clear similarity between groups at baseline. In most other cases, baseline values were not reported. The proportion of the patients followed up was unclear in seven articles and examiner blinding to smoking status was unclear in most studies (11 articles). Potential confounding factors were listed in 11 studies, but only five studies reported making an adjustment for these factors. Three of the studies evaluated all sites in the mouth, seven studies only sites above a certain threshold of baseline probing depth, and three studies both full-mouth and deeper sites.

The heterogeneity between studies was investigated using meta-analysis regression. No statistically significant difference between studies was found after adjustment for baseline values and the duration of follow-up. Similarly, no statistically significant difference was found when considering whether or not the studies were adjusted for baseline values, suggesting a reasonable similarity between groups.

Primary outcomes (Table 3)

Tooth loss

No studies reported data on tooth loss.

Probing depth reduction in smokers compared to nonsmokers

All sites. The difference in full-mouth probing depth reduction after nonsurgical therapy between smokers and nonsmokers was assessed in six studies, of which five showed a better response in nonsmokers, although the difference was small (Fig. 2). The results showed a mean difference in probing depth reduction of 0.133 mm (95%CI [0.038,0.227], $P = 0.006$) with a chi-squared value for heterogeneity of 7.69 (5 df, $P = 0.18$), i.e. the reduction in probing depth was 0.133 mm greater in nonsmokers than in smokers and there was no evidence to suggest that the studies were dissimilar in their estimates of this result (no evidence of heterogeneity, $P > 0.05$).

Only sites with an initial probing depth of 5 mm. A separate analysis was undertaken for the 'threshold' studies, evaluating only sites with an initial probing depth of ≥5 mm. Eight out of nine available studies were included. One study (31) could not be included in the meta-analysis because it was not comparable with the others, due to an upper limit for probing depth of experimental sites (i.e. only sites 4–6 mm were evaluated). A random effects meta-analysis indicated a weighted mean difference in probing

Table 1. Characteristics of included studies

1st Author/year (ref. no.)	No. of smokers	No. of nonsmokers	No. of treatment sessions	Duration of treatment sessions (h)	Follow-up (months)	Experimental sites
Preber & Bergstrom (31)	40	35	mean: 7.8	1 h/session	1	Initial probing depth: 4–6 mm
Palmer et al. (28)	n.r.[a]	n.r.	2	3 h	6	Initial probing depth: ≥5 mm
Grossi et al. (12)	60	28	4–6	n.r.[a]	3	Full-mouth and initial probing depth: ≥5 mm
Machtei et al. (21)	n.r.	n.r.	4	n.r.	15	Full-mouth
Williams et al. (38)	91	159	1	n.r.	9	Initial probing depth: ≥5 mm
Haffajee et al. (14)	n.r.	n.r.	4	3–4 h	9	Full-mouth
Pucher et al. (34)	38	59	1	1 h	9	Initial probing depth: ≥5 mm
Preshaw et al. (33)	15	12	up to 4	up to 4 h	6	Full-mouth and test sites (8 × subject)
Preber et al. (32)	17	15	6–8	n.r.	2	Full-mouth and 1 site with initial probing depth: ≥5 mm
Winkel et al. (39)	32	17	3–6	1 h/session	6	Full-mouth
Ryder et al. (36)	61	48	2	n.r.	9	Initial probing depth: ≥5 mm
Renvert et al. (35)	13	15	n.r.	n.r.	6	Initial probing depth: ≥6 mm
Mongardini et al. (24)	5	7	4	n.r.	8	Initial probing depth: ≥7 mm

[a]n.r., not recorded.

Table 2. Quality of included studies

1st Author/year (ref. no.)	Similarity between groups at baseline	Confounding factors		Examiner blind to smoking status	Proportion followed-up
		Listed	Adjusted		
Preber & Bergstrom (31)	Yes	Yes	Unclear	Unclear	100%
Palmer et al. (28)	Yes	Yes	Yes	Unclear	Unclear
Grossi et al. (12)	Yes	Yes	Yes	Unclear	Unclear
Machtei et al. (21)	Unclear	Yes	No	Unclear	Unclear
Williams et al. (38)	Unclear	Yes	Unclear	Unclear	Unclear
Haffajee et al. (14)	No	Yes	No	Unclear	100%
Pucher et al. (34)	Yes	Yes	No	Yes	87–91%
Preshaw et al. (33)	Unclear	No	No	Unclear	Unclear
Preber et al. (32)	Unclear	Yes	Yes	No	100%
Winkel et al. (39)	Yes	Yes	Yes	Unclear	Unclear
Ryder et al. (36)	Unclear	No	No	Unclear	85–94%
Renvert et al. (35)	Yes	Yes	Unclear	Unclear	100%
Mongardini et al. (24)	Yes	Yes	Yes	Unclear	Unclear

