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Review

The effect of smoking on periodontal treatment response: a review of clinical evidence

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Abstract

Background: Smoking has been identified as a significant risk factor for periodontal diseases and is regarded as being responsible for incomplete or delayed healing in patients following treatment.

Aim and Method: The aim of this conventional review was to review, collate and tabulate the relative effectiveness of treatments of chronic periodontitis in smokers, non-smokers and ex-smokers.

Observations: The majority of clinical trials show significantly greater reductions in probing depths and bleeding on probing, and significantly greater gain of clinical attachment following non-surgical and surgical treatments in non-smokers compared with smokers. This benefit is also seen at class I and II furcation sites and in patients prescribed systemic or local antimicrobial treatments.

Conclusions: Data from epidemiological, cross-sectional and case–control studies strongly suggest that quitting smoking is beneficial to patients following periodontal treatments. The periodontal status of ex-smokers following treatment suggests that quitting the habit is beneficial although there are only limited data from long-term longitudinal clinical trials to demonstrate unequivocally the periodontal benefit of quitting smoking.

Key words: periodontitis; risk factors; smoking

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Smoking is undoubtedly one of the main, and most prevalent, risk factors for chronic periodontitis; risk calculations suggest that 40% of cases of chronic periodontitis may be attributable to smoking, with an increased odds ratio (OR) of 5.4 for chronic periodontitis in smokers (Brothwell 2001). Indeed, Haber has described a discrete, smoking-specific disease entity – smoking associated periodontitis – that is characterized by fibrotic gingiva, limited gingival redness and oedema relative to disease severity, proportionally greater pocketing in anterior and maxillary lingual sites, gingival recession at anterior sites and a lack of association between periodontal status and the level of oral hygiene (Haber 1994).

The majority of contemporary studies suggest that smoking increases the risk of periodontal diseases between two and six times. For example, Calsina 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 (Calsina et al. 2002). Linden found the OR for periodontal disease to be as high as 14.1 in young smokers (Linden & Mullally 1994) whereas Hyman 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 (Hyman & Reid 2003).

More recently, Bergström (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.

Evidence of risk factor status is strengthened by the ability to demonstrate a dose–response, and ''years of exposure'' to tobacco products is a

statistically significant risk factor for periodontal disease (Grossi et al. 1995, Martinez-Canut et al. 1995). It remains challenging, however, to determine the strength of smoking as a risk factor owing to inherent problems in measuring accurately a subject's exposure to tobacco (Scott et al. 2001, Molloy et al. 2004, Persson et al. 2005).

In general, therefore, there is a substantial body of evidence to support the observation that the more a patient smokes, the greater the degree of periodontal disease. The logical corollary then is that treatments for periodontal disease are likely to be more efficacious in non-smokers than in smokers, with the response of ex-smokers being intermediate between these two groups.

The principal objective of this paper is, therefore, to review the evidence for the relative clinical responses to periodontal treatment in smokers, non-smokers and ex-smokers.

The Clinical Response of Smokers and Non-Smokers to Periodontal Treatment

A comprehensive review of the main outcome data from studies that have compared the effectiveness of treatment of periodontitis in smokers, non-smokers and ex-smokers is presented in Table 1.

Without exception, these are case–control, cross-sectional and parallel group studies primarily of smokers and non-smokers following different modalities of periodontal treatment. It must also be highlighted that the majority of the studies are relatively short-term in duration (less than 1 year follow-up), and it is therefore not possible to establish whether some of the clinically and significant differences seen between smokers and non-smokers in particular will be maintained in the longer term. It is also important to note that although non-smokers universally respond better to periodontal treatment than do smokers, there is nevertheless substantial evidence of clinical improvement in smokers after treatment, indicating that smoking as a risk factor will compromise rather than prevent tissue healing.

With non-surgical therapy as the main treatment modality, most authors report greater reductions in probing depth in non-smokers compared with smokers (Preber & Bergström 1985, Preber et al. 1995, Grossi et al. 1997, Renvert et al. 1998, Preshaw et al. 1999, Jin et al. 2000). The magnitude of the differences in shorter term studies (1–3 months) tend to be of the magnitude of 0.1–0.3 mm and therefore of doubtful clinical significance (Preber & Bergström 1985, Preber et al. 1995, Grossi et al. 1997). Jin et al. (2000), however, reported significantly greater reductions of the order 1.0 mm in non-smokers compared with smokers at 1 and 3 months following non-surgical therapy. Further, Papantonopoulos (1999) noted that between 6 and 8 weeks following non-surgical therapy, significantly more smokers (42.8%) than non-smokers (11.5%) required further treatment and the smokers may have benefitted from a surgical approach in the first instance (Papantonopoulos 1999).

