Impact of Cigarette Smoking on Clinical Outcomes of Periodontal Flap Surgical Procedures: A Systematic Review and Meta-Analysis

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Background: Periodontal flap surgery is frequently used to remove subgingival deposits, yielding consequential reductions in gingival inflammation and probing depth (PD) with a gain in clinical attachment level (CAL) to treat advanced periodontal disease. However, clinical studies have reported diminished periodontal healing in smokers compared with non-smokers. The aim of the present systematic review and meta-analysis was to assess the impact of cigarette smoking on clinical outcomes following periodontal flap surgical procedures.

Methods: A systematic electronic review of articles relevant to periodontal flap surgical procedures in smokers was conducted from 1977 to March 2014 inclusive, using predefined, optimized search strategies. Meta-analyses were done separately for changes in the two primary outcomes of PD and CAL.

Results: The initial search yielded 390 titles and abstracts. After screening, eight controlled clinical studies were finally selected. Three studies were assessed as having a low risk of bias, two as having moderate risk of bias, and three as having a high risk of bias. Qualitative assessment of the articles consistently showed an improved treatment effect among non-smokers versus smokers. The reduction in PD in smokers and non-smokers ranged from 0.76 to 2.05 mm and 1.27 to 2.40 mm, respectively. For CAL, the gain in non-smokers versus smokers ranged from 0.29 to 1.6 mm and 0.09 to 1.2 mm, respectively. Meta-analysis on eight studies reporting on 363 study participants demonstrated an increased reduction in mean (95% confidence interval) PD of 0.39 (0.33 to 0.45) mm. Similar results were found for mean gain in CAL (0.35 [0.30 to 0.40] mm, n = 4 studies).

Conclusions: Considering the relatively homogenous information available, the authors conclude that active smokers could be candidates for periodontal flap surgical procedures. However, the magnitude of the therapeutic effect is compromised in smokers compared with non-smokers. Therefore, cigarette smokers should be: 1) encouraged to abstain from smoking; and 2) thoroughly informed preoperatively of substantial reduction in clinical outcomes compared with non-smokers. J Periodontol 2015;86:254-263.

KEY WORDS

Meta-analysis; periodontal debridement; periodontal pocket; periodontitis; review; smoking.

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urrent data reveal an estimated 4.1 million smokers aged >30 years with severe peri-
odontitis in the United States.1 Smoking has
been thoroughly investigated as a risk factor for peri-
odontal disease, and several studies have shown
the association between habitual cigarette use and
deterioration of clinical indices of periodontal dis-

ease.2-5 Nonetheless, smokers often exhibit lower
scores of bleeding on probing (BOP) than non-smokers
mainly because of the vasoconstrictive effect of
nicotine on gingival blood vessels.6 Therefore, smokers
may remain unaware of their compromised peri-
odontal health status until they present for examina-
tion and subsequent treatment at more advanced
stages of periodontal disease. Thus, habitual smokers
often represent a subpopulation with increased preva-

lence of advanced periodontal disease.1

In terms of periodontal treatment, non-surgical
periodontal therapy is routinely performed as cause-
related therapy for the control of periodontal inflam-
nation. However, it may not always yield substantial
reduction in probing depth (PD), especially among
deeper pockets.7-9 Periodontal flap surgery is often
required to achieve adequate reduction in PD in
patients with advanced periodontal disease, as in the
case of habitual smokers.10 Yet, results from clinical
studies have reported diminished outcomes of the
healing response following periodontal surgery in
smokers compared with non-smokers.11-14 In a
randomized, controlled clinical trial, the treatment
outcome in periodontal furcation defects following
periodontal flap surgery was compared between
cigarette smokers and non-smokers.14 Six-month
follow-up results revealed twice as much gain of
clinical attachment level (CAL) in non-smokers
than smokers.14 Other studies have also supported
a trend for less favorable healing following periodontal
surgical procedures in smokers as well as increased
risk for relapse during post-surgical maintenance.13,15

Various mechanisms by which smoking impairs
the healing response have been investigated to ex-
plain these unfavorable clinical findings.16 These
mechanisms encompass: 1) impaired neutrophilic
function,17 2) decreased immunoglobulin A (IgA)
and IgG production in saliva and serum,18,19 3) in-
creased proliferation of periodontal pathogens,20 and
4) impaired fibroblastic proliferation and function.21