Table 3. Meta-analysis of differences in treatment effect in the smoking groups

Smoking groups	Variable	Probing depth category	Pooled estimate	95% CI		P-value for estimate	P-value or hetero-geneity	Effects	Studies (ref. nos.)
				Lower	Upper				
S vs. NS	Probing depth reduction	Full mouth	0.133	0.038	0.227	0.006	0.180	Fixed	(12, 14, 21, 32, 33, 39)
S vs. NS	Probing depth reduction	≥5 mm	0.433	0.235	0.631	< 0.001	0.009	Random	(12, 24, 28, 32, 34–36, 38)
QS vs. NS	Probing depth reduction	Full mouth	−0.016	−0.117	0.085	0.753	0.728	Fixed	(12, 14, 33)
QS vs. NS	Probing depth reduction	≥5 mm	0.130	−0.340	0.600	0.588	0.005	Random	(12, 36)
S vs. NS	Clinical attachment level gain	Full mouth	0.114	−0.021	0.249	0.097	0.996	Fixed	(12, 14, 21, 39)
S vs. NS	Clinical attachment level gain	≥5 mm	0.116	−0.047	0.278	0.164	0.337	Fixed	(12, 24, 28, 34–36)
QS vs. NS	Clinical attachment level gain	Full mouth	−1.059.	−4.027	1.910	0.485	< 0.001	Random	(12, 14)
QS vs. NS	Clinical attachment level gain	≥5 mm	1.340	0.654	2.025	< 0.001	0.006	Random	(12, 36)
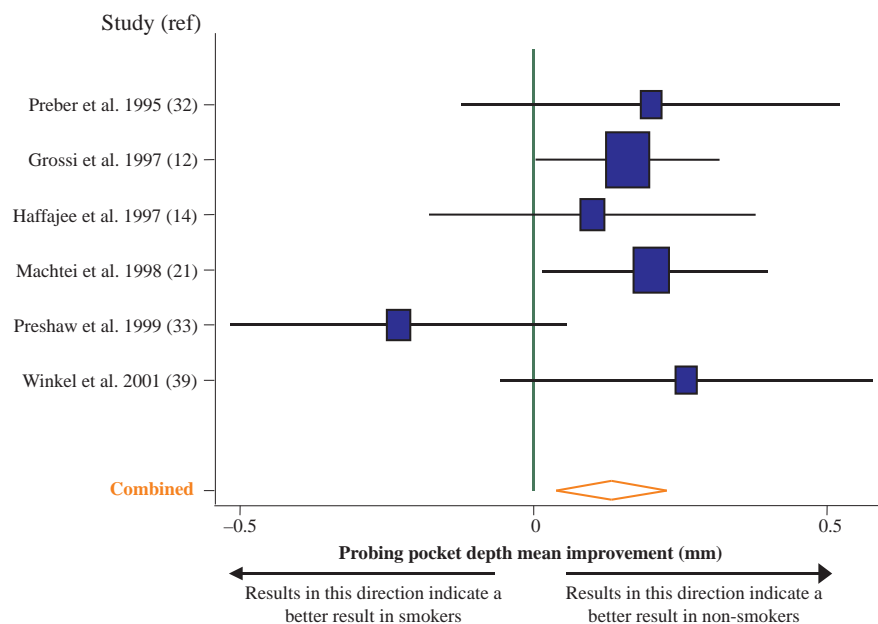
Note: Each study is represented by a blue box with a black horizontal line. The centres of the boxes represent each study's mean estimate of the difference in PD change between smokers and non-smokers. The size of each box is proportional to the weight given to each study when calculating the combined (pooled) estimate. The lengths of the horizontal bars represent the 95% confidence intervals for each study's mean estimate. The orange diamond represents the pooled estimate of the mean difference from all the studies combined. The centre of the diamond is the point estimate and the left and right tips are the 95% confidence interval for the pooled estimate. The green vertical line represents the position at which there would be no difference in improvement between smokers and non-smokers. Thus any confidence intervals that cross the green line indicate no statistically significant difference between smokers and non-smokers.

