Impact of Smoking on the Healing of Apical Periodontitis after Nonsurgical Endodontic Treatment

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Impact of Smoking on the Healing of Apical Periodontitis after Nonsurgical Endodontic Treatment

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Abstract

Objectives The aim of this prospective study was to compare the healing of periapical bone between smokers and nonsmokers after root canal therapy. The effects of duration and intensity of smoking on the healing rate of apical periodontitis were analyzed.

Materials and Methods Fifty-five smokers were included in this study. The control group consisted of healthy nonsmokers who matched the smoker group in age and sex. Only teeth with a favorable periodontal prognosis and adequate coronal restoration were included in the study. The periapical status of treated teeth was assessed using the periapical index system at follow-ups after 6 and 12 months.

Statistical Analysis The chi-squared test and Mann–Whitney U test were used to assess the changes in periapical index score at baseline and in subsequent time intervals between the two groups examining dichotomized and ordinal data, respectively. Multivariate logistic regression analysis was used to test the association of independent variables age, gender, tooth type, arch type, and smoking index with the outcome variable. The outcome variable was set as the presence versus absence of apical periodontitis.

Results The analysis at 12-month follow-up revealed a significantly higher healing rate in control group than in smokers (90.9 vs. 58.2; $\chi 2 = 13.846$; $p < 0.001$). Smokers had significantly higher periapical index scores than the control group ($p = 0.024$). The multivariate logistic regression analysis demonstrated that an increase in the value of the smoking index significantly increases the risk of apical periodontitis persistence (odds ratio [OR] =7.66; 95% confidence interval [CI]: 2.51–23.28; $p < 0.001$) for smoking index < 400 and (OR = 9.65; 95% CI: 1.45–64.14; $p = 0.019$) for smoking index 400 to 799.

Conclusion The results from this study show a lower rate of apical periodontitis healing in a group of smokers at 1-year follow-up. Delayed periapical healing seems to be associated with the cigarette smoking exposure.

Keywords
- cigarette smoking
- periapical periodontitis
- root canal therapy
- treatment outcome

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Introduction

Apical periodontitis (AP) is an acute or chronic inflammation of the apical periodontium caused by bacterial infection of the root canal system.[1] Diagnosis is based primarily on radiographic findings of periradicular radiolucency, sometimes accompanied by clinical signs.[2] AP often presents as a chronic, asymptomatic condition leading to underestimation of its prevalence and burden.

Healing of periapical bone lesions is a lengthy process, monitored clinically and radiographically, and influenced by local and systemic predisposing factors.[3] Tobacco smoking is recognized as a global public health problem that negatively affects both systemic and oral health.[4,5] Smoking habit has been suggested as a modulating factor that could negatively affect the healing of periapical bone lesions through multiple mechanisms.[6] It affects the microvasculature by decreasing nutrient and oxygen levels,[7] limiting pulp defense mechanisms, and contributing to its necrosis.[8] Smoking impedes tissue repair leading to fibroblast dysfunction and impaired collagen synthesis.[9] It can alter the immune response to infections by suppressing immune cell functions and causing a stronger systemic inflammatory response.[10]

According to the 2015 study by the Croatian Institute for Public Health, cigarette smoking is a widespread habit in Croatia. The questionnaire revealed that 31.1% of the Croatian population consumes cigarettes (35.3% smokers among men and 27.1% among women), 27.5% of them daily.[11] Similarly, smoking is a common habit globally with estimates of 32.6% adult male smokers and 6.5% female smokers in 2020.[12]

Previous studies investigating the effects of smoking on endodontic variables were cross-sectional studies with contradictory conclusions.[13–15] These studies did not consider the presence of confounding variables such as periodontal disease affecting the tooth, diabetes mellitus that could affect the healing process or the socioeconomic status (SES) of the patient. Healing of AP can take up to 4 years, which prevents the evaluation of treatment outcome through a cross-sectional study design, as it is unclear whether the lesion is persistent or in a healing phase.[16]

The aim of this cohort study was to compare the healing of periapical bone after nonsurgical endodontic treatment between smokers and nonsmokers and to assess the influence of smoking intensity and duration on the healing rate of AP at 1 year follow-up. We hypothesized that smoking habit is associated with prolonged or absent healing of AP.

Materials and Methods

This prospective study was conducted at the Department of Endodontics and Restorative dentistry University Dental Clinic, Rijeka Clinical Hospital Centre, Rijeka, Croatia. Adult patients who agreed to participate by signing an informed consent form were enrolled in the study. The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Rijeka Clinical Hospital Centre (003-05/20-1/131). This study is registered in ClinicalTrials.gov registry with an associated identifier number NCT04812171.

