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Research Article

Predictors of periodontal pocket depth reduction in smokers with dentin hypersensitivity

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Abstract

Background: Smokers undergoing non-surgical periodontal therapy show smaller pocket depth reductions than non-smokers [13,14], and the same patients, who present with more baseline root exposure, are disproportionately prone to dentin hypersensitivity once instrumentation exposes their dentinal tubules [4].

Objective: This review synthesizes the literature on predictors of periodontal pocket depth reduction in smokers and situates dentin hypersensitivity as a co-occurring outcome.

Methods: We synthesized clinical trials, individual patient data meta-analyses, mixed-effects cohort studies, and mechanistic studies addressing smoking, systemic disease, and dentin hypersensitivity after periodontal therapy.

Findings: Baseline pocket depth is the strongest single predictor of reduction [10,11]; the smoking effect is graded, with nicotine dependence and cigarette count independently predicting smaller reductions [15]; diabetes and poor compliance compound rather than substitute for smoking [20,11]; cessation moves outcomes toward never-smoker levels [18]; and hypersensitivity tracks the recession burden smokers carry into treatment [1,22].

Conclusions: Clinicians should assess baseline depth, nicotine dependence, diabetes status, and recession extent together, and anticipate a desensitizing step for smokers, since no study has modelled both outcomes jointly in one cohort [21,15].

Keywords: periodontal pocket depth; smoking; dentin hypersensitivity; non-surgical periodontal therapy; nicotine dependence

1. Introduction

Non-surgical periodontal therapy remains the first line of treatment for chronic periodontitis, and probing pocket depth reduction after scaling and root planing is the outcome by which most clinicians judge whether treatment has worked. For a sizeable minority of treated patients it also arrives bundled with an unexpected complaint: newly exposed root surfaces become reactive to cold, sweet, and tactile stimuli, and dentin hypersensitivity emerges or worsens in the weeks after instrumentation even as periodontal parameters improve [1]. Smokers occupy an uncomfortable intersection of both problems. Tobacco use is the most consistently replicated modifiable risk factor for periodontal disease severity and for a blunted response to therapy [2,3], and smokers also tend to present with more baseline root exposure and recession, which independently predisposes them to hypersensitivity once instrumented [4]. Understanding what predicts pocket depth reduction in this population matters for treatment planning, for setting realistic expectations, and for deciding who needs adjunctive desensitizing measures built into the sequence rather than added as an afterthought.

The mechanistic case for why smoking blunts periodontal healing is well established even if no single pathway accounts for the whole effect. Nicotine and other compounds in cigarette smoke impair gingival microvascular perfusion through chronic vasoconstriction, altering delivery of oxygen and immune effectors to the healing wound bed [5]. At the cellular level, smoke condensate and nicotine both suppress periodontal ligament fibroblast attachment in vitro [6], promote collagen degradation through matrix metalloproteinase pathways [7], and inhibit the myofibroblast differentiation wound contraction depends on [8]. These translate into a clinical literature that has, since at least the late 1990s, found smokers healing less completely and quickly after the same instrumentation their non-smoking counterparts receive [9].

What has been less consistently synthesized is how smoking status as a modifier of pocket depth reduction and dentin hypersensitivity as a co-occurring sequela relate to each other and to the other variables clinicians juggle chairside: baseline pocket depth, diabetes status, plaque control, site anatomy, and nicotine dependence intensity rather than simple smoker/non-smoker status. This review synthesizes the published literature on predictors of pocket depth reduction in smokers, situates hypersensitivity within that same treatment window, and draws out the predictors that appear most reproducibly across the included studies.

2. Methods

We conducted a narrative synthesis of peer-reviewed literature addressing three overlapping questions: which patient- and site-level factors predict probing pocket depth reduction after non-surgical periodontal therapy; how smoking status, intensity, and nicotine dependence specifically modify that response; and how dentin hypersensitivity behaves as a co-occurring outcome in the same treatment window, particularly in smokers with greater baseline root exposure. Relevant records were identified through PubMed, Crossref, and citation snowballing from recent systematic reviews and meta-analyses, prioritizing randomized and controlled trials, individual patient data meta-analyses, cohort studies with multivariable or mixed-effects modelling of pocket depth outcomes, and mechanistic in vitro studies where they clarified a plausible pathway for an observed clinical association. Twenty-two sources meeting these criteria are synthesized below. Because no single trial models smoking intensity, dentin hypersensitivity, and pocket depth reduction as co-primary outcomes in one cohort, this review integrates findings across studies rather than reporting a pooled estimate, and flags where a given predictor rests on a single study rather than a converging body of work.