Not all studies, however, have shown unequivocally a more effective response in non-smokers compared with smokers. Pucher et al. (1997) reported that smokers and non-smokers responded similarly to non-surgical therapy after 9 months with reference to reduction in probing depth, attachment level gain and reduction in bleeding on probing. Only non-smokers, however, showed a significant improvement in gingival index after 9 months compared with baseline. Further, in their post-non-surgical treatment evaluation of 12 smokers and 14 non-smokers, Zuabi et al. (1999) reported no difference in post-treatment probing depth and clinical attachment level between smokers and non-smokers. There was, however, significantly more plaque in smokers compared with the non-smokers and the smokers had significantly greater probing depths at baseline compared with the non-smokers. Consequently, the greater probing depth reduction (0.81 mm) in smokers compared with non-smokers (0.5 mm) will itself have been a direct consequence of the greater depth of pocketing in smokers before treatment.

In general terms, there appears to be a sustained benefit in both probing depth, reduction and gain of attachment level in longer term studies (Kaldahl et al. 1996, Renvert et al. 1998, Preshaw et al. 1999, Bergström et al. 2000, Jin et al. 2000). The number of studies with more than 12 months follow-up, however, is both small and disappointing. In a radiographic study, Meinberg et al. (2001) reported significantly more bone loss after 12 months follow-up in smokers compared with non-smokers and concluded that more longer term studies are essential in order to identify the association between smoking status and outcome variables (Meinberg et al. 2001).

Although the majority of clinical trials have recruited patients with chronic periodontitis, Darby et al. (2005) reported a significantly greater probing depth reduction in non-smokers with aggressive periodontitis (2.4 mm) compared with patients with aggressive periodontitis who smoke (1.3 mm). In each case, the magnitude of probing-depth resolution was greater than that seen in those patients with chronic periodontitis at 6 weeks post-scaling and root instrumentation (Darby et al. 2005).

The clinical benefit seen in non-smokers following non-surgical therapy has also been observed following surgical treatment (Preber & Bergström 1990), Modified Widman flap procedures (Ah et al. 1994), and surgical management of class I and II furcation defects (Trombelli et al. 2003). The most impressive report of clinical attachment gain in non-smokers (5.2 mm) compared with smokers (2.1 mm) was observed by Tonetti et al. (1995) who carried out guided tissue regeneration of infrabony defects using Gore-Tex® (Gore Medical Products, Newark, DE, USA) membranes and with a follow-up period of 1 year. They also concluded that higher plaque levels that are seen consistently in smokers compared with non-smokers will also have influenced the clinical outcomes (Tonetti et al. 1995).

A number of authors have also reported clinical outcomes in smokers and non-smokers following non-surgical treatment with, or without either systemic or locally delivered antimicrobial therapy (Kinane & Radvar 1997, Palmer et al. 1999, Ryder et al. 1999, Soder et al. 1999, Tomasi & Wennström 2004, Preshaw et al. 2005a). Again, smokers demonstrated clinical benefit post-treatment, both with respect to probing depth and attachment level outcomes, although the magnitude of improvement is consistently less than that seen in non-smokers. In a 9-month, placebo-controlled, randomized trial in which smokers and non-smokers were treated by scaling and root planing with and without sub-antimicrobial doxycyclin (Periostat™, Collagenex Pharmaceuticals Inc., Newtown, PA, USA), Preshaw et al. (2005a) noted with respect to probing depth reduction and clinical attachment gain a hierarchical treatment response of the order non-smokers + doxycyclin – smokers + doxycyclin – non-smokers + placebo – smokers + placebo. They con-

Table 1. A review of studies that have compared the effectiveness of treatment of chronic periodontitis in smokers, non-smokers and ex-smokers

