ORIGINAL ARTICLE

Smoking as a Prognostic Modifier in Endodontic Care: A Mixed-Methods Study of the Evidence-Practice Gap

ABSTRACT

Aim *This study aimed to evaluate the evidence-practice gap regarding smoking as a potential prognostic modifier in endodontic care, focusing on tobacco-use assessment, prognostic reasoning, risk communication, and reported clinical decision-making.*

Methodology *A sequential mixed-methods study with a qualitative follow-up phase was conducted with dentists involved in endodontic care. In the quantitative phase, 201 dentists completed an online questionnaire. Associations between professional setting and clinical behaviours were analysed using Pearson's chi-square test, with Bonferroni and Benjamini–Hochberg corrections and Cramer's V effect sizes. All quantitative participants were subsequently invited to the qualitative follow-up; 30 accepted the invitation and completed the open-ended questions, which were analysed using reflexive thematic analysis. Integration occurred at the interpretative stage by examining qualitative themes alongside the aggregate quantitative response patterns.*

Results *Among the 201 participants, 111 (55.2%) routinely asked about smoking status, whereas 57 (28.4%) assessed smoking frequency, 59 (29.4%) adapted case explanation according to smoking status, and 97 (48.3%) associated smoking with case prognosis. Only 56 (27.9%) reported prior awareness of the smoking–periapical healing relationship described in the questionnaire. No association between professional setting and clinical behaviour remained statistically significant after correction for multiple testing. The qualitative findings provided interpretative context for these quantitative patterns, showing variable depth of smoking-history assessment and inconsistent integration into prognostic reasoning and risk communication.*

Conclusions *The findings indicate inconsistencies between documenting tobacco use and clinically interpreting this information in endodontic care. The principal evidence-practice gap concerned the depth of tobacco-use assessment, prognostic reasoning, and risk communication rather than the absence of a specific smoking-related technical modification to endodontic treatment. More consistent documentation and risk communication may be warranted, while the clinical implications of smoking-specific management strategies require further prospective investigation.*

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Introduction

Tobacco smoking remains an important determinant of oral morbidity and is associated with impaired tissue healing and less predictable outcomes in several areas of dentistry. It is also a well-established risk factor for periodontal disease and impaired tissue healing (1–2). In Endodontics, growing evidence suggests an association between tobacco use and a higher prevalence of periapical lesions and potentially less favourable outcomes following root canal treatment (3–7).

However, findings across endodontic studies remain heterogeneous, and causality has not been established (4–6,8–9). Smokers have been reported to be approximately 2.4 times more likely to present apical periodontitis or a history of endodontic treatment, although this association does not establish a causal relationship (4). Consequently, the recognition and incorporation of smoking as a potential prognostic factor in clinical endodontic practice remain insufficiently characterised (8).

This study did not aim to test biological outcomes directly. Instead, it examined how evidence on tobacco-related risks is recognised, interpreted, and incorporated into routine endodontic decision-making. Characterising this potential disconnect may help clarify how tobacco-related evidence is interpreted in clinical practice and identify barriers to more consistent history taking, risk communication, and evidence-informed decision-making in Endodontics (10).

Therefore, this study aimed to evaluate the evidence-practice gap regarding smoking as a potential prognostic modifier in endodontic care, focusing on how tobacco-use history is assessed and incorporated into prognostic reasoning, risk communication, and reported clinical decision-making.

Methods

Ethical considerations

The study was approved by the Research Ethics Committee of the University of Fortaleza (no. 6,482,408), in accordance with Resolution 466/2012 of the Brazilian National Health Council and the ethical standards laid down in the 1964 Declaration of Helsinki. All participants provided informed consent electronically before accessing the questionnaire.

Study design

The questionnaire was developed specifically for this study through an iterative consensus-based process involving five investigators with academic and/or clinical experience in Endodontics, led by the principal investigator, an endodontist. The proposed items were reviewed internally against the prespecified conceptual and clinical assumptions of the study, and wording was refined through discussion to enhance clinical relevance, clarity, and comprehensibility. No formal pilot testing, psychometric validation, or quantitative face/content-validity assessment was performed. Following the quantitative phase, all 201 respondents were invited to participate in a qualitative follow-up designed to provide additional interpretative depth regarding dentists' perceptions of the relationship between smoking and periapical disease and its relevance to clinical reasoning. Thirty participants accepted the invitation and completed the qualitative phase. Participants were not selected according to their previous quantitative responses or professional category. The qualitative questions explored the same prespecified conceptual domains in greater depth; participants were not informed of the quantitative findings, and integration of the two datasets occurred at the interpretative stage rather than through independent confirmation of one dataset by the other.

Sample characterisation
The quantitative sample comprised dentists involved in endodontic care in public and/or private clinical settings in Fortaleza, Ceará, Brazil, including public dental services, Specialised Dental Centres, university-based professionals with endodontic expertise, and clinicians working in private practice. The initial sample was recruited by invitation, targeting dentists actively involved in endodontic care. For the qualitative follow-up, all 201 quantitative respondents were invited, and 30 accepted and provided responses to the open-ended questions. The qualitative sample therefore reflected participation uptake rather than investigator selection according to quantitative responses. During analysis, the resulting dataset was considered by the research team to provide sufficient depth, diversity, and informational adequacy to address the qualitative aims, as additional interpretative categories were not required to account for the range of meanings present in the responses.