There have also been reports on the increase in ex-
pression of receptor of advanced glycation end pro-
ducts in gingival tissues as a potential mechanism
of action that is currently under investigation.22

Results of a systematic review that assessed the
effect of cigarette smoking on clinical outcomes
following periodontal surgical procedures showed
that periodontal wound healing is compromised in
cigarette smokers compared with non-smokers.23

The same review also reported that the hazardous
effect of smoking manifests itself regardless of fre-
quency or duration.23 That systematic review included
case reports and case series in addition to controlled
studies and dealt with a range of surgical procedures
including periodontal flap surgical procedures, root
coverage procedures, and guided tissue regeneration.
To the authors’ knowledge, a meta-analysis of studies
assessing the effect of cigarette smoking on periodontal
flap procedures has not been previously performed.
Thus, the aim of the present systematic review and
meta-analysis is to assess the impact of cigarette
smoking on the clinical outcomes following periodontal
flap surgical procedures (flap debridement surgery,
modified Widman flap, apically positioned flap).

MATERIALS AND METHODS

Search Strategy
Based on the Preferred Reporting Items for System-

atic Reviews and Meta-Analyses (PRISMA) guide-
lines, a specific question was constructed according
to the participants, interventions, control, outcomes
(PICO) principle.24 Participants (P)—persons in the
included studies must have been smokers diagnosed
with moderate and/or advanced periodontitis; types
of interventions (I)—the intervention of interest was
periodontal flap surgery; control intervention (C)—
non-smoker patients receiving the same intervention
were considered as controls; and outcome measures
(O)—changes in CAL and PD post-surgically were
set as efficacy outcomes. Adverse events associated
with surgical treatment in smokers were recorded as
safety outcomes.

To identify studies relevant to the PICO question,
the Medline (Ovid) database, the EMBASE database,
and the Cochrane Central Register of Controlled Trials
(CENTRAL) were electronically searched for available
data. The search included articles published from
January 1946 up to and including March 2014. Ar-
ticles available online in electronic form ahead of print
were considered eligible for inclusion.

The first phase of the evaluation of the literature
included a search of the electronic databases using
both Medical Subject Heading terms and free text
relevant to periodontal flap procedures (i.e., flap
debridement surgery, modified Widman flap, apically
positioned flap) and smoking/tobacco use. The com-
plete search strategy for Medline (Ovid) is available in
Table 1. An additional search of the Medline (Ovid) In-
Process database was conducted to capture the latest
relevant articles.

Selection Criteria
Two reviewers performed the screening independently
(GK, FJ) after reviewing the title and abstract of each
potentially relevant article according to the following
inclusion criteria: 1) original articles; 2) human controlled, clinical studies; 3) ≥10 participants; 4) ≥6 months of follow-up post-intervention; and 5) surgical interventions that included flap debridement surgery, modified Widman flap, and apically positioned flap procedures in smokers and non-smokers.

Exclusion criteria were: 1) review articles; 2) case series; 3) case reports; 4) surgical interventions for correction of mucogingival defects (i.e., soft tissue grafting procedures); 5) use of biologic factors and/or grafting procedures; and 6) animal studies.

For articles that were considered potentially relevant by at least one reviewer, the full-text articles were obtained for eligibility evaluation against the predetermined inclusion criteria. An additional manual search of the following journals was performed from January 1990 to March 2014: Journal of Clinical Periodontology, Journal of Periodontology, The International Journal of Periodontics & Restorative Dentistry, and Journal of Periodontal Research. The reference list of each of the selected full-text articles was also reviewed for article titles suggesting surgical periodontal treatment in smokers. If required, an attempt was made to contact the corresponding authors to obtain missing, unclear, or unpublished data.

As in the first phase of selection, the two reviewers conducted the second phase independently and in duplicate. K scores (Cohen’s k coefficient) were used to determine the level of agreement between the two reviewers.25

Data Extraction
Two reviewers independently mined data regarding the year of publication, location of data, source of funding, patient characteristics, baseline plaque levels, number of participants in each group, number of interventions, smoking frequency and duration, and outcomes for each study and entered them in electronic spreadsheets.

Outcome Variables
The effects of smoking on CAL and PD were evaluated as the primary outcome variables.