Fig. 2. Forest plot of mean difference in probing pocket depth reduction between smokers and non-smokers (all sites).

depth reduction of 0.433 mm, favoring nonsmokers (95%CI: [0.235,0.631], $P < 0.001$; chi-squared test for heterogeneity 18.666, 8 df, $P = 0.009$) (Fig. 3). The highly significant heterogeneity suggests that these studies are not similar in their estimate of the result. Because of limitations in reporting within the original
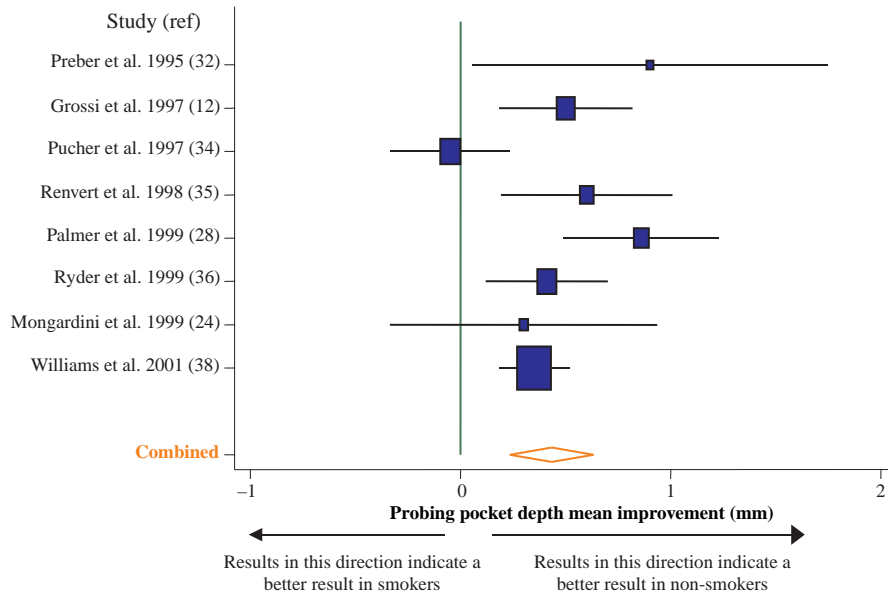
Fig. 3. Forest plot of mean difference in probing pocket depth reduction between smokers and non-smokers ('threshold' studies).

publications, it was not possible to account for the differences. As a cautious observation, it is clear that, with one exception (34), all studies produced a summary estimate favoring nonsmokers, although the difference in Mongardini et al. (24) was not statistically significant. It is therefore possible that unreported differences in characteristics between these studies might account for these differences in outcomes.

Probing depth reduction in quit-smokers compared with nonsmokers

All sites (Fig. 4). Only five of the included studies assessed the response of quit-smokers to nonsurgical therapy (12, 14, 21, 33, 36). Among these, three could be included in the meta-analysis for full-mouth

evaluation, as one (21) did not report results for quit-smokers and another (36) considered only sites initially ≥5 mm. Fixed effects meta-analysis showed no statistically significant difference between quit-smokers and nonsmokers (−0.016 mm, 95%CI [0.117,0.085], $P = 0.753$) and no significant heterogeneity (chi-squared test for heterogeneity 0.636, 2 df, $P = 0.728$).

Only sites with an initial probing depth of ≥5 mm (Fig. 5). Three threshold studies comparing quit-smokers and nonsmokers were found (12, 21, 36). However, one (21) was not eligible to be included in the meta-analysis because results were not reported for quit-smokers. The difference was not statistically significant by random effects meta-analysis (0.130 mm, 95%CI [−0.340,0.600], $P = 0.588$; chi-
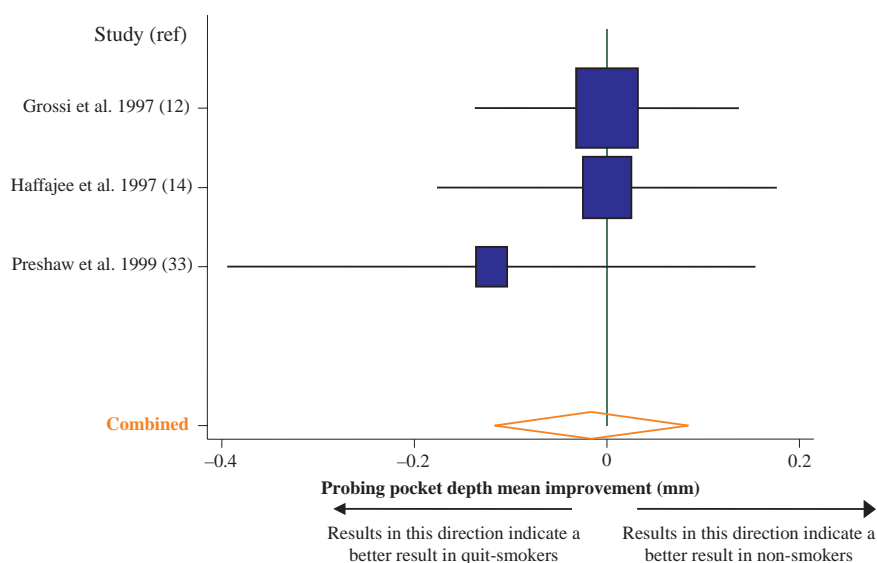