Data collection
Data were collected using an online form (forms.google.com). Given the relatively identifiable professional community under investigation, demographic variables such as age, sex, and years of experience were intentionally omitted to maximise anonymity and encourage candid reporting of clinical behaviours. This approach supported confidentiality and may have reduced prestige-related response bias, while limiting the assessment of individual-level influences. In the qualitative stage, the form included open-ended questions exploring participants' perceptions of the potential relationship between smoking and periapical lesions, their approach to investigating smoking habits, and their views on including tobacco-use assessment in endodontic anamnesis. Participants were not informed of the quantitative findings or of the response patterns observed in the first phase before completing the qualitative questions.

Sample size calculation
Sample size was calculated using R Studio (v. 2024.12.0+467; Posit Software, PBC). A chi-square test of independence with two degrees of freedom (df = 2), a type I error of 5% ($\alpha = 0.05$), and a statistical power of 80% ($1-\beta = 0.80$) were considered. An expected small-to-medium effect size ($w = 0.22$) was adopted, based on Cohen's theoretical parameters and Cramer's V values from similar studies. These assumptions yielded a minimum required sample size of 196 participants to ensure adequate statistical power.

Statistical analysis
Data analysis was performed using R Studio (v. 2024.12.0+467; Posit Software, PBC). Descriptive results were expressed as absolute and relative frequencies. Associations between professional category and questionnaire responses were assessed using Pearson's chi-square test. Bonferroni and Benjamini–Hochberg adjustments were applied to account for multiple testing, and Cramer's V was calculated as an effect-size measure. Statistical significance was set at an adjusted $p < 0.05$.
Qualitative data from the open-ended questions were analysed using reflexive thematic analysis following Braun and Clarke's six-phase framework (11): (i) familiarisation with the responses, (ii) generation of initial codes, (iii) development of candidate themes, (iv) reviewing themes, (v) defining and naming themes, and (vi) producing the final report. Analytical matrices were developed in Google Sheets (sheets.google.com) to organise responses, codes, preliminary themes, and interpretative notes. Initial coding was led by the principal investigator, an endodontist with academic research experience. Code interpretation and theme development were subsequently discussed iteratively by the full eight-member research team, which included researchers with backgrounds in Endodontics, Public Health, Medicine, and academic research. The multidisciplinary