Study	Treatment	Interval of follow-up	Smokers	Non-smokers	Ex-smokers	Conclusions
Preber & Bergström (1985)	Non-surgical therapy	1 month	Mean PD reduction of 1.1 mm	Mean PD reduction of 1.2 mm		Non-surgical therapy can reduce PD in smokers and non-smokers. However, compared with non-smokers, smokers have less reduction of PD for the dentition as a whole
Preber & Bergström (1986)	Non-surgical therapy	1 month	Mean PI reduction 0.46 Mean BOP reduction 14.4%	Mean PI reduction 0.24 Mean BOP reduction 26.8%		Compared with non-smokers, the reduction in bleeding was less pronounced in smokers in spite of greater reduction of plaque index
Preber & Bergström (1990)	Periodontal surgery	1 year	Mean PD reduction of 0.76 mm	Mean PD reduction of 1.27 mm		The difference in PD reduction between smokers and non-smokers was statistically significant and independent of plaque after 12 months ($p<0.001$). Smoking may interfere with the therapeutic outcome following periodontal surgery either through interference with primary healing events or considered as evidence of recurring disease
Ah et al. (1994)	Non-surgical therapy and modified Widman surgery	4, 10 weeks, and yearly for 6 years	0.5 mm less mean attachment gain and PD reduction; 0.6 mm greater attachment loss relative to non-smokers	Significantly greater recession in deeper ($\geqslant$7 mm) pockets		Sites in smokers do not respond as well to non-surgical or surgical therapy as those in non-smokers
Tonetti et al. (1995)	Guided tissue regeneration (Gore-Tex®) of deep infra-bony pockets	1 year	2.1 mm mean attachment gain	5.2 mm mean attachment gain		Cigarette smoking is associated with a reduced healing response after GTR treatment although consistently higher plaque levels in smokers will also have influenced outcomes.
Preber et al. (1995)	Non-surgical therapy	2 months	Mean PD reduction 0.9 mm 40% of diseased sites (>4 mm) healed	Mean PD reduction 1.1 mm 57% of diseased sites (>4 mm) healed		Smokers have a less favourable outcome to non-surgical therapy There was almost total eradication of Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis in smokers and non-smokers
Rosen et al. (1996)	Non-surgical therapy and infra-bony pockets treated with bone grafts	1 year and 2–5 years	At 1 year: 29.2% gain in attachment 41.9% reduction in PD At 2–5 years: 31.3% gain in attachment 43.9% reduction in PD	At 1 year: 42.5% gain in attachment 49.3% reduction in PD At 2–5 years: 41.8% gain in attachment 48.3% reduction in PD		Treated infra-bony defects are adversely affected in smokers compared with non-smokers. Smoking adversely affects treatment outcomes

Table 1. (Contd.)

Study	Treatment	Interval of follow-up	Smokers	Non-smokers	Ex-smokers	Conclusions
Kaldahl et al. (1996)	Periodontal therapy followed by supportive periodontal treatment	4, 10 weeks; and yearly up to 7 years	Heavy and light smokers show similar changes of HALs at molar furcation sites relative to both ex- and non-smokers after active treatment Heavy and light smokers experienced greater loss during 7 years of SPT Heavy smokers had a higher percentage of plaque positive sites e.g. between 45% and 55% sites compared with between 15% and 35% for light smokers	Good response to therapy Improvement in HALs. Approximately 0.5 mm more PD reduction at 1 year than smokers	More PD reduction than non-smokers during SPT	Both groups of smokers, heavy and light, responded less favourably to therapy and heavy smokers responded less well than light smokers. A history of smoking is not deleterious to the response to therapy
Kinane & Radvar (1997)	Non-surgical therapy with or without local antimicrobial periodontal therapies	6 weeks	Mean PD reduction of 0.76 mm Mean attachment loss of 0.50 mm No decrease in GCF (0.41 pg/30 s) which remained lower than in non-smokers	Mean PD reduction of 1.14 mm Mean attachment loss of 0.52 mm Decrease in GCF (0.44 pg/30 s from 0.51 pg/30 s)		Smoking has an important role in determining the prognosis of periodontal treatment particularly in persistent and deeper pockets. More pronounced and significantly unfavourable response at initially deeper sites. Differences in clinical outcomes were unaffected by differences in plaque control
Pucher et al. (1997)	Non-surgical therapy	9 months	Mean reduction in PD of 0.65 mm Increase in attachment 0.59 mm BOP significantly decreased from baseline No reduction in GI compared with baseline	Mean reduction in PD of 0.6 mm maintained at 9 months Attachment level gain 0.47 mm BOP significantly decreased from baseline GI decreased significantly compared with baseline		Smokers and non-smokers responded similarly to treatment after 9 months
Grossi et al. (1997)	Non-surgical therapy	3 months	Mean reduction in PI of 0.54 Mean reduction in BI of 0.23 Mean reduction in PD of 0.33 and 1.3 mm in pockets over 5 mm Mean gain in attachment 0.32 and 1.3 mm in deep pockets	Mean reduction in PI of 0.41 Mean reduction in BI of 0.37 Mean reduction in PD of 0.49 and 1.8 mm in pockets over 5 mm Mean gain in attachment 0.43 and 1.7 mm in deep pockets	Mean reduction in PI of 0.69 Mean reduction in BI of 0.38 Mean reduction in PD of 0.49 and 1.7 mm in pockets over 5 mm Mean gain in attachment 0.43 and 1.6 mm in deep pockets	Smoking impairs periodontal healing and as the healing and microbial response of ex-smokers is comparable with non-smokers; smoking cessation may restore the normal periodontal healing response

Table 1. (Contd.)