Assessment of Bias Within Studies
A specific protocol was used independently by the two reviewers for the assessment of the screened articles. The clinical studies included in the present systematic review were assessed using criteria from the revised Consolidated Standards of Reporting Trials (CONSORT) statement criteria for evaluation of randomized, controlled trials according to a previously described protocol.26-28 The aim of the assessment was to identify within-study bias related to reporting of randomization, masking, follow-up, statistical analysis, and reporting of outcomes for the selected studies. A cumulative score was formed for each study following quality assessment, and an overall estimation of risk of bias was assigned to each included randomized clinical trial.27,29 Studies in which all of the criteria were met were assigned a low risk of bias. A moderate risk was considered when at least one of the criteria was partially met, and a high risk of bias was estimated when one or more of the criteria were not met.27,29

Statistical Analyses
Meta-analyses were conducted separately for each of the two primary outcomes: PD and CAL, as previously described.27 In case of missing data for the meta-analyses, the corresponding authors were contacted twice and the response rate was recorded. The mean differences between the reduction in PD and gain in CAL in smokers versus non-smokers groups were estimated as the effect-size measures. By definition, a mean difference <0 indicated a greater effect size in non-smokers. Heterogeneity among the included studies for each outcome was assessed using the q statistic and $I^2$ statistic.30

Outcome measures were combined with a random-effects model using the DerSimonian-Laird method.
due to its robustness in comparison to fixed-effects models in the case of small sample sizes. Forest plots were produced reporting weighted average of outcomes and 95% confidence intervals (CIs). The α level was set at 0.05. Funnel plots were constructed to explore potential publication bias across studies. All the statistical analyses were carried out with a specialized statistical software. ¶

RESULTS

A total of 390 titles and abstracts were identified after the electronic search using the prespecified search strategy (Fig. 1). No additional studies were identified as relevant after a search of the reference lists and manual search of the selected journals. After removal of duplicates (n = 70), initial screening of titles and abstracts was performed, and 305 articles were excluded as irrelevant to the PICO question (κ score for inter-reviewer agreement [95% CI]: 0.92 [0.88 to 0.97]). Scrutiny of the full-text articles from the remaining 15 articles led to the exclusion of seven studies after application of the prespecified exclusion criteria, due to either lack of clinical data on smoking status, use of regenerative techniques only, or multiple reports from the same study population (κ score for inter-reviewer agreement [95% CI]: 0.87 [0.62 to 0.99]) (see Supplementary Fig. 1 in online Journal of Periodontology). In total, eight clinical trials fulfilled the inclusion criteria and were included in this review.

Risk of Bias Assessment of Included Studies

Three studies were assessed as having a low risk of bias, two as having moderate risk of bias, and three as having a high risk of bias. In the three studies that were assessed as having a high risk of bias, at least four of the six methodologic criteria were fully or partially met (see Supplementary Fig. 2 in online Journal of Periodontology).

Qualitative Results of Studies

All studies reported changes in PD as an outcome, whereas four studies also reported on gain in CAL post-treatment. In total, the included studies reported on 363 study participants with ages ranging from 23 to 80 years. The mean ages of smokers and non-smokers ranged from 39.6 to 59.7 years and 42.9 to 61.9 years, respectively. In none of the studies were there any significant differences between mean ages of smokers and non-smokers. Duration of smoking was reported in three studies and ranged from 1 to 27.8 years. Smoking frequency thresholds for patient inclusion were reported in six studies and ranged from ≥1 cigarette per day to at least 20 daily (Table 2). Periodontal treatment was performed by means of modified Widman flap in three studies and flap debridement surgery with or without osseous recontouring in four studies. And both treatment approaches were used in the remaining study. The follow-up period ranged from 6 months up to 7 years.

In terms of clinical results, similar reduction in BOP was noted overall for smokers and non-smokers (Table 3). All studies reported significantly less reduction in PD among smokers compared with
Table 2.
Main Characteristics for Studies Included After Second Phase of Selection