3. Results

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Baseline pocket depth and site-level anatomy as the dominant predictors

Across the studies that modelled pocket depth reduction as a continuous or ordinal outcome, baseline probing depth itself was consistently the strongest single predictor of subsequent reduction, robust enough that it tends to be entered as a covariate before any smoking-related term is considered. Werner and colleagues, in a retrospective analysis of 746 patients treated with non-surgical therapy, reported an overall mean probing depth reduction of 1.20 mm (SD 1.51 mm) across stratified baseline depths, but found that sites at 6 mm or deeper improved distinctly less relative to their starting depth than shallower sites, and that tooth type and site position independently modified the response even after baseline depth was accounted for [10]. Kaisaier and colleagues, applying a linear mixed-effects model to a periodontitis cohort, likewise identified baseline pocket depth as a leading prognostic factor for subsequent reduction, alongside smoking status, diabetes, and plaque control as independent contributors [11]. The effect is partly regression to the mean and partly biological: deeper pockets carry more subgingival biofilm to begin with, so instrumentation removes a larger absolute burden, but they also carry more residual disease risk after treatment, which is why residual pocket depth has itself become a recommended endpoint for judging when supportive care needs to intensify [12].

Smoking intensity, nicotine dependence, and the pocket-depth response

Table 1 and Table 2 summarize the smoking-related evidence base. The broadest signal comes from Leite and colleagues' systematic review and meta-regression, which found a dose-response relationship between cigarette consumption and periodontitis severity across the pooled epidemiological literature, with heavier smoking associated with greater probing depth and attachment loss even before any treatment is considered [3]. Once treatment enters the picture, Chambrone and colleagues' individual patient data meta-analysis of smoking cessation trials found that current smokers achieved smaller probing depth reductions after non-surgical therapy than never-smokers, with the gap between groups most pronounced at initially deep and moderately deep sites, and that patients who quit smoking during or shortly after therapy trended toward outcomes closer to those of never-smokers than of persistent smokers [13]. Chang and colleagues' more recent systematic review and meta-analysis reached a concordant conclusion: smokers showed significantly smaller pocket depth reductions and higher residual probing depths after scaling and root planing than non-smokers across the pooled trials, a finding stable enough across heterogeneous study designs that the authors treated smoking status as a variable that should routinely be reported and adjusted for in periodontal treatment-outcome research going forward [14].

Table 1: Predictors of periodontal pocket depth reduction identified across the synthesized literature.

Predictor	Direction of association	Representative source(s)
Baseline pocket depth	Larger baseline depth associated with larger absolute reduction but more limited proportional improvement at deep sites	[10,11]
Smoking status / intensity	Current smoking, higher nicotine dependence, and higher cigarette count attenuate reduction	[13,14,15]
Smoking cessation	Former smokers show intermediate outcomes between current and never-smokers	[18,19]
Diabetes mellitus	Independently associated with poorer treatment outcome and lower long-term tooth survival	[20,11]
Tooth / site anatomy	Molar vs non-molar and interproximal vs buccal/lingual sites respond differently	[10]
Plaque control / compliance	Poorer hygiene and lower maintenance attendance associated with smaller, less durable reduction	[11,16]

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Table 2: Reported effect of smoking status and intensity on non-surgical periodontal therapy outcomes across key clinical studies.

Study	Design / population	Comparison	Key finding
[13]	Individual patient data meta-analysis, smoking cessation trials	Current smokers vs never-smokers	Smaller pocket depth reduction in current smokers, gap largest at deep and moderate sites
[14]	Systematic review and meta-analysis of SRP outcomes	Smokers vs non-smokers	Significantly smaller pocket depth reduction and higher residual depth in smokers
[15]	Cohort of smokers, non-surgical treatment	Nicotine dependence score and cigarettes/day as continuous predictors	Higher dependence and cigarette count associated with smaller pocket depth reduction
[9]	Retrospective clinical study	Smokers vs non-smokers	Smoking associated with poorer healing and more frequent escalation to surgical therapy
[16]	Supportive periodontal therapy cohort	Smokers vs non-smokers	Persistently elevated bleeding on probing in smokers despite comparable maintenance

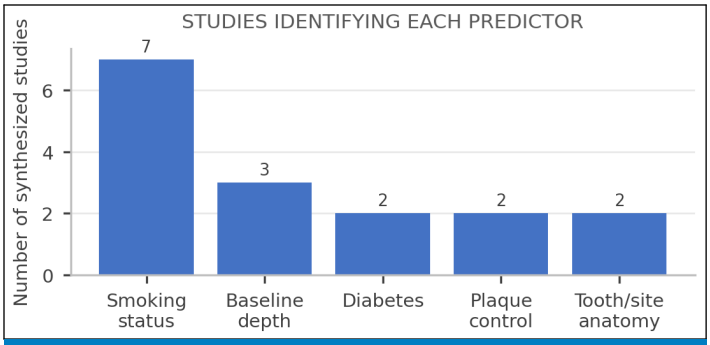

Figure 1: Number of studies synthesized in this review that identified each factor as an independent predictor of probing pocket depth reduction or periodontal treatment outcome, reflecting how consistently each predictor recurs across the included clinical and mechanistic literature [3,13,14,15,9,2,16,10,11,12,20,1].