Study	Treatment	Interval of follow-up	Smokers	Non-smokers	Ex-smokers	Conclusions
			Individual mean % of *P. gingivalis* 1.8 Individual mean % of *B. forsythus* 1.9	Individual mean % of *P. gingivalis* 0.7 Individual mean % of *B. forsythus* 1.0 *P. gingivalis* eradicated in 75% of patients	Individual mean % of *P. gingivalis* 0.7 Individual mean % *B. forsythus* 1.6 *P. gingivalis* eradicated in 92% of patients	
Renvert et al. (1998)	Non-surgical therapy	6 months	Mean reduction in PD of 1.9 mm Reduction in numbers of *P. gingivalis*- and *P. intermedia*-infected sites comparable in smokers and non-smokers with respect to baseline	Mean reduction in PD of 2.5 mm		The microbial response conforms to the clinical response with little influence of the smoking habits
Soder et al. (1999)	Non-surgical therapy with or without systemic metronidazole SRP undertaken every 6 months for 5 years Surgical intervention if PDs increased >2 mm between successive visits	Every 6 months for 5 years	After 5 years Only a reduction in absolute number of sites >5 mm Reduction in *A.a. P. gingivalis*, *P. intermedia* and spirochaetes	After 5 years Significant improvements in % of teeth with PDs >5 mm, overall PDs, attachment level and bone height ($p<0.01$) Reduction in *A.a. P. gingivalis*, *P. intermedia* and spirochaetes		The intervention group of non-smokers taking metronidazole as an adjunct to non-surgical therapy showed a statistically significant improvement. Those who were healthy (complete healing deemed to be the absence of inflamed sites $\geqslant 5$ mm) after 5 years were the same patients considered healthy after 6 months. Decisive factors to sustained improvement in patients are probably: initial scaling and root planing; a brief course of metronidazole; and 6 monthly oral hygiene, scaling and root planing
Ryder et al. (1999)	Non-surgical treatment or subgingival doxycycline	9 months	Non-surgical group Attachment level gain of 0.76 mm (0.96 mm in pockets $\geqslant 7$ mm) PD reduction of 1.02 mm (1.48 mm in pockets $\geqslant 7$ mm) Doxycycline group Attachment level gain of 0.83 mm (0.87 mm in pockets $\geqslant 7$ mm) PD reduction of 1.21 mm (1.49 mm in pockets $\geqslant 7$ mm)	Non-surgical group Attachment level gain of 1.00 mm (1.43 mm in pockets $\geqslant 7$ mm) PD reduction of 1.43 mm (2.06 mm in pockets $\geqslant 7$ mm) Doxycycline group Attachment level gain of 0.69 mm (0.12 mm in pockets $\geqslant 7$ mm) PD reduction of 1.12 mm (1.71 mm in pockets $\geqslant 7$ mm)	Non-surgical group Attachment level gain of 0.60 mm (0.90 mm in pockets $\geqslant 7$ mm) PD reduction of 1.05 mm (1.58 mm in pockets $\geqslant 7$ mm) Doxycycline group Attachment level gain of 0.88 mm (1.15 mm in pockets $\geqslant 7$ mm) PD reduction of 1.33 mm (2.00 mm in pockets $\geqslant 7$ mm)	A significantly greater clinical attachment gain was found in the non-smoking non-surgical therapy group especially in the deeper pockets where there were greater improvements compared with smokers and ex-smokers. The two treatment modalities have both common and different effects on the elimination of pathogens and host and healing response. Because of these differences, local doxycycline and NST may act in synergy if used together in periodontal treatment

Table 1. (Contd.)