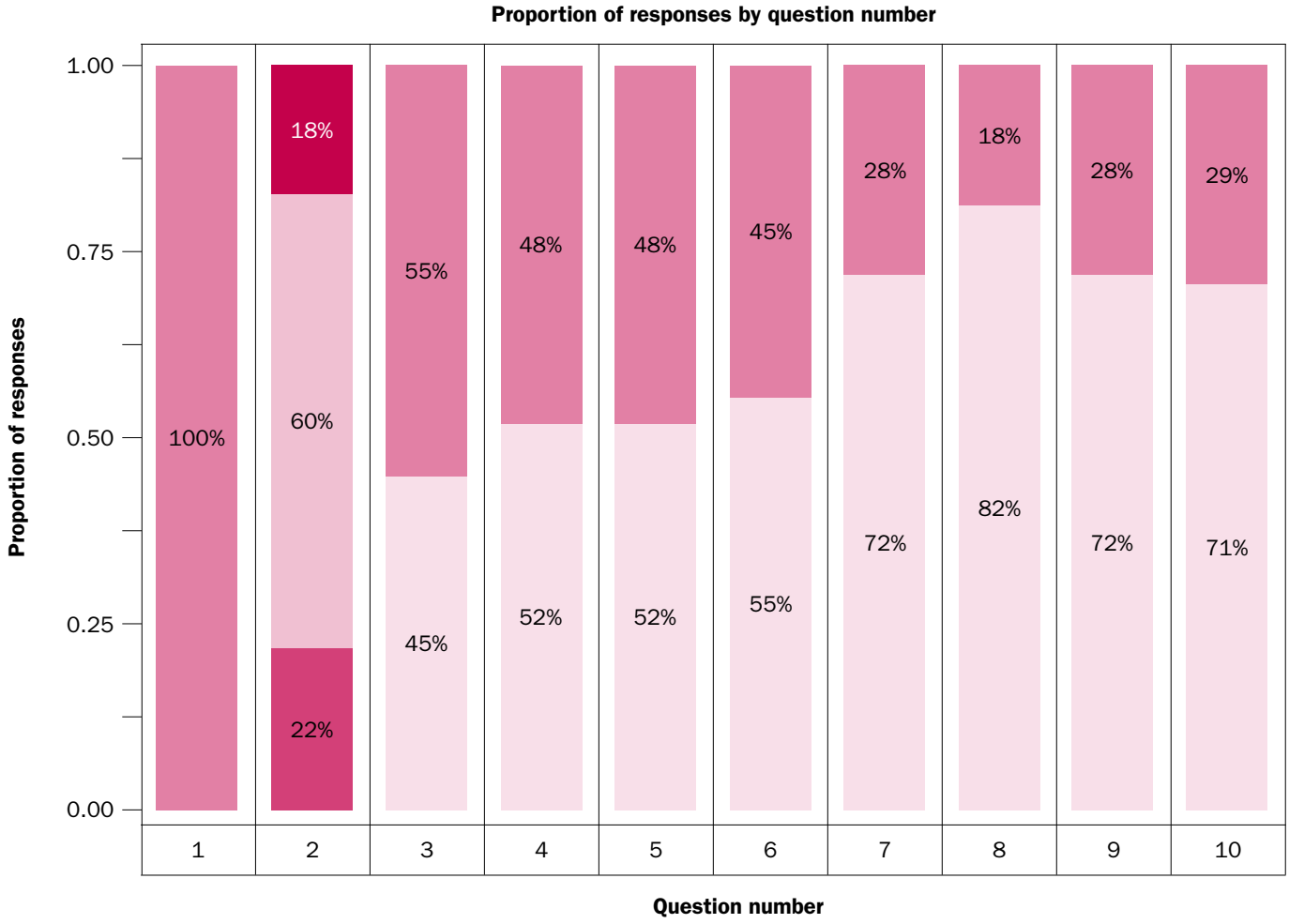


Figure 1
Distribution of responses across questionnaire items.

composition of the team broadened the interpretative perspective and helped challenge assumptions arising from a solely endodontic viewpoint. Different interpretations were discussed and used to refine code boundaries, thematic meanings, and relationships among themes rather than to establish inter-coder reliability, consistent with a reflexive thematic-analysis approach. The researchers recognised that prior clinical and academic familiarity with the smoking–endodontic literature could influence interpretation, and this positioning was considered throughout theme development and refinement.

Results

Responses from 201 dentists (100%) working across different service settings were analysed (Figure 1). The first two items referred to informed consent (Q1) and professional category (Q2), respectively. Q1 documented eligibility and consent, whereas Q2 was used for sample characterisation and as the grouping variable in the association analyses. All 201 participants (100%) confirmed having read and agreed to the informed consent form. Among respondents, 121 (60.2%) worked exclusively in private practice, 45 (22.4%) worked in both public and private sectors, and 35 (17.4%) worked exclusively in the public sector. Questions Q3–Q10 addressed clinical behaviours and perceptions related to smoking in endodontic contexts (Table 1).

Response distribution

Table 2 presents the absolute and relative

Code	Question
Q1	Have you read the informed consent form and do you agree to participate in the study?
Q2	Do you work in the public or private sector?
Q3	Before performing an endodontic treatment, do you inquire whether the patient is a smoker?
Q4	Do you ask about the patient's smoking frequency?
Q5	If the patient is an ex-smoker, do you inquire how long they have been abstinent?
Q6	If the patient is an ex-smoker, do you ask for how long they had smoked?
Q7	If the patient is a smoker, do you adopt different strategies in case explanation?
Q8	If the patient is a smoker, do you modify any aspects of the treatment?
Q9	If the patient is a smoker, do you associate smoking with case prognosis?
Q10	The scientific literature indicates that smoking negatively affects the healing of periapical lesions in endodontically treated teeth. Prior to completing this questionnaire, were you aware of this fact?
Note: Q8 and Q10 are reproduced verbatim as administered. Q8 did not specify the nature of treatment modification and was interpreted descriptively. Q10 described the smoking-periapical healing relationship more definitively than warranted by the heterogeneous evidence and referred to the relationship as a "fact"; this wording is acknowledged as a study limitation.	

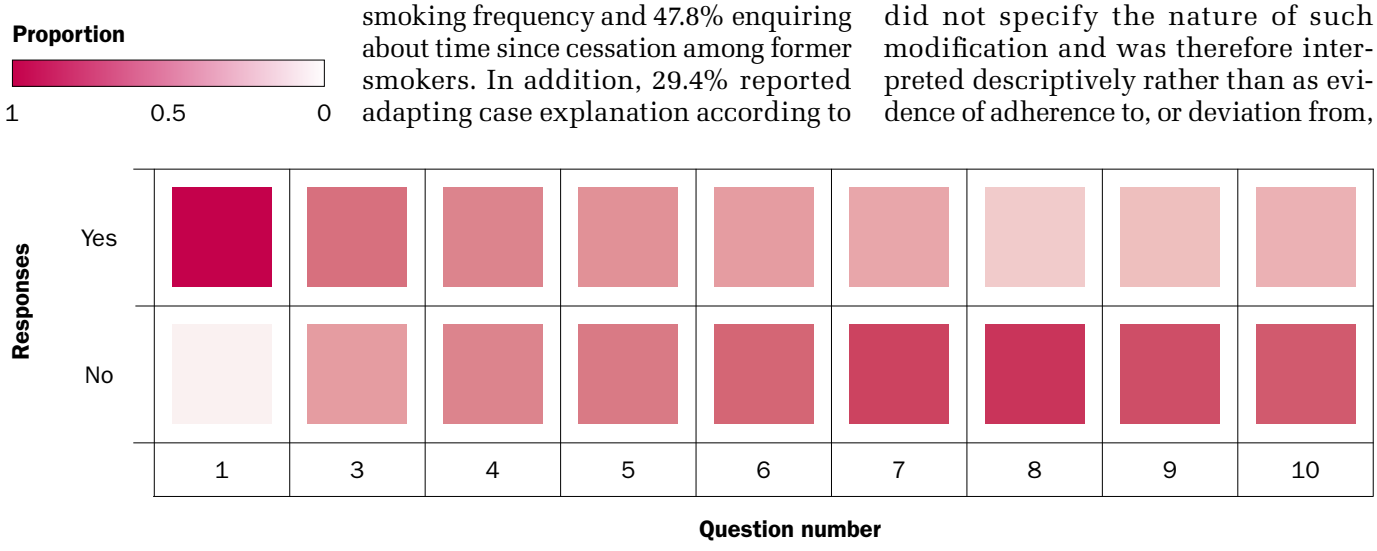
Table 1
Structure and content of the questionnaire items administered to participants