Fig. 4. Forest plot of mean difference in probing pocket depth reduction between quit-smokers and non-smokers (all sites).
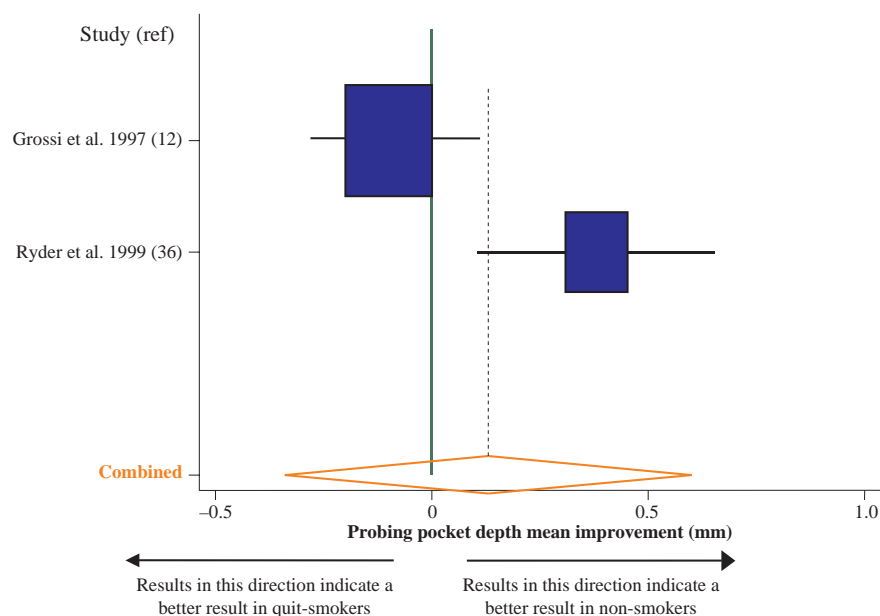
Fig. 5. Forest plot of mean difference in probing pocket depth reduction between quit-smokers and non-smokers ('threshold' studies).
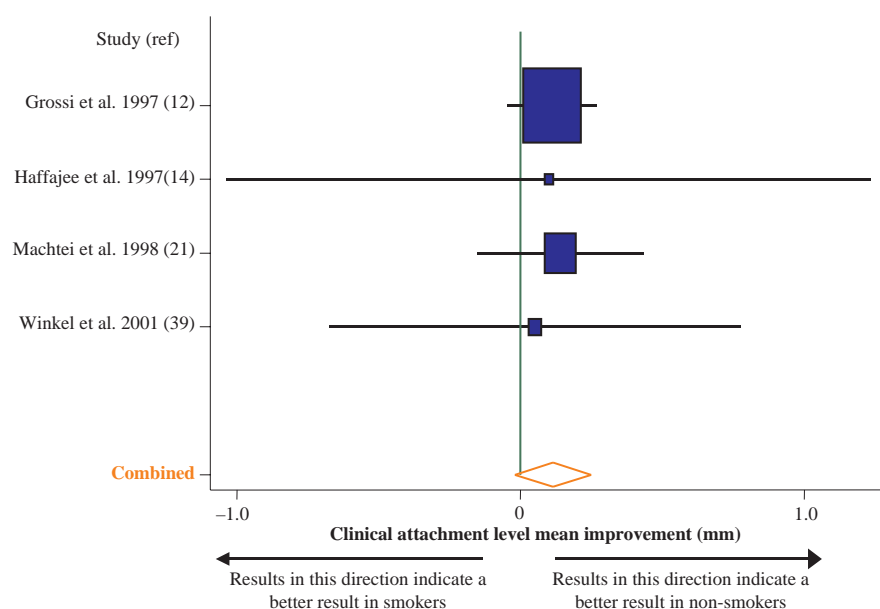
Fig. 6. Forest plot of mean difference in clinical attachment level gain between smokers and non-smokers (all sites).

squared test for heterogeneity 7.784, 1 df, $P = 0.005$). However, the heterogeneity between studies indicates that it may not be appropriate to pool the studies into a single overall estimate.

Clinical attachment level gain in smokers compared to nonsmokers (Figs 6 and 7)

Four studies could be included in the meta-analysis of the difference in clinical attachment level gain between smokers and nonsmokers after nonsurgical periodontal therapy. No statistically significant difference was found between the two study groups (0.114 mm, 95% CI [−0.021,0.249], $P = 0.097$; chi-square for heterogeneity 0.063, 3 df, $P = 0.996$). For sites with an initial probing depth of ≥5 mm (six

studies), the difference in clinical attachment level gain between smokers and nonsmokers was not statistically significant (0.116 mm, 95%CI [−0.047, 0.278], $P = 0.164$; chi-squared test for heterogeneity 5.699, 5 df, $P = 0.337$).