Participants

►**Fig. 1** shows the flowchart of participants through stages of recruitment and treatment. Only participants with a radiologically confirmed diagnosis of AP were included in the study, and strict inclusion and exclusion criteria were applied. Each participant provided one tooth into the research that had not been previously treated. Data on the health status of each participant were collected using an FDI Health Questionnaire, and data on smoking habits and SES were collected using a self-administered structured questionnaire. Participants were classified as smokers if they answered in the affirmative to the questions: "Have you consumed at least 100 cigarettes in your lifetime," and "Do you currently smoke?" The smoking index is a unit for measuring cigarette consumption over a long period and was calculated using data on duration of smoking habit in years and cigarette consumption per day (CPD).[17] It quantifies smoking exposure and consists of the following categories: nonsmoker, less than 400, 400 to 799, and 800 and over.[18] Smoking intensity was assessed using data on CPD, classifying heavy smoking as 20 or more CPD and mild smoking as less than 20 CPD.[19] A cutoff was set at 20 cigarettes because differences in CO_2, cotinine, and nicotine levels were observed between a group of smokers who smoked 20 or less CPD and a group who consumed more CPD.[20] Participants diagnosed with a systemic disease or taking medications known to alter immunologic response or bone metabolism were excluded from the study. Also, former and occasional smokers, pregnant patients, and individuals who refused to participate were excluded from the study. The control group consisted of healthy nonsmokers who matched the smoker group in age and gender. Even though age and gender were not identified as confounding factors, previous studies observed that the prevalence of AP increases with age and that men has a higher percentage of teeth with AP.[14,21] Regarding the SES, participants provided information on their education level, monthly household income, self-assessed SES, and urbanization level.

To minimize the role of confounding factors, only teeth with a favorable periodontal prognosis ($<$ 5 mm attachment loss and marginal bone loss of $<$ one-third of the root length) were included in the study.[22,23]

Methods

AP was diagnosed based on clinical and radiographic examination. Endodontic specialists performed root canal treatments according to the standardized endodontic treatment protocol, which includes administration of local anesthesia and isolation with rubber dam. The teeth included in the study had well-performed root canal treatment with homogeneously obturated canals up to 1 mm from the radiographic apex and direct or indirect coronal restoration with clinically and radiographically well-sealed margins.

Analysis of marginal bone loss was performed using periapical radiographs at baseline and both follow-ups.