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Funding</th>
<th>Intervention</th>
<th>Follow-Up</th>
<th>Medical History</th>
<th>Periodontal Status</th>
<th>Baseline Plaque Levels</th>
<th>Smoking Duration</th>
<th>Smoking Dosage (cigarettes/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preber and Bergström</td>
<td>1990</td>
<td>Swedish health and dental care company*</td>
<td>MWF</td>
<td>12 months</td>
<td>NA</td>
<td>Moderate-to-severe periodontitis</td>
<td>&lt;0.5 PI</td>
<td>≥5 years</td>
<td>≥20/day</td>
</tr>
<tr>
<td>Kaldahl et al.</td>
<td>1996</td>
<td>United States federal grant</td>
<td>MWF; FDS with osseous surgery</td>
<td>Yearly, ≤7 years</td>
<td>NA</td>
<td>Moderate-to-severe periodontitis</td>
<td>&lt;30% plaque control record</td>
<td>NA</td>
<td>Overall: ≥10/day; heavy smokers: ≥20/day; light smokers: &lt;19/day; past smokers: history of daily smoking in the past</td>
</tr>
<tr>
<td>Boström et al.</td>
<td>1998</td>
<td>Swedish Dental Association; Swedish health and dental care company*</td>
<td>MWF</td>
<td>5 years</td>
<td>Non-contributory</td>
<td>Moderate periodontitis</td>
<td>28.5% ± 15.7% plaque control record</td>
<td>Current smoker: 19.9 years; past smoker: 16.6 years</td>
<td></td>
</tr>
<tr>
<td>Scabbia et al.</td>
<td>2001</td>
<td>Ministrio Dell` Universita e della Ricerca, Italy</td>
<td>FDS without osseous recontouring</td>
<td>6 months</td>
<td>Non-contributory</td>
<td>Moderate-to-severe periodontitis</td>
<td>43% ± 21% plaque control record</td>
<td>NA</td>
<td>≥10/day (range: 10 to 60 cigarettes)</td>
</tr>
<tr>
<td>Orbak et al.</td>
<td>2003</td>
<td>NA</td>
<td>FDS</td>
<td>NA</td>
<td>NA</td>
<td>CP</td>
<td>1.99 ± 0.5 PI</td>
<td>≥1 year</td>
<td>≥15/day</td>
</tr>
<tr>
<td>Trombelli et al.</td>
<td>2003</td>
<td>Ministrio Dell` Universita e della Ricerca, Italy</td>
<td>FDS without osseous recontouring</td>
<td>6 months</td>
<td>Non-contributory</td>
<td>Moderate-to-severe periodontitis</td>
<td>47% ± 24% plaque control record</td>
<td>NA</td>
<td>≥10/day</td>
</tr>
<tr>
<td>Hellström et al.</td>
<td>2008</td>
<td>Specialty pharmaceutical company†</td>
<td>MWF</td>
<td>6 months</td>
<td>Non-contributory</td>
<td>Moderate-to-severe CP</td>
<td>44% ± 31% plaque control record</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Kim et al.</td>
<td>2007</td>
<td>Private dental products company‡</td>
<td>FDS</td>
<td>3 and 6 months</td>
<td>Non-contributory</td>
<td>Moderate-to-severe CP and AgP</td>
<td>0.16 ± 0.51 PI</td>
<td>NA</td>
<td>≥1/day</td>
</tr>
</tbody>
</table>

MWF = modified Widman flap, PI = plaque index, FDS = flap debridement surgery, NA = information not available.

* Praktikertjanst, Stockholm, Sweden.
† OraPharma, Horsham, PA.
‡ Ivoclar Vivadent, Schaan, Liechtenstein.
The reduction in PD in smokers and non-smokers ranged from 0.76 mm to 2.05 mm and 1.27 mm to 2.40 mm, respectively. For CAL, the gain in non-smokers versus smokers ranged from 0.29 mm to 1.6 mm and 0.09 mm to 1.2 mm, respectively. All studies reported greater CAL gain in non-smokers compared with smokers but also consistently noted a mean gain in post-surgical CAL for smokers (Table 3). None of the studies reported any adverse events associated with smoking status.

Quantitative Results of Studies

After data extraction and, when necessary, communication with authors (authors' response rate: one of four), eight studies11,13-15,38-41 were included in the meta-analysis of the weighted mean differences of PD and four11,14,15,39 in the meta-analysis of the weighted mean differences of CAL (Fig. 1). The remaining four studies were excluded from the latter meta-analysis due to lack of CAL data in accordance with smoking status.13,38,40,41 For both outcomes (PD and CAL), no evidence of publication bias was demonstrated across the included studies (see Supplementary Figs. 3 and 4 in online Journal of Periodontology).