Clinical attachment level gain in quit-smokers compared to nonsmokers

All sites (Fig. 8). Two studies were available to examine the difference in clinical attachment level change between quit- and nonsmokers. A random effects meta-analysis showed no statistically significant difference between quit- and nonsmokers in terms of full-mouth clinical attachment level gain (difference in clinical attachment level gain: 1.06 mm
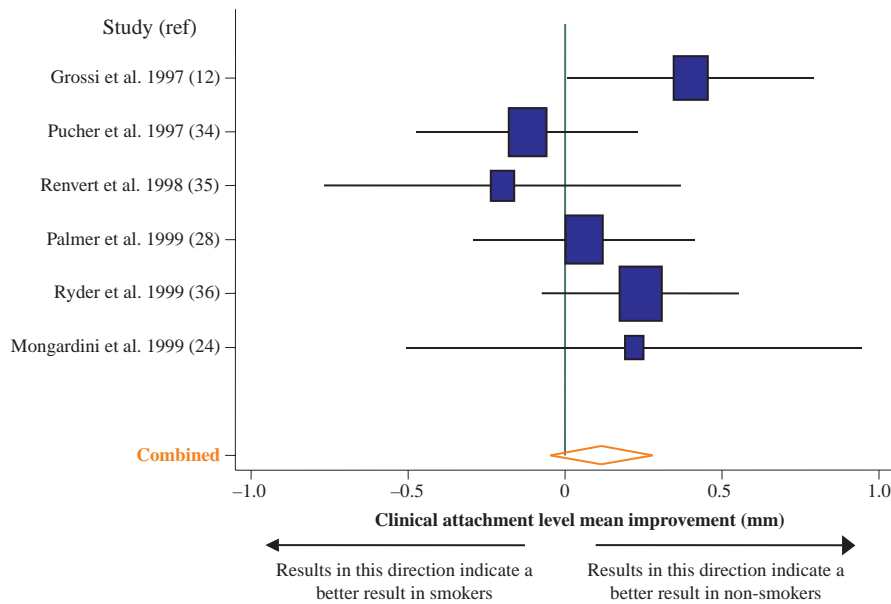
Fig. 7. Forest plot of mean difference in clinical attachment level gain between smokers and non-smokers ('threshold' studies).
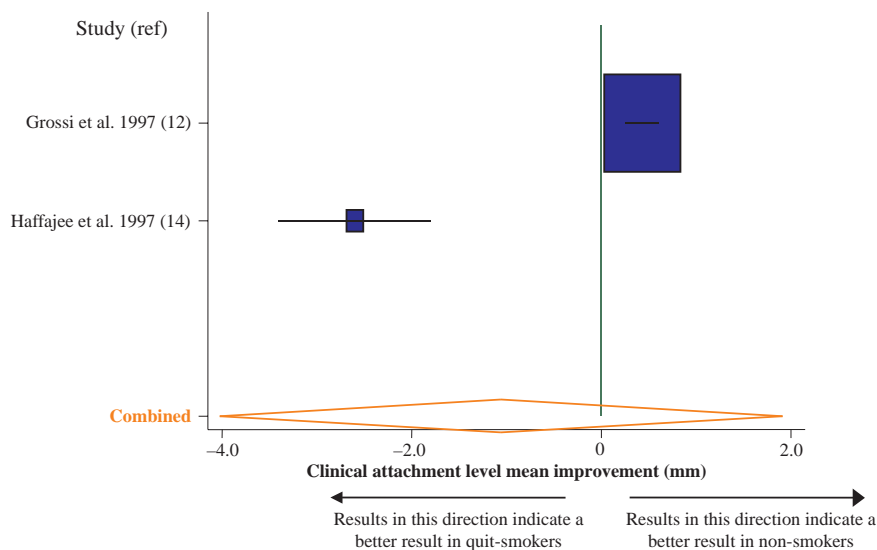

Fig. 8. Forest plot of mean difference in clinical attachment level gain between quit-smokers and non-smokers (all sites).

in favor of quit-smoking group: 95%CI [−4.027,1.910], $P = 0.485$; chi-squared test for heterogeneity 52.105, 1 df, $P < 0.001$) with highly statistically significant heterogeneity ($P < 0.001$).

Only sites with an initial probing depth of ≥5 mm (Fig. 9). The meta-analysis of the two studies comparing the change in clinical attachment level between quit-smokers and nonsmokers in sites with an initial probing depth of ≥5 mm showed a difference in clinical attachment level gain of 1.34 mm, favoring the nonsmokers (95% CI [0.654,2.025], $P < 0.001$; chi-squared test for heterogeneity 7.470, 1 df, $P = 0.006$). In both of these analyses, the degree of heterogeneity suggests that it is not appropriate to pool the results as the studies appear to be estimating different results.