What moves this literature from a binary smoker/non-smoker framework toward something clinically actionable is more granular dose modelling. Salhi and colleagues modelled probing depth reduction specifically in a cohort of smokers, using nicotine dependence scores and self-reported daily cigarette consumption as continuous predictors rather than treating "smoker" as a single category, and found that greater dependence and higher daily counts were each associated with smaller reductions, indicating the smoking effect on healing is graded rather than a threshold phenomenon [15]. This is consistent with Papantonopoulos' earlier retrospective study, where smoking status influenced not only measured healing but the treatment decisions clinicians made, smokers being more likely escalated to surgical therapy after an inadequate response [9], and with Ramseier and colleagues' finding that smokers in supportive periodontal care showed persistently elevated bleeding scores despite comparable maintenance schedules, suggesting the effect persists well past the active-therapy phase [16]. A recent trial by Pupovac and colleagues tested whether adding a chlorhexidine-based antiseptic could narrow this gap, finding that while the antiseptic improved several parameters overall, tobacco exposure continued to exert an independent, only partly attenuated effect [17].

Smoking cessation, systemic disease, and compliance as modifiers

Smoking cessation studies provide a useful natural experiment for whether the smoking effect on pocket depth reduction is reversible on a clinically meaningful timescale. Caggiano and colleagues' systematic review of smoking cessation and periodontal and peri-implant health found that former smokers generally occupied an intermediate position between current smokers and never-smokers on most periodontal outcome measures, with the degree of improvement after quitting related to how long the patient had been abstinent and how much periodontal damage had already accrued before cessation [18], a pattern broadly consistent with Madi and colleagues' narrative review of smoking's influence on periodontal and implant therapy outcomes, which emphasized that cessation counseling delivered as part of periodontal treatment planning is one of the few interventions clinicians can offer that plausibly shifts a patient's healing trajectory rather than merely working

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around it [19]. The mechanistic literature on nicotine's direct effects on periodontal ligament and gingival fibroblasts supports a biologically reversible model, since nicotine's suppression of fibroblast attachment and myofibroblast differentiation in vitro is a pharmacological rather than a permanently structural effect [6,8], though clinical recovery in patients with established attachment loss will always be bounded by the anatomical damage already present.

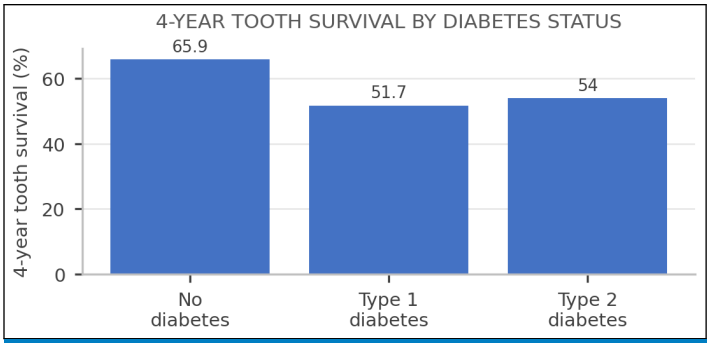

Figure 2: Four-year tooth survival after periodontal treatment by diabetes status, drawn from a large-scale administrative claims analysis of over 400,000 treated patients, 65.9% in patients without a diabetes diagnosis against 51.7% in patients with type 1 diabetes and 54.0% in medicated type 2 diabetics [20].

Systemic disease compounds the smoking effect rather than substituting for it. Raedel and colleagues' analysis of over 400,000 patients found that diabetes diagnosis was independently associated with poorer outcomes after periodontal treatment, with four-year tooth survival of 65.9% in non-diabetic patients against 51.7% in patients with type 1 diabetes and 54.0% in medicated type 2 diabetics (Figure 2), a gap of a magnitude comparable to, and additive with, the smoking effect reported elsewhere in this literature [20]. Kaisaier and colleagues' mixed-effects model similarly retained diabetes as an independent negative predictor of probing depth reduction alongside smoking and baseline depth, rather than finding that either variable explained the other away [11]. Plaque control and patient compliance with maintenance visits behaved the same way across the studies that measured them: patients with poorer self-performed hygiene and lower attendance at supportive care appointments showed smaller and less durable pocket depth reductions regardless of smoking status, though smokers were disproportionately represented among poor compliers in several of the included cohorts, which makes disentangling the independent contribution of compliance from that of smoking itself genuinely difficult with observational data alone [11,16].