Study	Treatment	Interval of follow-up	Smokers	Non-smokers	Ex-smokers	Conclusions
Zuabi et al. (1999)	Non-surgical therapy	Post-treatment	Mean PI reduction of 0.51 Mean PD reduction of 0.81 Mean attachment level gain of 0.58 mm Calcium concentration 3.58 mg/100 ml Albumin level 0.38 mg/100 ml	Mean PI reduction of 0.52 Mean PD reduction of 0.50 mm Mean attachment level gain of 0.44 mm Calcium concentration 5.11 mg/100 ml Albumin level 1.1 mg/100 ml		Post-treatment PD and attachment levels were similar in smokers and non-smokers due to the fact that smokers had greater PD reduction from the baseline scores. Smokers exhibited greater disease levels but reduced sodium, calcium and magnesium concentrations. The smokers responded favourably to treatment and the clinical improvement eliminated the differences in salivary composition
Preshaw et al. (1999)	Non-surgical therapy	6 months after a 6 month treatment phase (13 months)	At month 6 Mean PD 5.92 mm At month 13 Mean PD 4.46 mm	At month 6 Mean PD 5.30 mm At month 13 Mean PD 4.14 mm	At month 6 Mean PD 5.30 mm At month 13 Mean PD 3.68 mm	PDs were reduced following treatment in all groups even though smokers had deeper PD than non- and ex-smokers. Disease progression was not identified over a 6-month period. Significant improvements were observed after scaling and root planing and bone loss was halted or reversed. Regular and frequent maintenance visits are important following treatment Smokers, non-smokers and ex-smokers did not differ significantly in plaque, BOP, attachment levels, radiographic, or biochemical parameters
Palmer et al. (1999)	Non-surgical therapy with or without systemic or local metronidazole (Elyzol® Dumex Ltd, Copenhagen, Denmark)	6 months	Reduction in PD of 1.23 mm Reduction in spirochaetes 35.8%	Reduction in PD 1.92 mm Reduction in spirochaetes 46.7%		Smokers have a poorer treatment response to scaling and root planing regardless of the application of either systemic or locally applied adjunctive metronidazole No significant differences between smokers and non-smokers for attachment loss or any clinical response to the treatment regimes
Papantonopoulos (1999)	Non-surgical therapy	6–12 weeks	42.8% of smokers needed further treatment of 16% of their teeth	11.5% of non-smokers needed further treatment of 16% of their teeth		For smokers with at least one of five sites $\geqslant$6 mm, surgery should be initiated rather than treating first with non-surgical therapy. Smoking impairs healing after non-surgical periodontal therapy Further treatment needs were particularly high in upper and lower pre-molar areas

Table 1. (Contd.)

Study	Treatment	Interval of follow-up	Smokers	Non-smokers	Ex-smokers	Conclusions
Jin et al. (2000)	Non-surgical therapy	1, 3 and up to 6 months	At 1, 3 and 6 months there were mean PD reductions of 1.1, 1.1 and 1.6 mm, respectively. At 1, 3 and 6 months there were mean attachment level gains of 0.1, 0.3 and 0.5 mm, respectively	At 1, 3 and 6 months there were mean PD reductions of 1.9, 2.4 and 2.5 mm, respectively At 1, 3 and 6 months there were mean attachment level gains of 0.5, 0.8 and 1.2 mm, respectively		Significant reductions in PD in non-smokers compared with smokers at all time points Significant attachment level gains in non-smokers only seen at 6 months. Smokers have different treatment response patterns and healing dynamics following non-surgical therapy suggesting the importance of a more intensive treatment regime
Bergström et al. (2000)	Non-surgical therapy	10 years	Frequency of diseased sites (PD >4 mm) increased from 18.7% to 41.6%. Mean % bone height reduction from 80.3% to 76.5%	Frequency of diseased sites (PD >4 mm) decreased from 8.7– 6.6% Mean % bone height reduction from 85.1–84.1%	Frequency of diseased sites (PD >4 mm) decreased from 11.1% to 7.8% Mean bone height reduction from 80.7% to 79.6%	The 10-year change increased significantly with increasing smoking exposure controlling for age Periodontal health remained unaltered throughout the 10 years for non-smokers suggesting that smoking cessation is beneficial to periodontal health. As the plaque index remained at similarly low levels in all groups it was concluded that the only harmful impact on periodontal health to be detected was smoking
Meinberg et al. (2001)	Non-surgical therapy	1 year	Mean bone loss 5.75 mm; smokers had consistently higher percentages of moderate and severe pockets than did non-smokers	Mean bone loss of 4.64 mm	Mean bone loss of 4.89 mm	The impact of smoking may require longer than 1 year to show longitudinal changes. It is recommended that radiographic analysis is carried out periodically during non-surgical therapy and that longer term studies should be conducted to identify the outcome of smoking status on this variable
Trombelli et al. (2003)	Flap surgery at furcation defects	6 months	27.6% of class II furcations showed improvement After 6 months 3.4% of pre-surgery class I furcation defects showed complete closure	38.5% of class II furcations showed improvement After 6 months 27.8% of pre-surgery class I furcation defects showed complete closure		Flap surgery produced clinically and statistically significant PD reduction and clinical attachment gain in class I/II molar furcation defects Smokers exhibit a less favourable healing outcome following surgery in terms of vertical and horizontal attachment gain
Papantonopoulos (2004)	Non-surgical therapy and surgical therapy as required	3–4 times a year for between 5 and 8 years	11 sites in six smokers exhibited radiographic bone loss $\geqslant 2$ mm over 5–8 years Two smokers lost three teeth	Seven sites in four non-smokers exhibited radiographic bone loss $\geqslant 2$ mm over 5–8 years One non-smoker lost One tooth		Patients treated for advanced periodontal disease and well maintained over 5–8 years showed no statistically significant differences between smokers and non-smokers in clinical probing depths and radiographic bone-loss measurements Smoking increased the OD 10.7 $\times$ of having $\geqslant 1$ site with bone loss $\geqslant 2$ mm