Investigating the heterogeneity between the threshold studies

The reports of the studies included sufficient information to investigate the effects of two of the *a priori* defined potential sources of heterogeneity. Differences between smokers and nonsmokers in baseline disease severity were available for seven of the eight studies (not for [38]). Meta-analysis regression indicated no evidence that this influenced the pooled estimate (change in estimate per 1 mm change in baseline difference = 0.523, 95%CI [− 0.099,1.145]). Seven of eight studies (not [35]) could be utilized to investigate the effect of the number of sessions of treatment on the outcome. These also provided no evidence that differences in this factor between
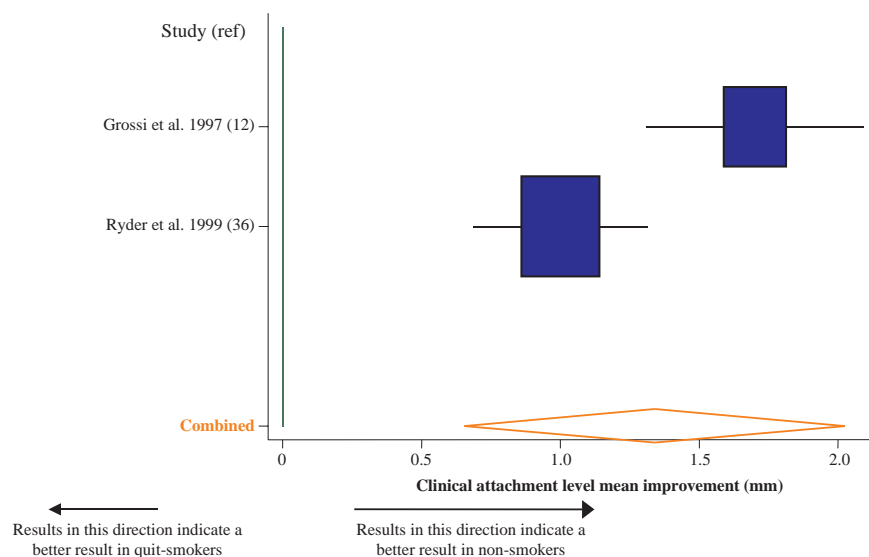
Fig. 9. Forest plot of mean difference in clinical attachment level gain between quit-smokers and non-smokers ('threshold' studies).

Discussion

studies were a source of heterogeneity in the pooled estimate (change in pooled estimate per additional session of treatment = 0.079, 95%CI [−0.048, 0.020]).

Secondary outcomes

Bleeding was assessed after therapy in seven studies. However, due to great heterogeneity in the methods used to of assess bleeding, it was decided not to perform a meta-analysis and data are reported for each study individually in Table 4. Of these seven studies, one (33) reported bleeding on probing results only on graphs and it was not possible to extrapolate the data because of the small scale. No statistically significant differences in bleeding were found between smokers and nonsmokers either at baseline or after therapy in most of the studies. However, one study (12) found significantly less bleeding in smokers than in nonsmokers at baseline and another (35) found a reduced response in terms of bleeding in smokers compared to nonsmokers. In the two studies evaluating the change in bleeding in quit-smokers also (12, 36) no statistically significant difference was found after treatment.

Patient-centered outcomes

No data were reported for any of the included studies on patient-centered outcomes such as quality of life, ease of maintenance, changes in aesthetic appearance, or patient experience.

This systematic review has shown that smoking can have a negative effect on mechanical nonsurgical periodontal therapy as indicated by less probing depth reduction in smokers than in nonsmokers. Whereas the analysis for sites with an initial probing depth of at least 5 mm indicated statistical heterogeneity, a glance at the forest plot (Fig. 3) demonstrates that six out of the eight studies had outcomes that statistically significantly favored nonsmokers and the other two studies had outcomes showing no statistically significant difference between smokers and nonsmokers. Therefore, although the degree of heterogeneity is troubling, the most likely conclusion is that smoking decreases the effect of probing depth reduction.

Data were only available to explore the heterogeneity in terms of differences in baseline disease severity between smokers and nonsmokers and the number of sessions of treatment that patients received. Neither of these factors significantly influenced the outcome of the studies. Nevertheless, this should not be taken to mean that the impact of smoking is unclear. Instead, we would interpret this finding as indicating that smoking very likely affects the treatment response, but that the size of the effect remains uncertain.