Dentin hypersensitivity as a co-occurring outcome of the same treatment window

Root instrumentation removes cementum, exposes dentinal tubules, and, in patients with pre-existing gingival recession, does so over a larger area of already-exposed root surface, precisely the anatomical substrate dentin hypersensitivity requires. Schwarz and colleagues demonstrated that a targeted intervention, an Er:YAG laser applied to hypersensitive dentin, produced measurable and sustained reductions in stimulus-evoked pain scores, confirming that the tubule-occlusion mechanism underlying most desensitizing approaches is clinically exploitable in a periodontal population [21]. Giassin and colleagues examined sensitivity across both non-surgical and surgical treatment phases and found it typically rose in the weeks immediately following instrumentation before resolving over the following months, with an arginine- and calcium-carbonate-based desensitizing agent producing modestly faster resolution than placebo [1]. Ohanessian and colleagues, studying hypersensitivity alongside gingival recession and root coverage procedures, found that pre-existing root exposure and the success of root coverage were both meaningful determinants of whether hypersensitivity resolved, reinforcing that recession extent, which smokers accumulate disproportionately, is itself a predictor worth tracking alongside pocket depth [22]. Nausheen and colleagues' cross-sectional survey found the condition common enough, and its risk factors (recession, erosive diet, aggressive brushing) broad enough, that it should be actively screened for rather than left as an incidental

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complaint [4]. Taken together, dentin hypersensitivity in smokers undergoing periodontal therapy is not an unrelated nuisance but a largely predictable consequence of the same recession burden and instrumentation intensity that shapes their pocket depth outcome, arguing for a desensitizing step built into the treatment sequence rather than added after the complaint arises.

4. Discussion

Three findings recur clearly enough across this literature to be treated as reasonably settled. First, baseline pocket depth is the strongest determinant of the magnitude of subsequent reduction, with smoking status and every other predictor here operating as modifiers of that relationship rather than competitors to it [10,11]. Second, the smoking effect is graded rather than categorical: nicotine dependence severity and daily cigarette consumption both carry independent predictive weight beyond a simple ever-smoker classification, so treatment-planning conversations that reduce smoking to a yes-or-no checkbox discard information a brief dependence-screening question could recover [15,3]. Third, the effect is at least partially reversible with cessation, and former smokers occupy a consistently intermediate position between current smokers and those who never used tobacco, giving clinicians a genuine reason to fold cessation counseling into periodontal treatment planning rather than treating it as a separate concern [18,13].

Where the evidence is thinner is in the direct intersection of smoking and dentin hypersensitivity as jointly modelled outcomes. No study identified here enrolled smokers specifically to track hypersensitivity and pocket depth reduction together as co-primary endpoints; the two literatures have largely developed in parallel, connected mechanistically through shared anatomical substrate but rarely tested together in the same cohort [1,4]. This is a genuine gap rather than a settled null result: the inference that smokers with substantial baseline recession are more likely to need active desensitizing management layered onto a periodontal plan that will itself likely underperform rests on combining two separately well-supported literatures rather than a single confirmatory trial. Diabetes and compliance add further complexity that the included studies handle unevenly, some modelling them as independent covariates alongside smoking [11,20], others unable to fully separate a compliance effect from a smoking effect because the two correlate in their study populations [16].

The practical implication is to treat baseline pocket depth, nicotine dependence intensity, diabetes status, and recession extent as a bundle of predictors assessed together at the initial visit, rather than defaulting to a single smoking-status flag, and to set outcome expectations and desensitizing protocols accordingly.

5. Conclusion

Across the synthesized literature, pocket depth reduction after non-surgical periodontal therapy in smokers is most reliably predicted by baseline pocket depth, smoking intensity and nicotine dependence rather than smoker status alone, diabetes status, and plaque control and maintenance compliance, with cessation offering a genuine, evidence-supported route toward outcomes closer to those of never-smokers [10,15,13,20,18]. Dentin hypersensitivity in the same population tracks the recession burden and instrumentation intensity that smokers disproportionately carry into treatment, and while it has rarely been modelled jointly with pocket depth outcomes, the evidence converges closely enough to justify building a desensitizing step into the treatment sequence whenever a smoker with visible recession presents for therapy [21,1,22]. Prospective studies enrolling smokers across a range of nicotine dependence severities and tracking both outcomes together would close the most consequential remaining gap.

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8. Conflict of Interest

The authors declare no conflict of interest.

9. Author Contributions

All authors contributed to the conception of the review, the literature search and synthesis, drafting and critical revision of the manuscript, and approved the final version for submission.

10. Ethics Statement

This article is a narrative synthesis of previously published literature and did not involve new data collection from human or animal subjects; institutional ethics review was therefore not required.

11. Data Availability

No new data were generated for this review. All findings discussed are drawn from the published sources cited in the reference list.

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