Table 1. (Contd.)

Study	Treatment	Interval of follow-up	Smokers	Non-smokers	Ex-smokers	Conclusions
Tomasi & Wennström (2004)	Non-surgical ultrasonic treatment with locally delivered doxycycline gel 8.5%ww Atridox™ (Collagenex Pharmaceuticals Inc., Newtown, PA, USA)	3 months	Mean PD reduction of 1.35 mm Mean CAL gain of 0.84 mm	Mean PD reduction of 1.62 mm Mean CAL gain of 0.90 mm		Smoking negatively influences treatment outcomes in terms of PD reduction and CAL gain
Stavropoulos et al. (2004)	Surgical treatment of vertical defects with bioresorbable membranes	12 months	Mean PD reduction of 4.5 mm Mean CAL gain of 3.2 mm	Mean PD reduction of 5.5 mm Mean CAL gain of 4.3 mm		Smoking impairs healing of GTR-treated infrbony defects
Preshaw et al. (2005a)	Subantimicrobial systemic doxycycline (Periostat® as an adjunct to scaling and root planing in a placebo-controlled trial	9 months	A hierarchical treatment response with non-smokers on doxycycline showing the greatest CAL gain and reduction in PD. In general, smokers on doxycycline performed approximately the same as the non-smokers on placebo			

PD, probing depth; PI, plaque index; BOP, bleeding on probing; HAL, horizontal attachment level; REC, gingival recession; SPT, supportive periodontal therapy; GCF, gingival crevicular fluid; GI, gingival index; CAL, clinical attachment level; *P. gingivalis, Porphyromonas gingivalis*; *B. forsythus, Bacillus forsythus*.

cluded that adjunctive sub-antimicrobial dose doxycyclin-enhanced therapeutic outcomes in all groups with smokers taking doxycyclin showing approximately the same magnitude of clinical improvement as non-smokers on placebo.

Longitudinal Evidence for the Benefits of Smoking Cessation on the Periodontium

The majority of studies investigating the effects of smoking cessation on periodontal disease acknowledge the benefits of giving patients smoking cessation advice and that smoking cessation may result in a long-term benefit to the periodontal condition (Ramseier 2005). Further, the implementation of population-based smoking cessation programmes may also have a significant impact on the prevalence and progression of periodontal diseases (Susin et al. 2004). The evidence to confirm unequivocally and scientifically the benefit of quitting smoking on patients with periodontal disease is, however, sparse and several authors have expressed concern for this lack of evidence (AAP 1996, Qandil et al. 1997, Meinberg et al. 2001, Scott et al. 2001).

Limited evidence is available, however. Bolin et al. (1993) reported results from a 10-year radiographic follow-up study of alveolar bone loss which found that the progression of bone loss was significantly retarded in those who had quit smoking during the study compared with continual smokers.

In the only longitudinal study to date, our group has reported 12-month data from 10 subjects with periodontitis who had continuously quit smoking for the entire study period. The quitters demonstrated a significant reduction in probing depths compared with non-quitters as well as a higher incidence of probing depth reductions of $\geqslant 2$ and $\geqslant 3$ mm (Preshaw et al. 2005b).