It is not possible to reject the null hypothesis of no difference between smokers and nonsmokers for all outcomes. There were no statistically significant differences in the change in clinical attachment level between smokers and nonsmokers either when

Table 4. Bleeding: mean baseline, final and change (mm) for different smoking groups by study

Study (ref. no.)	Experimental sites	Bleeding index	Smokers			Nonsmokers			Quit-smokers		
			Baseline	Final	Change	Baseline	Final	Change	Baseline	Final	Change
Renvert et al. (35)	Full-mouth	Bleeding on probing (%)	63.0 (SD 21.3)	36.5 (SD 19.9)	−26.5 (SD 13.7)	53.0 (SD 24.3)	22.7 (SD 12.3)	−30.9 (SD 24.3)	n.a	n.a.	n.a.
Ryder et al. (36)	Full-mouth	Bleeding index	1.54 (SE 0.05)	n.r.	n.r.	1.55 (SE 0.05)	n.r.	n.r.	1.68 (SE 0.05)	n.r.	n.r.
	≥5 mm				−0.57 (SE 0.06)			−0.54 (SE 0.07)			−0.52 (SE 0.06)
Winkel et al. (39)	Full-mouth	Bleeding index	0.77 (SD 0.13)	0.41 (SD 0.11)	n.r.	0.8 (SD 0.16)	0.37 (SD 0.13)	n.r.	n.a.	n.a.	n.a.
Palmer et al. (28)	≥5 mm	Bleeding on probing (%)	21.4 (SD 9.4)	12.1 (SD 6.9)	47.4% (SD 12.6)	24.2 (SD 18.7)	18.2 (SD 16.3)	28.2% (SD 29.6)	n.a.	n.a.	n.a.
Pucher et al. (34)	≥5 mm	Bleeding on probing (0 no, 1 yes)	1.00 (SD 0.09)	0.67 (SD 0.39)	n.r.	1.00 (SD 0.07)	0.78 (SD 0.30)	n.r.	n.a.	n.a.	n.a
Grossi et al. (12)	Full-mouth	Bleeding index	0.23 (SE 0.02)	0.11 (SE 0.05)	n.r.	0.37 (SE 0.04)	0.18 (SE 0.05)	n.r.	0.38 (SE 0.03)	0.16 (SE 0.05)	n.r.

n.r., not reported. n.a., not applicable. SD, standard deviation. SE, standard error.

studies considered full mouth sites or when they considered only initially deeper sites.

We could speculate that the finding of a difference between smokers and nonsmokers with respect to probing depth and not clinical attachment level could be explained, at least in part, by a reduced level of edema in the periodontal tissues of smokers at baseline. The increased vasoconstriction of peripheral blood vessels observed in smokers has been related to reduced bleeding and edema in periodontal patients who smoke, compared to nonsmokers (3, 8). If this is generally the case, smokers would have less potential for resolution of inflammation and edema within the marginal tissues and therefore less potential for gingival recession. Thus, there could be a decrease in probing depth reduction, but no difference in clinical attachment level change.

Possible sources of heterogeneity between studies in the size of treatment effect were investigated by subgroup analysis and meta-analysis regression. One possible source of heterogeneity could be related to differences in initial disease severity, since previous studies have shown that clinical outcomes of both nonsurgical and surgical periodontal therapy are related to the initial attachment level and probing depth (6, 15, 29). However, meta-analysis regression failed to detect an effect of initial defect depth or duration of follow-up on the difference in probing depth reduction and clinical attachment level gain after nonsurgical therapy in smokers, nonsmokers and quit-smokers. No statistically significant difference was found when considering whether or not the studies were adjusted for baseline values, suggesting a reasonable similarity between groups. Other factors that were initially planned to be investigated could not be assessed due to missing data. These factors included plaque level and tooth type.

When the difference in response to nonsurgical treatment between quit-smokers and nonsmokers was assessed, the data were not consistent. Regarding probing depth change, the data from studies including all sites regardless of initial probing depth suggest no difference between groups. For studies done only on pockets initially 5 mm and deeper, one study showed no statistically significant difference and one indicated greater pocket depth reduction in nonsmokers. For clinical attachment level gain, in the meta-analysis for studies on all sites, one study favored quit smokers and one study nonsmokers. For initially diseased sites only, both studies favored nonsmokers over quit-smokers, although the sizes of the difference between the two studies was quite different. Possible causes of the differences between

these two studies are treatment characteristics and the definition of smoking. Four to six sessions of scaling and root-planing were performed in one of the studies (12) and outcomes assessed at 3 months, whereas only two sessions were performed in the other study (36) and the patients were reevaluated at 9 months. The definition of smokers was also different. Any smoker was included in one study (12), whereas only subjects smoking 10 cigarettes or more per day were selected in the other (36).

Clearly, the validity of drawing conclusions about the early effects of quitting smoking from the available data is questionable. This is due partly to the limited number of studies and partly to differences in their outcomes. Therefore, the data on the effect of quitting smoking (compared with nonsmokers) is currently and perhaps surprisingly inconclusive. This is an area that should be a high priority for future research.