Table 2
SDistribution of responses to clinical items.

Figure 2
Heatmap of binary responses.

Question	No (n, %)	Yes (n, %)
Q3	90 (44.8%)	111 (55.2%)
Q4	144 (71.6%)	57 (28.4%)
Q5	105 (52.2%)	96 (47.8%)
Q6	110 (54.7%)	91 (45.3%)
Q7	142 (70.6%)	59 (29.4%)
Q8	164 (81.6%)	37 (18.4%)
Q9	104 (51.7%)	97 (48.3%)
Q10	145 (72.1%)	56 (27.9%)

frequencies of responses (Q3–Q10), also illustrated in Figures 1 and 2. Among participants, 55.2% reported routinely asking about smoking status before endodontic treatment. More detailed smoking assessment was less frequent, with 28.4% reporting assessment of smoking frequency and 47.8% enquiring about time since cessation among former smokers. In addition, 29.4% reported adapting case explanation according to smoking status, and 48.3% associated smoking with case prognosis. Only 27.9% reported prior awareness of the smoking–periapical healing relationship presented in Q10. Although 18.4% reported modifying some aspect of treatment according to smoking status, Q8 did not specify the nature of such modification and was therefore interpreted descriptively rather than as evidence of adherence to, or deviation from,



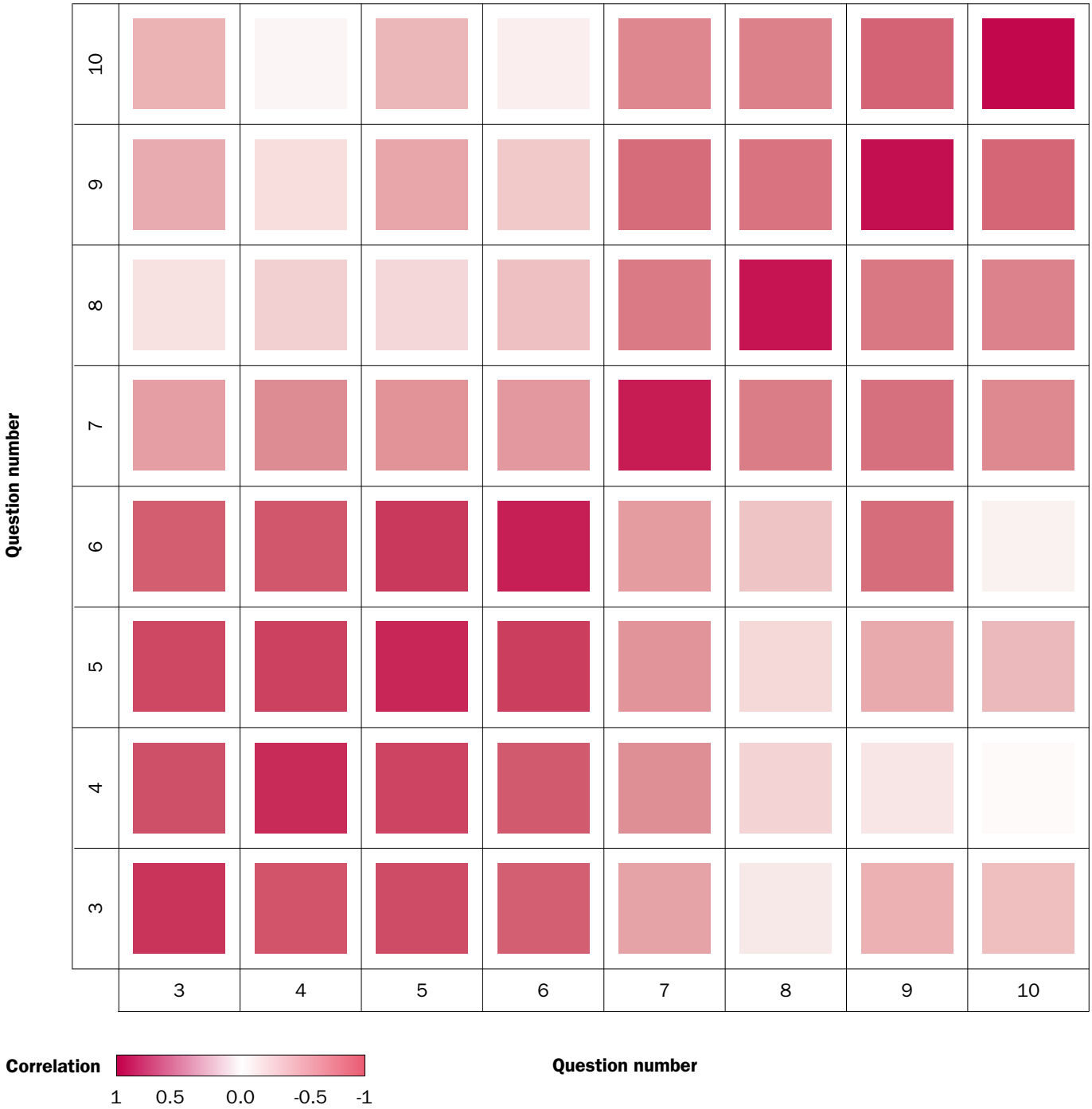


Figure 3
Correlation heatmap among questionnaire items (Q3–Q10).

an established smoking-specific treatment recommendation.

Associations

Table 3 details the associations between professional category and clinical behaviours. None of the associations remained statistically significant after correction for multiple testing using either the Bonferroni or Benjamini–Hochberg procedures (all adjusted $p \geq 0.058$). Cramer's V values ranged from 0.126 to 0.214, indicating small effect sizes. Accordingly, no differences between professional settings were inferred from these analyses.

Table 3
Association between professional category and clinical behaviours (Pearson's Chi-square test).

Question	X^2 (df=2)	p (original)	p (Bonferroni)	p (BH)	Cramer's V
Q3	4.64	0.098	0.787	0.112	0.152
Q4	7.23	0.027	0.215	0.058	0.190
Q5	7.09	0.029	0.231	0.058	0.188
Q6	7.07	0.029	0.234	0.058	0.188
Q7	3.19	0.202	1.000	0.202	0.126
Q8	6.34	0.042	0.336	0.066	0.178
Q9	6.01	0.050	0.396	0.066	0.173
Q10	9.19	0.010	0.081	0.058	0.214
Note: No association remained statistically significant after either Bonferroni or Benjamini-Hochberg correction. Cramer's V values ranged from 0.126 to 0.214.					

Thematic analysis

Qualitative responses from 30 dentists were analysed according to Braun and Clarke's six-phase framework (11). Participant-level characteristics, response summaries, and code assignments are presented in Supplementary Table S1. The analytical audit trail is detailed in Supplementary Table S2, and the initial codebook is presented in Supplementary Table S3. Exemplar extracts are provided in Supplementary Table S4, the progression from initial codes to candidate and final themes in Supplementary Table S5, and additional illustrative extracts supporting the final themes in Supplementary Table S6.