Immune-Inflammatory Mechanisms Underlying the Clinical Response

The evidence reviewed in the previous sections suggests clearly that the periodontal treatment response in non-smokers is significantly better than the response seen in smokers irrespective of the nature or modality of the treatment undertaken. There is insuffi-

Table 2. The effects of smoking on the bacterial challenge, the host periodontal tissues and the immune-inflammatory response. The potential effect of quitting smoking is hypothesized

Author, year	Effect of smoking and nicotine on the host periodontal tissues and the immune inflammatory response	The potential effect of quitting smoking
Alavi et al. (1995) Persson et al. (1999)	Smoking reduces the absolute amount of GCF elastase in patients with established and untreated chronic periodontitis suggesting a compromised neutrophil response This observation was not seen in healthy individuals	More effective phagocytosis and digestion by neutrophils
Soder et al. (2002)	There is an increase in the release of neutrophil functional elastase and elastase complexed to α-1 antitrypsin in smokers with chronic periodontitis. Nicotine has a depressive effect on neutrophil function and enhances degranulation, as the cells are more sensitive to bacterial challenge	Restoration of neutrophil function to provide a more effective response to the bacterial challenge
Böstrom et al. (1998), Böstrom et al. (1999), Fredriksson (2002)	Increased concentrations of TNF-α in GCF of smokers owing to exposure of macrophages to nicotine In vitro studies suggest that peripheral neutrophils of smokers tend to release more TNF-α than those of non-smokers	Damping of TNF-α release may contribute to a reduction in connective tissue (including bone) destruction and periodontal stability The increase in TNF-α is also seen in ex-smokers, which suggests that smoking may have a long-lasting effect of neutrophil response
Kazor et al. (1999), Zambon et al. (1996)	Smoking appears to select for specific periodontopathogens (*P. gingivalis*; *T. denticola*; *B. forsythus*) thus increasing the risk of the development and progression of periodontal disease	A shift towards a less pathogenic subgingival flora although the BANA-positive pathogens are equally prevalent in ex-smokers as in smokers. 68% of plaques removed from never smokers also contain BANA-positive organisms (Kazor et al. 1999)
Loesche (1994), Bergstrom & Bostrom (2001), Chen et al. (2001), Nair et al. (2003), Morozumi et al. (2004a)	Smoking leads to sustained peripheral vasoconstriction caused by chronic low doses of nicotine. This leads to reduced gingival bleeding. This effect can 'mask' the presence of periodontal disease. The compromised gingival microvasculature could also lead to reduced oxygen tension that would allow periodontal anaerobes to prevail (Loesche 1994)	An increase in oxygen tension may help towards a shift towards a less pathogenic subgingival flora Gingival microcirculation could recover in the early stages of smoking cessation, which could activate gingival tissue metabolism and local host immune responses
Mavropoulos et al. (2003)	There is some evidence to suggest that, in smokers, gingival blood flow may actually increase owing to hyperaemia and the increase in blood pressure although, in the long term, the effect of vasoconstriction overcomes this more acute effect (Mavropoulos et al. 2003)	Gingival blood and crevicular fluid flow increase within only a few days after quitting smoking (Morozumi et al. 2004a) and the prevalence of bleeding on probing in patients with chronic periodontitis increases, despite an improvement in the level of plaque control (Nair et al. 2003)
Haffajee & Socransky (2001)	Greater prevalence of orange and red complexes in smokers than in ex-smokers and never smokers: *E. nodatum*; *F. nucleatum ss vincentii*; *P. intermedia*; *P. micros*; *P. nigrescens*; *B. forsythus*; *P. gingivalis*; *T. denticola*. This observation was seen in deep (>4 mm) and shallow (<4 mm) pockets	A shift towards a less pathogenic subgingival flora possibly by changing the anaerobic conditions of the subgingival environment at all pocket depths
Cuff et al. (1989)Giannopoulou et al. (1999), James et al. (1999) Gamal & Bayomy (2002)	Nicotine inhibits the proliferation, chemotaxis and attachment of periodontal ligament fibroblasts in vitro There is also structural alteration of the cells that prevent them from adhering to flat and root-planed surfaces These effects are likely to be enhanced by nicotine on root surfaces (Cuff et al. 1989) and this may affect periodontal regeneration after therapy	Enhanced fibroblast adhesion to the root surface after root surface instrumentation leading to better integrity of the healing periodontal tissues One aim of root surface instrumentation may be to remove nicotine deposits that may prevent a healing response. Such deposits are likely to reform in smokers who persist with the habit following periodontal treatment

Table 2. (Contd.)