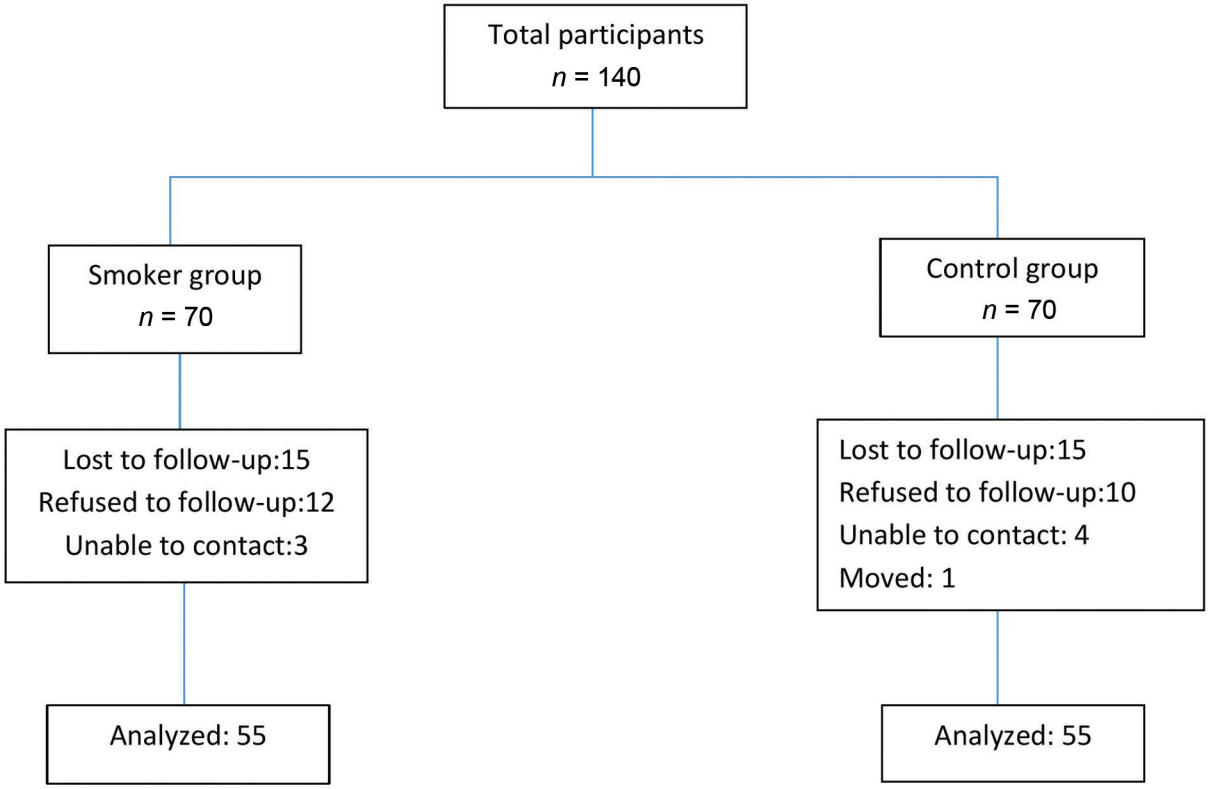

Fig. 1 The flowchart for patient recruitment and treatment.

Marginal bone loss was classified as more or less than one third of the root length.[23]

Follow-up examinations were arranged 6 and 12 months after root canal treatment. Teeth were evaluated clinically by percussion and palpation tests, periodontal probing and examination of the coronal restoration. Immediately after treatment and at each follow-up visit, a standardized periapical radiograph of each tooth was taken using a sensor holder and the Planmeca ProX intraoral X-ray unit and Planmeca Romexis software (Planmeca Oy, Asentajankatu 6, Helsinki, Finland).

The periapical status of treated teeth was assessed using the periapical index system (PAI).[24] A PAI score was determined by one calibrated examiner using visual references for each of the five categories on an ordinal scale. The highest PAI value of all roots was used to determine the periapical status in multirooted teeth. Kappa values for inter-and intra-examiner agreement were 0.75 and 0.81 respectively. The PAI scores were dichotomized, recording AP as absent (PAI scores 1 and 2) and present (PAI scores 3, 4, and 5). Clinically, AP was assessed as healed in asymptomatic teeth, not sensitive to palpation or percussion.

Sample Size Determination

Using Medcalc software size determination was used based on input of previously reported prevalence of AP in smokers and nonsmokers.[25] The computation was performed at p-value less than 0.05 and power 0.10 and resulted with a minimum sample size of 51 participants in each group. Keeping in mind the patient dropout rate, 70 patients per group were recruited.

Statistical analysis was carried out using IBM SPSS 26 (IBM Corp, Armonk, New York, United States) and MedCalc statistical software (MedCalc Software Ltd., Ostend, Belgium) with level of statistical significance set at p-value less than 0.05. The Kolmogorov–Smirnov test was used to test data for normal distribution. Because the data were not normally distributed, nonparametric tests were applied.

The chi-squared test and Mann–Whitney U test were used to assess the changes in PAI score at baseline and in subsequent time intervals between the two groups examining dichotomized and ordinal data, respectively. Multivariate logistic regression analysis (enter model) was used to test the association of independent variables age, gender, tooth type, arch type, and smoking index with outcome variable.

Results

A hundred and ten patients were included in the study, 78 women and 32 men (70.9 vs. 29.1%) ranging in age from 18 to 66 years (median: 35.0; interquartile range: 29–46). The basic group characteristics are presented in ►**Table 1**. There were no significant differences between the two groups regarding age, gender, tooth type, SES or PAI scores at the baseline.

On average, smokers consumed 12.22 cigarettes per day (median 12.0; interquartile range: 5–20) and most of them (72.7%) were categorized as "mild smokers." Duration of a smoking habit ranged from 1 to 40 years (median: 15.0; interquartile range: 8–22).

Men consumed significantly more cigarettes per day in comparison to women ($p < 0.001$). Significantly more men