Figures 2 and 3 present the forest plots and summary estimates for weighted mean differences of PD and CAL between smokers and non-smokers, respectively. Tests for heterogeneity demonstrated non-significant heterogeneity for both PD (q = 3.85, degrees of freedom = 7, P = 0.80, I² = 0%) and CAL (q = 0.58, degrees of freedom = 3, P = 0.58, I² = 0%). The pooled effect sizes for reduction in PD were found to be highly significant in favor of non-smokers in the range of 0.39 mm (P < 0.001). Similarly, 0.35 mm more CAL gain was observed in non-smokers.11,13-15,38-41

Table 3.

Outcomes Assessment of Included Studies (n or mean ± SD)

<table>
<thead>
<tr>
<th>Reference</th>
<th>Smokers</th>
<th>Non-Smokers</th>
<th>Smokers</th>
<th>Non-Smokers</th>
<th>Smokers</th>
<th>Non-Smokers</th>
<th>Smokers</th>
<th>Non-Smokers</th>
<th>Smokers</th>
<th>Non-Smokers</th>
<th>Smokers</th>
<th>Non-Smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preber and Bergström et al.41</td>
<td>24</td>
<td>25</td>
<td>NA</td>
<td>0.76 ± 0.36</td>
<td>NA</td>
<td>1.27 ± 0.43</td>
<td>NA</td>
<td>1.27 ± 0.38</td>
<td>NA</td>
<td>1.0 ± 0.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kaldahl et al.11</td>
<td>Heavy smokers = 31, light smokers = 15</td>
<td>Non-smokers = 18, past smokers = 10</td>
<td>6</td>
<td>1.30 ± 0.10</td>
<td>0.65 ± 0.1</td>
<td>12</td>
<td>1.70 ± 0.20</td>
<td>1.0 ± 0.10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boström et al.13</td>
<td>Non-smokers = 17, past smokers = 20</td>
<td>NA</td>
<td>0.9 ± 1.70</td>
<td>NA</td>
<td>NA</td>
<td>1.6 ± 1.83</td>
<td>non-smokers = 1.2 ± 1.40</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scabia et al.15</td>
<td>28</td>
<td>29</td>
<td>22 ± 23</td>
<td>1.9 ± 0.7</td>
<td>1.20 ± 0.70</td>
<td>25 ± 21</td>
<td>2.4 ± 0.9</td>
<td>1.60 ± 0.70</td>
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</tr>
<tr>
<td>Orbak et al.40</td>
<td>25</td>
<td>25</td>
<td>0.45 ± 0.45</td>
<td>1.21 ± 0.67</td>
<td>NA</td>
<td>0.64 ± 0.41</td>
<td>1.58 ± 0.67</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trombelli et al.14</td>
<td>19</td>
<td>12</td>
<td>28 ± 24</td>
<td>1.90 ± 1.40</td>
<td>1.0 ± 1.30</td>
<td>33 ± 23</td>
<td>1.80 ± 1.30</td>
<td>1.30 ± 1.10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hellström et al.38</td>
<td>17</td>
<td>13</td>
<td>54 ± 4</td>
<td>2.05 ± 0.09</td>
<td>NA</td>
<td>59 ± 6</td>
<td>2.37 ± 0.22</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kim et al.39</td>
<td>3</td>
<td>Non-smokers = 5, past smokers = 7</td>
<td>NA</td>
<td>0.80 ± 0.86</td>
<td>0.09 ± 0.44</td>
<td>NA</td>
<td>0.95 ± 0.87</td>
<td>0.29 ± 0.31</td>
<td></td>
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</tr>
</tbody>
</table>

GI = gingival index, NA = information not available.
DISCUSSION

The present study is, to the authors’ knowledge, the first meta-analysis to assess the magnitude of therapeutic effect of periodontal flap surgical procedures in terms of non-smokers versus smokers. Results reveal that although periodontal flap procedures performed in active smokers generally moderately improved clinical measures of periodontal disease, the magnitude of benefit was significantly less than that observed in non-smokers. Comparison of weighted estimates among the included studies demonstrated that non-smokers exhibited a 0.4-mm greater reduction in mean PD response following surgical treatment compared with smokers. A similar difference in enhanced gain of CAL for non-smokers was also noted.