Four final themes were developed: (i) perceived biological and prognostic relevance of smoking to periapical healing, reflecting participants' perceptions that smoking may affect vascularisation, immune response, bone repair, lesion regression, and endodontic prognosis; (ii) smoking history as present in anamnesis but weakly integrated into endodontic reasoning, indicating that tobacco use was often recorded, but not consistently explored in terms

Table 4
Final themes from the thematic analysis.

Theme	Analytic meaning	Codes informing theme*	Illustrative evidence	Justification
(1) Perceived biological and prognostic relevance of smoking to periapical healing	Participants who articulated a specific link described smoking through vascular, immune, inflammatory, and repair pathways that they perceived as potentially relevant to lesion regression and endodontic prognosis.	C03, C04, C05	P03: reduced vascularisation, immune compromise, difficult bone healing and lesion persistence. P05: reduced vascularisation, reduced immune response and delayed healing. P09: reduced immunity, bone resorption and poorer healing.	This theme captures how participants perceived smoking as potentially relevant to periapical healing and endodontic prognosis.
(2) Smoking history is often present in anamnesis but weakly integrated into endodontic reasoning	Although many participants said that smoking was asked about during anamnesis, only a smaller subset described detailed investigation or explicit correlation with endodontic findings.	C06, C07, C08, C09	P08: detailed history and correlation with clinical/radiographic findings. P20: smoking is recorded but not associated. P29: only asks whether the patient smokes.	This theme captures the practice gap between recording tobacco use and using it as a prognostic variable.
(3) Anamnesis as risk communication, individualised care and professional accountability	Participants justified tobacco-related questions as a way to guide treatment planning, align expectations, educate patients and document risks that may affect outcomes.	C10, C11, C12	P03: individualising conduct and aligning expectations. P13: adjusting treatment plans and anticipating unfavourable prognosis. P16: health promotion and legal support.	This theme captures participants' views that smoking-related anamnesis may support risk communication, expectation alignment, individualised planning, and documentation.
(4) Knowledge gaps and structural constraints limit implementation	Limited awareness of the smoking–periapical lesion relationship, uncertainty about evidence and work-context barriers restricted the clinical use of tobacco history.	C01, C02, C13, C14	P15: lacks specific knowledge and does not address it in practice. P22: necessary but not realistic in public service. P24: important but no time.	This theme helps explain why knowledge and clinical behaviour were inconsistent, but the detailed participant-level evidence is better suited to supplementary material.
*Codes are defined in detail in Supplementary Table S3; their development into candidate and final themes is presented in Supplementary Table S5.				