Table 1 Basic group characteristics

Variables	Smokers ($n = 55$)	Control ($n = 55$)	Statistical test	Statistics
Age (median, interquartile range)	34 (28.25-45.5)	35 (29.0-45.75)	Mann–Whitney U test	0.900
Gender				
Male	16	16	Chi-squared test	$\chi2 = 0.044$ $p = 0.834$
Female	39	39		
Tooth type				
Single rooted	8	8	Chi-squared test	$\chi2 = 2.286$ $p = 0.319$
Premolars	13	7		
Molars	34	40		
Arch type				
Maxilla	22	18	Chi-squared test	$\chi2 = 0.354$ $p = 0.552$
Mandible	33	37		
Level of education				
Elementary school High school University	2 37 16	0 35 19	Chi-squared test	$\chi2 = 2.304$ $p = 0.316$
Average monthly household income expressed in Kunas				
1,000–4,000 4,000–6,000 6,000–10,000 Above 10,000	6 14 14 19	5 12 20 16	Chi-squared test	$\chi2 = 1.561$ $p = 0.668$
Self-assessed SES				
Below average Average Above average	6 26 22	3 33 18	Chi-squared test	$\chi2 = 2.231$ $p = 0.328$
Urbanization level				
Urban area Suburban area Rural area	30 17 8	29 17 8	Chi-squared test	$\chi2 = 0.08$ $p = 0.996$
PAI scores baseline (mean rank)	53.19	57.81	Mann–Whitney U test	$p = 0.415$

Abbreviations: PAI, periapical index; SES, socioeconomic status.

were categorized as "heavy smokers" in comparison to women (50 vs. 17.9%, $p = 0.015$) consuming 20 or more cigarettes per day. However, no significant difference in healing outcome was found with regard of the gender of the smokers ($p = 0.43$).

The chi-squared test was used to analyze the difference in healing rate in smokers and control group at 6-month and 12-month follow-up (►**Table 2**). There was no significant difference in healing rate at 6-month follow-up. Conversely, analysis at 12-month follow-up revealed significantly higher healing rate in control group than in smokers (90.9 vs. 58.2; $\chi2 = 13.846$; $p < 0.001$). Analysis according to full-scale PAI also revealed difference only at this point. Smokers had significantly higher PAI scores than control group ($p = 0.024$; ►**Table 3**).

Multivariate logistic regression analysis was used to test the association age, gender, tooth type, arch type, and smoking index with outcome variable. The dichotomous

Table 2 Treatment outcome in smoker and control group at the 6- and 12-month follow-up

Group	Healed (6-month follow-up) *n* (%)	Not-healed (6-month follow-up) *n* (%)	Chi-squared test	Healed (12-month follow-up) *n* (%)	Not-healed (12-month follow-up) *n* (%)	Chi-squared test
Smokers	20 (36.4)	35 (63.6%)	$\chi2 = 0.341$ $p = 0.559$	32 (58.2%)	23 (41.8%)	$\chi2 = 13.846$ $p < 0.001$
Control	24 (43.6)	31 (56.4%)		50 (90.9%)	5 (9.1%)	
Total	44 (40.0)	66 (60.0%)		82 (74.5%)	28 (25.5%)	

Table 3 Difference between smoker and control group in PAI at the 6- and 12-month follow-up

Group	PAI (6-month follow-up)	Mann–Whitney U test	PAI (12-month follow-up)	Mann–Whitney U test
Smokers	3 (2–3)	$p = 0.295$	2 (1–3)	$p = 0.024$
Nonsmokers	3 (1–3)		1 (1–2)	

Abbreviation: PAI, periapical index.

Table 4 Multivariate logistic regression analysis of independent variables on the AP healing outcome

Variable	Total	AP healed, *n* (%)	AP not healed, *n* (%)	OR	95% CI	*p*-Value
Age	Continuous variable			0.98	0.94–1.03	0.389
Gender						
Male	32	23 (71.9)	9 (28.1)	1	Reference	
Female	78	59 (75.6)	19 (24.4)	0.84	0.30–2.39	0.748
Tooth type						
Anterior	16	13 (81.2)	3 (18.8)	1	Reference	
Premolar	20	15 (75.0)	5 (25.0)	1.04	0.18–6.07	0.964
Molar	74	54 (73.0)	20 (27.0)	1.66	0.35–7.86	0.525
Arch type						
Maxilla	40	30 (75.0)	10 (25.0)	1	Reference	
Mandible	70	52 (74.3)	18 (25.7)	1.09	0.39–3.02	0.873
Smoking index						
Nonsmoker	55	50 (90.9)	5 (9.1)	1	Reference	
< 400	47	27 (57.4)	20 (42.6)	7.66	2.51–23.28	<0.001
400–799	8	5 (62.5)	3 (37.5)	9.65	1.45–64.14	0.019

Abbreviations: AP, apical periodontitis; CI, confidence interval; OR, odds ratio.

outcome variable was set as the AP healing versus AP non-healing at 12-month follow-up (►**Table 4**). The only variable significantly associated with the outcome variable was the smoking index. The regression analysis demonstrated that the risk of AP persistence significantly increases with increase in the value of the smoking index (odds ratio [OR] =7.66; 95% confidence interval [CI]: 2.51–23.28; $p < 0.001$) for smoking index less than 400 and (OR = 9.65; 95% CI: 1.45–64.14; $p = 0.019$) for smoking index 400 to 799.