Secondary clinical outcomes such as tooth loss and complications post-treatment were never reported. On the other hand, changes in bleeding after therapy were reported in about half of the included studies (six of 13). The great variability in the methods of assessing bleeding did not allow us to perform the meta-analysis. However, it is apparent that most studies did not find a difference between smokers and nonsmokers with respect to this. All but one study (12) reported no statistically significant difference in bleeding in smokers, nonsmokers and quit-smokers at baseline. Similarly, no significant differences between groups were found after treatment. Only one study (35) found a statistically significant difference between smokers and nonsmokers in terms of a change in bleeding after therapy ($P < 0.05$).

Limitations

One recurring problem in this review was the variability (or complete absence) of definitions of smoking status. In addition, no study verified self-reported smoking status with biochemical measures such as salivary cotinine or exhaled carbon monoxide. Self-reported history may not be a reliable method to assess smoking exposure; biochemical tests to measure serum levels of metabolites of nicotine should be used instead (10). We would recommend that future studies investigate the utility of biochemical measures of smoking exposure in periodontal therapy. We have such a study in progress and hope to report the results soon.

A further limitation was the lack of data on tooth loss. This meant that we had to rely on surrogate

measures such as change in probing depth and clinical attachment level. Capturing the impact of smoking on tooth loss would require follow-up periods lasting several years and such studies are difficult to conduct. Rigorous observational studies could provide such data and could also examine the effect of smoking on additional treatment needs to secure oral health. Such data would be valuable to estimate the impact of this impaired treatment response.

Limiting the search to English language studies could have introduced a selection bias. However, no non-English studies were identified despite the search of EMBASE, which has a greater coverage of non-English journals.

Clinical implications

The reduction in the effectiveness of nonsurgical periodontal treatment in periodontal patients indicates that smoking cessation therapy should be offered to smokers requiring such treatment. Smoking cessation interventions can be successful in the dental setting (37) but may require further training and resources. Although this review has not investigated the impact of smoking on future periodontal treatment needs, other data also suggest that the recurrence of disease is a greater problem for smokers. Thus, proper consent to treatment for smokers with periodontal disease should include this information.

The further aim of this review was to assess the effect of smoking on the response to nonsurgical treatment in terms of patient-centered outcomes such as quality of life, ease of maintenance, changes in aesthetic appearance, and patient satisfaction. However, no data on these outcomes were found in any of the included studies.

Implications for future research

Studies evaluating the effect of smoking on treatment response should be based on reliable methods of assessing smoking exposure, in place of patient-reported data. These methods include the assessment of salivary or serum levels of metabolites of nicotine, such as cotinine, and the measurement of exhaled carbon monoxide. Such objective measures are needed to investigate the impact of quitting smoking on treatment outcomes. More emphasis should also be given to the difference in the long-term response to periodontal therapy in smokers, nonsmokers and quit-smokers. In this respect, a useful outcome measure could be tooth loss.

Conclusions

- Following nonsurgical periodontal therapy, people who smoke will experience less reduction in probing depth than nonsmokers. There is no evidence of a difference in gain in clinical attachment between smokers and nonsmokers or a reduction of bleeding on probing between smokers and nonsmokers. Differences in study design and lack of data precluded an adequate and complete pooling of data for a more comprehensive analysis.
- In short-term studies, it is unclear whether people who quit smoking will respond as favorably to nonsurgical therapy to those who have always been nonsmokers.
- Progress in understanding the effects of smoking on periodontal therapy will require the evaluation of objective measures of smoking exposure such as cotinine and exhaled carbon monoxide in place of sole reliance on patient reported information.

Declaration of interest

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Appendix 1

Search strategy

1 exp PERIODONTITIS/th [Therapy]

2 periodontal therapy.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

3 periodontal treatment.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

4 initial periodontal therapy.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

5 mechanical periodontal therapy.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

6 non surgical periodontal therapy.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

7 non surgical periodontal treatment.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

8 dental scaling.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

9 exp Dental Scaling/

10 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9

11 NICOTINE/

12 smok$.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

13 smoking cessation.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

14 previous smokers.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

15 former smokers.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

16 SMOKING/or exp SMOKING CESSATION/

17 TOBACCO/or exp 'TOBACCO USE CESSATION'/

18 cigarette smoking.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

19 cigarettes.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

20 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19

21 exp Gingival Pocket/th [Therapy]

22 Periodontal Attachment Loss/th [Therapy]

23 Periodontal Pocket/th [Therapy]

24 Periodontal Diseases/th [Therapy]

25 Dental Plaque/th [Therapy]

26 prophylaxis.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

27 planing.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

28 'Root Planing'/

29 planing.ab. or planing.in. or planing.ti.

30 debridement.mp. [mp = title, abstract, cas registry/ec number word, mesh subject heading]

31 DEBRIDEMENT/or debridement.mp.

32 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29 or 30 or 31

33 10 or 32

34 20 and 33