of frequency, duration, previous exposure, cessation, or clinical implications; (iii) anamnesis as risk communication, individualised care, and professional accountability, showing that smoking-related questions were considered relevant for patient counselling, expectation alignment, treatment planning, and documentation of risk factors; and (iv) knowledge gaps and structural constraints limiting implementation, underscoring limited lesion-specific knowledge, broader non-endodontic framings of smoking-related harm, and practical barriers such as time pressure and service-context limitations (Table 4).

Integrated interpretive summary

The quantitative and qualitative findings together indicated variability in both the depth of smoking assessment and the clinical interpretation of tobacco-use history. Although 55.2% of respondents reported asking whether patients smoked, substantially fewer reported assessing smoking frequency or adapting case explanation according to smoking status. No association between these behaviours and professional category remained statistically significant after correction for multiple testing. The qualitative findings provided additional context for these aggregate patterns. Some participants described detailed investigation of smoking status, frequency, duration, quantity consumed, previous exposure, and cessation history, whereas others described only superficial yes/no inquiry, simple documentation without endodontic interpretation, or no smoking-related investigation. Participants also varied in how they perceived the potential biological and prognostic relevance of smoking. Overall, the integrated findings suggest that the principal gap concerned the depth and interpretation of tobacco-use information in prognostic reasoning and risk communication rather than the absence of a specific smoking-related technical modification to endodontic treatment.

Discussion

Smoking is a well-established risk factor for several systemic and oral conditions and has been associated with impaired tissue healing in multiple clinical contexts. In Endodontics, available evidence suggests associations between tobacco use, a higher prevalence of periapical disease, and potentially less favourable healing following root canal treatment (1,3,5–9). However, findings remain heterogeneous, and the available observational evidence does not establish causality. The present study did not evaluate biological or treatment outcomes directly; rather, it examined how tobacco-related evidence is recognised, interpreted, and incorporated into reported endodontic clinical reasoning.

Beyond the existing systematic reviews (3–6), epidemiological and prospective clinical studies have reported associations between tobacco use, periapical disease, and endodontic outcomes (12–14). Population-based investigations have suggested smoking as a possible modifying factor in periapical pathosis rather than a direct causal agent, while some prospective studies have reported lower healing rates or greater postoperative symptomatology among smokers (12–14). These findings should nevertheless be interpreted within the context of the methodological heterogeneity and residual uncertainty identified in the broader literature. In the present qualitative data, some participants similarly perceived smoking as potentially relevant to vascularisation, immune response, tissue repair, bone healing, lesion persistence, and prognosis, whereas others reported little lesion-specific knowledge or framed smoking mainly through broader oral-health outcomes.

Despite uncertainty in the available evidence, smoking may reasonably be considered a potential prognostic variable worthy of documentation and clinical consideration (3–6,12–14). The study-specific questionnaire was de-

signed to capture reported tobacco-related assessment and clinical reasoning through concise response options (Table 1). The findings suggest inconsistency between the documentation of smoking status and the depth with which this information is investigated and interpreted. The qualitative component provided interpretative depth by showing that tobacco use could be present as a formal anamnesis item without necessarily being incorporated into endodontic-specific prognostic reasoning. Similar gaps between theoretical knowledge and clinical application have been reported in previous surveys conducted in other populations (8).

Evidence from other areas of dentistry and wound healing provides biological plausibility for a potential association between smoking and impaired periapical repair, particularly through vascular, inflammatory, immune, and bone-healing pathways (15–16). However, such cross-disciplinary evidence cannot by itself establish a smoking-specific causal effect on endodontic healing. In the qualitative data, several participants invoked these mechanisms when explaining why they considered smoking potentially relevant to endodontic prognosis, whereas others framed smoking primarily through broader oral-health consequences. This variation further supports interpreting the qualitative findings as participants' clinical reasoning rather than as biological confirmation of the proposed mechanisms.

The principal evidence-practice gap identified in this study concerned the depth and clinical interpretation of tobacco-use information rather than the absence of a smoking-specific technical treatment modification. Although 55.2% of participants reported asking whether patients smoked, only 28.4% assessed smoking frequency, 29.4% adapted case explanation according to smoking status, and 48.3% associated smoking with case prognosis. The qualitative findings further showed substantial variation in smoking-history assessment, ranging from detailed investigation of frequency, duration, quantity, previous exposure, and cessation to simple yes/no recording without endodontic interpretation. Although only 18.4% reported modifying some aspect of treatment, Q8 did not define what such modification would entail, and current evidence does not establish a specific technical endodontic modification that should routinely be applied solely on the basis of smoking status. Accordingly, the Q8 finding should be interpreted descriptively rather than as evidence of failure to translate an established treatment recommendation into practice.

