Smokers have less reductions in probing depth than non-smokers following nonsurgical periodontal therapy

What is the effect of smoking and smoking cessation on the response to nonsurgical periodontal therapy in individuals who have chronic periodontitis?

Labriola A, Needleman I, Moles DR. *Systematic review of the effect of smoking on non-surgical periodontal therapy. Periodontol 2000 2005; 37:124–137*

Data sources Studies were located using Medline, Embase and the Cochrane Central Register of Controlled Clinical Trials, as well as searching bibliographies of identified references, review articles and consensus statements by hand.

Study selection As the habit of smoking cannot be randomised, studies included were both controlled clinical trials and arms of randomised controlled trials investigating the effects of nonsurgical periodontal treatment, which reported results separately for smokers and nonsmokers. Other inclusion criteria were that studies should assess otherwise healthy people who had been diagnosed with chronic or adult periodontitis and that 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.

Data extraction and synthesis General information about the paper, study characteristics, outcome measures, treatment characteristics and quality assessment was extracted independently, in duplicate. Where disagreement arose, this was resolved by discussion. Meta-analysis of data was performed and heterogeneity between studies was investigated using meta-analysis regression.

Results Out of the 330 studies initially identified, 13 were considered eligible for inclusion. No studies reported data on tooth loss. For all sites, the reduction in probing depth (PD) was 0.13 mm greater in nonsmokers than in smokers (six studies) and there was no evidence to suggest that the studies were dissimilar in their estimates of this result (no evidence of heterogeneity; $P>0.05$). For sites with an initial PD of 5.00 mm (eight studies), a random-effects meta-analysis indicated a weighted mean difference in PD reduction of 0.43 mm favouring nonsmokers (95% confidence interval (CI), 0.24–0.63; $P<0.001$). Because of significant heterogeneity between studies, only a cautious observation can be made but, with one exception, all studies produced a summary estimate favouring nonsmokers. Meta-analysis of the two studies that compared the change in clinical attachment level between nonsmokers and ex-smokers, who had given up their habit, in sites with an initial PD of ± 5.00 mm, showed a difference in clinical attachment level gain of 1.34 mm favouring the nonsmokers (95% CI, 0.65–2.03; $P<0.001$; χ^2 test for heterogeneity, 7.47 with 1 degree of freedom; $P=0.006$). In both of these analyses, the degree of heterogeneity is a cause for concern. Bleeding was assessed after therapy in seven studies but meta-analysis was not performed because of great heterogeneity in the methods used to assess bleeding. No statistically significant differences in bleeding were found between smokers and nonsmokers

either at baseline or after therapy in most of the studies. One study found significantly less bleeding in smokers than in nonsmokers at baseline and another found a reduced response in terms of bleeding in smokers than in nonsmokers. Two studies evaluating the change in bleeding in ex-smokers found no statistically significant difference after treatment. No data were reported for any of the included studies on patient-centred outcomes such as quality of life, ease of maintenance, changes in aesthetic appearance, or patient experience.

Conclusions Following nonsurgical periodontal therapy, people who smoke will experience less reduction in PD 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 stop smoking will respond as favourably to nonsurgical therapy as those who have never been smokers. Progress in understanding the effects of smoking on periodontal therapy will require the evaluation of objective measures of smoking such as nicotine exposure and exhaled carbon monoxide in place of sole reliance on patient-reported information.

Commentary

Epidemiological evidence has accumulated over the past two decades to show convincingly that tobacco smokers are at increased risk for periodontal disease, and also for tooth loss.¹ Smoking cessation is associated with risk reduction, although even 10 years after cessation the risk in former smokers does not decline to the level of people who have never been smokers.¹ Importantly, the use of sophisticated analytical techniques has made it possible to control for various behavioural characteristics of smokers in epidemiological studies: we know it is the smoking itself, and not some other characteristic of smokers, that is the key risk factor.

While the exact biological mechanisms by which tobacco smoke affects periodontal health remain to be elucidated, it is clear that smoking affects the inflammatory and immune responses as well as the microvasculature. Interestingly, evidence exists that smoking exerts a strong, chronic and dose-dependent suppressive effect on gingival bleeding on probing.²

It has been reported previously that smoking negatively affects the response to periodontal surgery, at least in initially deep sites.³ This systematic review of how smoking affects the response to nonsurgical periodontal therapy illustrates the limited availability of data for clinicians to use in making treatment recommendations to smokers. Although it was evident that smokers have less favourable results following nonsurgical periodontal therapy in terms of PD reduction, there were no significant differences between smokers and nonsmokers in clinical attachment gain.

At least two clinically important questions remain unanswered: first, what is the benefit, if any, of smoking cessation during

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periodontal treatment, or just prior to initiation of treatment? Second, how many years of smoking cessation does it take for the response to periodontal treatment in former smokers to equal the response in individuals who have never been smokers?

Although the authors recommend the use of physiological measures of tobacco exposure, such measures are of value only in current smokers (and in identifying those falsely selfreporting that they are former smokers). In order to accurately assess the effect of smoking cessation on periodontal outcomes, however, selfreported measures of past smoking behaviour are still required, including duration, frequency and intensity of use, and time since cessation. Recently, a comprehensive smoking index was developed that combines these multiple aspects of prior tobacco use into a single analytic variable.⁴ By more accurately modelling prior tobacco use, it should be possible to accurately determine the benefits of smoking on periodontal risk reduction and on response to periodontal treatments.

Practice point

● Smokers have less favourable results after nonsurgical periodontal therapy in probing depth reduction but there are no significant differences between smokers and nonsmokers in clinical attachment gain.

Note

Dr Needleman one of the authors of the original review has highlighted the fact that the review presented the available data comparing quit smokers with non-smokers, but the data are limited and inconclusive rather than not investigated. The authors also presented the available data for the effect of smoking on bleeding on probing, but again the data was inconclusive.

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