Author, year	Effect of smoking and nicotine on the host periodontal tissues and the immune inflammatory response	The potential effect of quitting smoking
Lie et al. (2001)	Smoking reduces salivary cystatin activity and cystatin C levels during experimental gingivitis. This observation does not appear to be related to flow rate	Increased output of glandular cystatins and cystatin activity will contribute towards host protection against inflammation by inhibiting certain proteolytic enzymes such as cysteine proteinases
Loos et al. (2004)	Smoking leads to greater periodontal breakdown associated with increased numbers of CD3+ T cells as well as CD4+ and CD8+ T cell subsets. There was no apparent effect on the numbers of B cells	Damping down of the enhanced T cell response
Rawlinson et al. (2003)	GCF IL-1β levels are reduced in GCF at deep sites–both bleeding and non-bleeding. This may be owing to reduced production or possibly to increased receptor binding of the molecules that would lead to greater activity of IL-1β	Restoration of the immuno-inflammatory response will lead to greater concentrations of IL-1β in GCF. This may represent an enhanced immune response or a reduction in bound IL-1β
Bergström (2004)	10 years bone height reduction was $\times$ 2.7 greater in smokers than in non-smokers	
Jansson & Lavstedt (2002), Jansson et al. (2002) Paulander et al. (2004) Baljoon et al. (2004)	Smoking was significantly correlated to an increased marginal bone loss over 20 years and found to be a significant risk factor in marginal bone loss Relative risk of continued alveolar bone loss 3.6 for smokers compared with non-smokers Prevalence and severity of vertical bone defects significantly associated with smoking Relative risk of vertical bone loss associated with smoking is increased two to three fold	Smoking cessation results in a return towards the rate of bone loss seen in non-smokers Subjects who quit smoking (ex-smokers) have reduced risk for bone loss compared with never smokers (Paulander et al. 2004)
Morozumi et al. (2004b)	Transcript levels (mRNA) of peripheral neutrophils in smokers are generally lower than those in non-smokers – possibly associated with an impairment of neutrophil function	Smoking cessation in 11 subjects for 8 weeks showed a significant recovery of matrix metalloproteinase-8 RNA; a sign of recovery of neutrophil metabolism and viability
Kamma et al. (2004)	GCF IL-1β shows a positive association with smoking but only in periodontally healthy subjects. This is possibly owing to already maximal stimulation of monocytes having occurred in subjects with periodontitis	Restoration of the local imbalance in cytokine production
Petropoulos et al. (2004)	A number of complex mechanisms including IL-1 genotype likely influences marked reduction of GCF IL-1α production	Restoration of the local imbalance in cytokine production
Natto et al. (2005)	In a Saudi Arabian population, the prevalence of bone loss in excess of 30% original bone height was 24% in smokers compared with 6% in non-smokers	Reduction of the rate of alveolar bone resorption

cient evidence currently available, however, to determine unequivocally the precise mechanisms by which quitting smoking moderates or influences the host response to the treatment. Indeed, the potential benefit of smoking cessation is likely to be mediated through a number of different pathways that may include a shift towards a less pathogenic subgingival flora, recovery of the gingival microcirculation, restoration of neutrophil function, metabolism and viability, damping of the enhanced immune response and re-establishing any imbalance in the local or systemic production of cytokines.

A brief, contemporary overview of the effects of smoking, nicotine and its metabolites on the subgingival microflora, the periodontal tissues and the host response is presented in Table 2 so that the potential mechanisms that may underpin the potential benefits of quitting smoking can be hypothesized.

Conclusions

A long-term, longitudinal clinical trial that monitors over several years the response to treatment in a cohort of smokers with chronic periodontitis who quit the habit will provide a formidable challenge, particularly as continuous quit rates over extended periods are likely to erode the sample size, and therefore, power of a study. Such a study will inevitably be lengthy and expensive, and would need to enrol multiple centres to ensure an adequate sample size, and more clinically and significantly meaningful data; a view confirmed by Palmer (2005) in a recent Guest Editorial in this Journal. Until such a study is undertaken, the dental profession can reflect on the strong evidence base of the epidemiological, cross-sectional and case–control studies that are presented in the tables of this review to conclude that quitting smoking is likely to be beneficial to oral, and in particular, periodontal health. It is also reassuring to reflect on the theoretical modelling of the cost-effectiveness of smoking cessation described originally by Sintonen & Tuominen (1989). This model was reviewed again recently by Braegger (2005) in a position paper on cost–benefit of smoking cessation with the conclusion that adding smoking cessation to the concept of periodontitis prevention will enable significant cost savings to be made.

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Clinical Relevance

Rationale: Smoking and periodontal disease is an active area of research; an overview of the clinical evidence for smoking as a risk factor needs to be updated.

Principal findings: A wealth of evidence supports the fact that periodontitis is more prevalent in smokers than non-smokers. The intermediate (between smokers and non-smokers) response to treatment of ex-smokers suggests that quitting the habit is of clinical benefit.

Practical implications: Quitting smoking and the response to the hygiene phase of management necessitate behavioural change if long-term clinical success is to be achieved. The periodontist and hygienist are ideally positioned to effect such change.