None of the associations between professional category and clinical behaviours remained statistically significant after either Bonferroni or Benjamini–Hochberg correction (Table 3). Cramer's V values ranged from 0.126 to 0.214 and were small. Therefore, the present data do not support inferential claims that smoking-related behaviours differed according to professional setting. The qualitative accounts of time pressure, service constraints, and workload should instead be interpreted as participant-reported contextual barriers rather than explanations for statistically demonstrated differences between professional groups (Supplementary Tables S1–S6).

This study has several limitations. Its cross-sectional design and invitation-based sampling restrict causal inference and may limit generalisability. The quantitative data were self-reported and may therefore be affected by recall or social-desirability bias. Demographic variables such as age, sex, years of experience, specialist training, and workload were intentionally omitted to preserve anonymity within a relatively identifiable professional community, limiting assessment of individual-level influences. The questionnaire was developed specifically for this study through internal expert discussion and did not undergo formal pilot

testing, psychometric validation, or quantitative face/content-validity assessment, which should be considered when interpreting the quantitative findings. Moreover, Q8 asked broadly whether participants modified any aspect of treatment without defining the nature of such modification; consequently, responses cannot be interpreted as adherence to or deviation from a specific evidence-based smoking-related endodontic protocol. Q10 also framed the smoking–periapical healing relationship more definitively than warranted by the heterogeneous literature and referred to this relationship as a "fact". Although participants in the qualitative phase were not informed of the quantitative findings or response distributions before providing their qualitative responses, they had previously been exposed to the wording of Q10. This exposure may have sensitised them to the hypothesised relationship and cannot be excluded as a potential influence on subsequent responses concerning mechanisms, prognosis, or clinical relevance. The qualitative participants were also self-selected from the quantitative sample by accepting the follow-up invitation, and open-ended responses varied in depth. Accordingly, the qualitative findings should be interpreted as providing explanatory and interpretative context rather than independent confirmation of the quantitative findings. Finally, the study did not assess whether smoking-specific modifications to endodontic treatment improve clinical outcomes. Longitudinal and prospective studies are needed to clarify both prognostic associations and the clinical utility of structured smoking-related assessment.

Overall, the combined quantitative and qualitative findings suggest that the principal evidence-practice gap lies not simply in whether smoking is recorded during anamnesis, but in the depth with which tobacco exposure is assessed and interpreted in relation to prognosis and patient communication. Smoking was often documented as a general health variable, whereas only a subset of participants described integrating this information into endodontic-specific reasoning. Importantly, these findings should not be interpreted as evidence that smokers require a specific technical modification of root canal treatment. Rather, they identify an area in which documentation, risk recognition, prognostic discussion, and patient communication remain inconsistent and warrant further investigation.

Conclusion

This study identified inconsistencies between the documentation of tobacco use and the depth with which this information is assessed and interpreted in endodontic care. The principal evidence-practice gap concerned detailed smoking-history assessment, prognostic reasoning, and patient communication rather than the absence of a specific smoking-related technical modification to treatment. Participant accounts also identified knowledge limitations and practical contextual barriers to more detailed assessment. The findings support more consistent documentation of tobacco-use history and risk communication, while the prognostic and management implications of smoking in Endodontics require further prospective investigation. Future studies should evaluate whether structured tobacco-use assessment or smoking-informed prognostic approaches improve clinical decision-making or patient outcomes.

Authors' contributions (credit)

Luiz Carlos Trevia Morais Correia Viana: Conceptualisation; Data curation; Formal analysis; Investigation; Methodology; Validation; Writing – original draft. Christina César Praça–Brasil: Conceptualisation; Data curation; Methodology; Project administration; Supervision; Writing – review & editing. José Evando da Silva-Filho: Conceptualisa-

tion; Data curation; Formal analysis; Investigation; Methodology; Writing – review & editing. Juliana Carneiro Melo – Data curation; Formal analysis; Investigation; Methodology; Writing – review & editing. Denise Nunes Oliveira – Data curation; Formal analysis; Investigation; Methodology; Writing – review & editing. Bernardo Almeida Aguiar: Data curation; Formal analysis; Investigation; Methodology; Writing – review & editing. Fábio Almeida Gomes: Data curation; Formal analysis; Investigation; Methodology; Writing – review & editing. Eduardo Diogo Gurgel-Filho: Data curation; Formal analysis; Investigation; Methodology; Writing – review & editing.

Ethical considerations
The study was approved by the Research Ethics Committee of the University of Fortaleza (no. 6,482,408), in accordance with Resolution 466/2012 of the Brazilian National Health Council and the ethical standards laid down in the 1964 Declaration of Helsinki. All participants provided informed consent electronically before accessing the questionnaire.

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Generative AI and AI-assisted technologies in the writing process
The authors used ChatGPT (https://chatgpt.com/) to assist with the language refinement and readability of this manuscript. The tool was employed exclusively to enhance the language and readability of the text. Following its use, the authors thoroughly reviewed and edited the output as necessary, assuming full responsibility for the final content of the publication.