The present results support the 2007 findings of Johnson and Guthmiller, who compiled the information available on periodontal therapy in smokers and concluded that smokers do benefit from periodontal surgical procedures. The same authors also suggested that periodontal surgery may be recommended to smokers for pocket reduction, but the results should be expected to range from 50% to 75% of those observed in non-smokers. The present meta-analysis verified these findings and also provided a quantitative estimate of the magnitude of the difference within groups for PD and CAL. The 0.4-mm difference in PD reduction in smokers versus non-smokers is not only statistically significant but also clinically relevant. Previous studies have shown that a PD reduction in this range can significantly increase the clinical response to treatment.

Williams et al. found that the additional benefit of a 0.3-mm reduction in PD, when using minocycline microspheres as an adjunct to scaling and root planing, significantly improved the response to treatment. Hellström et al. reported that the use of a local antimicrobial following a modified Widman flap further reduced the mean PD by 0.3 mm beyond the effect of modified Widman flap, which led to significantly more pockets experiencing >2-mm PD reduction. Thus, the calculated 0.4-mm greater reduction in mean PD observed in non-smokers in the present study is also expected to translate to a greater number of sites meeting or exceeding the threshold of 2-mm reduction in PD.

The strength of the aforementioned results is supported by the very narrow 95% CIs found for the weighted differences in PD and CAL in the meta-analyses (−0.45 to −0.33 and −0.39 to −0.30, respectively). Based on the lack of heterogeneity across the included studies, it is unlikely that the reduced healing response noted in smokers was a result of chance. Therefore, clinicians could interpret these findings as a guideline for preoperatively setting smokers’ expectations. Because the mean magnitude of expected PD reduction after periodontal surgery has been shown to not exceed 3 mm, a reduced effect in smokers may account for fewer pockets with a baseline depth of ≥7 mm that achieve adequate reduction. In such cases, it may be advisable to inform smokers before periodontal flap surgery that additional therapy may be required.

A limitation of this study on an outcome level is the lack of standardized reporting of smoking status in the included studies. Most of the studies reported...
arbitrarily selected thresholds for the inclusion of smokers that ranged from \( \geq 1 \) to \( \geq 20 \) cigarettes per day.\(^{39,41} \) Although this variation in smoking dosage may be expected to strengthen or attenuate the deleterious effect of smoking on periodontal healing accordingly, Javed et al. previously reported that a dose-dependent effect of smoking in periodontal healing has not been consistently shown in published studies.\(^{23} \) In particular, Kaldahl et al. actually compared the response of heavy smokers, defined as smoking \( \geq 20 \) cigarettes per day, to that of light smokers (\( \geq 10 \) and \(< 20 \) cigarettes per day).\(^{11} \) Results from Kaldahl et al. showed a trend for less PD reduction in heavy smokers versus light smokers that was found to be significant only after 6 years of follow-up.\(^{11} \) One plausible explanation for the lack of a significant dose-dependent effect of smoking may be the high concentration of nicotine in the crevicular fluid even after limited exposure. Nicotine’s concentration in the crevicular fluid has been found to be \( \leq 300 \)-fold that of plasma.\(^{16} \) Thus, even light cigarette consumption may cause a disproportionately harmful effect to periodontal tissue healing. Future studies that will use established definitions of smoking are needed to definitively address the equivocal association between smoking duration and dosage and impaired periodontal healing.

One example of this is pack-years. A pack-year is quantified as the number of packs of cigarettes smoked per day, multiplied by the number of years the individual has been a smoker.\(^{45} \) Consequently, 1 pack-year means that a person has been smoking one pack (20 cigarettes) per day for a year, or approximately 7,300 cigarettes for a year.\(^{45} \) Using such an approach would allow for direct comparisons between studies and would most likely provide more information on the potential effect of dosage and/or duration on periodontal wound healing. Another implication for future research could be the use of the comprehensive smoking index (CSI).\(^{46} \) Similar to pack-years, the CSI provides a record of smoking duration and dosage. In addition to this information, CSI also captures information on the recency of smoking and allows for the estimation of the half-life of the smoking effect.\(^{46} \) Using the CSI may be advantageous in periodontal research, as the robustness of this index for estimating the effect of smoking on periodontal health has been previously verified.\(^{46} \)

**CONCLUSIONS**

Overall, periodontal surgical treatment benefited patients irrespective of smoking status. However, the magnitude of the therapeutic effect was significantly more beneficial in non-smokers compared with smokers in terms of both PD and CAL. Clinicians should use findings of the present study during preoperative treatment planning to caution smokers about the potential need for further therapeutic interventions following periodontal flap surgery.

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262


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