Declarations

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Competing interest
The authors deny any conflicts of interest.

Data availability
All data generated are presented within the manuscript. Supplementary materials and data are available by request to the authors.

References

1. López-López J, Jané-Salas E, Martín-González J, et al. Tobacco smoking and radiographic periapical status: a retrospective case-control study. J Endod. 2012;38(5):584-588. doi:10.1016/j.joen.2012.02.011
2. Leite FRM, Nascimento GG, Scheutz F, López R. Effect of smoking on periodontitis: a systematic review and meta-regression. Am J Prev Med. 2018;54(6):831-841. doi:10.1016/j.amepre.2018.02.014
3. Aminoshariae A, Kulild J, Gutmann J. The association between smoking and periapical periodontitis: a systematic review. Clin Oral Investig. 2020;24(2):533-545. doi:10.1007/s00784-019-03094-6
4. Pinto KP, Ferreira CM, Maia LC, Sassone LM, Fidalgo TKS, Silva EJNL. Does tobacco smoking predispose to apical periodontitis and endodontic treatment need? A systematic review and meta-analysis. Int Endod J. 2020;53(8):1068-1083. doi:10.1111/iej.13316
5. Cabanillas-Balsera D, Segura-Egea JJ, Bermudo-Fuenmayor M, et al. Smoking and radiolucent periapical lesions in root filled teeth: systematic review and meta-analysis. J Clin Med. 2020;9(11):3506. Published 2020 Oct 29. doi:10.3390/jcm9113506
6. Ríos-Osorio N, Muñoz-Alvear HD, Jiménez-Castellanos FA, et al. Association between cigarette smoking and the prevalence of post-endodontic periapical pathology: a systematic review and meta-analysis. Restor Dent Endod. 2022;47(3):e27. Published 2022 Jun 13. doi:10.5395/rde.2022.47.e27
7. Viana LCTMC, Silva-Filho JE, Feitosa APO, Castelo Branco TC, Teixeira RC, Praça-Brasil CC. Impact of smoking on the anatomical distribution of periapical bone rarefactions: a Brazilian retrospective cross-sectional study. J Adv Med Med Res. 2025;37(11):104-112. doi:10.9734/

jammr/2025/v37i115970
8. dos Santos VC, Kublitski PMO, da Silva BM, Gabardo MCL, Tomazinho FSF. Periapical lesions associated with demographic variables, dental conditions, systemic diseases, and habits. J Contemp Dent Pract. 2023;24(11):864-870. Published 2023 Nov 1. doi:10.5005/jp-journals-10024-3596
9. Viana LCTMC, da Silva-Filho JE, Barreto LP, Almeida-Gomes F, Praça-Brasil CC. Oral health within tobacco control policies: global and Brazilian perspectives. Arch Curr Res Int. 2025;25(8):690-701. doi:10.9734/acri/2025/v25i81451
10. Viana LCTMC, da Silva-Filho JE, Barreto LP, Aguiar BA, Almeida-Gomes F, Praça-Brasil CC. Tobacco use and its effects on the pulp and periapical tissues: an integrative review and thematic analysis. Arch Curr Res Int. 2025;25(8):878-889. doi:10.9734/acri/2025/v25i81467
11. Braun V, Clarke V. Using thematic analysis in psychology. Qualitative research in psychology. 2006;3(2):77-101. doi: 10.1191/1478088706qp063oa
12. Bergström J, Babcan J, Eliasson S. Tobacco smoking and dental periapical condition. Eur J Oral Sci. 2004 Apr;112(2):115-20. doi: 10.1111/j.1600-0722.2004.00112.x.
13. Paljevic E, Brekalo Prso I, Hrstic JV, Pezelj-Ribaric S, Persic Bukmir R. Impact of Smoking on the Healing of Apical Periodontitis after Nonsurgical Endodontic Treatment. Eur J Dent. 2024;18(1):124-130. doi: 10.1055/s-0043-1761451.
14. Pirani C, Iacono F, Prati C, Arias A. Assessment of factors influencing post-operative pain: the impact of smoking habit in root canal treatments obturated with carrier-based technique and epoxy resin sealer: Smoking habit and post-endodontic pain. G. ital. Endod. 2024;38(3): 1-9. doi: 10.32067/GIE.2024.38.01.14.
15. Bonilla J, Rodríguez-Reyes, D, Serpa-Irizarry M, Díaz-Cortés H, Barreras F, & Rivera-Barrios A. The Impact of Nicotine on Wound Healing: A Comparative Review of Cigarettes, Vaping, and Nicotine Patches with Insights into Pathophysiological Mechanisms. Medical Research Archives, 2025;13(6): 1-13. doi:10.18103/mra.v13i6.6635.
16. Javed F, Al-Rasheed A, Almas K, Romanos GE, Al-Hezaimi K. Effect of cigarette smoking on the clinical outcomes of periodontal surgical procedures. Am J Med Sci. 2012;343(1):78-84. doi: 10.1097/MAJ.0b013e318228283b.