Discussion

To our knowledge, there are no previous prospective studies on the relationship between smoking and AP healing that would allow comparison with the results of the present study. However, the results are consistent with the findings of several cross-sectional studies that found a higher prevalence of AP in smokers.[14,25–27] The present study showed a significant difference in healing rate between smokers and nonsmokers (90.9 and 58.2%, respectively). A negative effect of smoking was also observed in a study investigating the relationships between smoking habits and periodontitis healing after mechanical periodontal therapy.[28] A lower AP healing rate in smokers could be attributed to the deleterious effect of cigarette consumption on the microvasculature, decreased pulp and periradicular tissue defense, and impaired tissue repair.[7–9]

Several previous studies have examined the association between smoking intensity and tooth loss. A study conducted among middle-aged Finnish adults found an exposure-related association between smoking intensity and tooth loss.[29] This study was based on a cohort project and measured smoking exposure in pack-years, calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked, without specifying the cause of tooth loss. When the reasons for tooth loss were considered, intensity and duration of smoking habits were significantly associated with tooth loss due to periodontal disease.[30] The number of cigarettes consumed and duration of smoking were positively associated with tooth loss in a study conducted in Denmark.[31] Current smokers who consumed more than 15 cigarettes per day for more than 27 years had increased scores of missing teeth and associated OR compared with never smokers. In the present study, the smoking index was calculated using data on the number of cigarettes consumed per day and years of tobacco use. Higher values of the smoking index were associated with a 9.65-fold increase in the risk for the presence of AP compared to nonsmokers (95% CI: 1.45–64.14; $p = 0.019$). Since the persistence of AP ultimately leads

to tooth loss, the results of this study are consistent with several previous studies that have identified smoking intensity and duration as a risk factor for tooth loss.[31–33]

The overall healing rate at 6-month follow-up was 40%, while after 1 year almost 75% of the teeth examined were free of radiographic and clinical signs consistent with AP. A limitation of this study is the relatively short follow-up period of 12 month, considering that the European Society of Endodontology recommends that the lesion be assessed over a 4-year period.[16] A study by Huumonen and Ørstavik reported statistically significant healing up to 2 years after nonsurgical endodontic treatment, and although recall rates were low at 3 and 4 years, the trend of healing was confirmed.[34]

Socioeconomic factors are associated with systemic and oral health and assert their influence through health-related variables. Individuals with lower SES reported a higher risk of tooth loss.[35] To avoid the confounding effect of socioeconomic variables, both groups were tested, and no significant difference was found with respect to SES.

Because the healing outcome was not influenced by age or gender, the results of this study are consistent with other studies observing variables that influence the outcome of nonsurgical endodontic treatment.[36,37]

Chronic endodontic and periodontal inflammation share several common features and to exclude the influence of marginal periodontitis on the healing of AP, only teeth with a favorable periodontal prognosis were included in the study. Previous studies have examined the relationship between apical and marginal periodontitis and the effect of smoking on marginal bone levels. A significant difference in marginal bone level between smokers and nonsmokers was observed, with smokers having a more reduced marginal bone level.[38,39]

In the present study, more female participants were found to seek endodontic treatment at secondary dental care. This could be a confounding factor since the representation of men (29.1%) and women (70.9%) was not even. This difference could be due to the health awareness of female participants who are more likely to seek dental care and attend check-ups.[40]

Conclusions

To the best of the authors' knowledge, this is the first prospective study to investigate the association between smoking habit and the healing of AP with strict inclusion and exclusion criteria. The results of this study show a significant association between smoking habit and prolonged healing of AP. Moreover, the odds of AP persistence increased with an increase in smoking exposure. The results of this study suggest that cigarette smoking may be a modulating factor that delays or inhibits periapical healing and influences clinical decisions and guidelines concerning smokers.

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Conflict of Interest
None declared.

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References

1 Graunaite I, Lodiene G, Maciulskiene V. Pathogenesis of apical periodontitis: a literature review. J Oral Maxillofac Res 2012;2 (04):e1. Doi: 10.5037/jomr.2011.2401
2 Tibúrcio-Machado CS, Michelon C, Zanatta FB, Gomes MS, Marin JA, Bier CA. The global prevalence of apical periodontitis: a systematic review and meta-analysis. Int Endod J 2021;54(05): 712–735
3 Marotta PS, Fontes TV, Armada L, Lima KC, Rôças IN, Siqueira JF Jr. Type 2 diabetes mellitus and the prevalence of apical periodontitis and endodontic treatment in an adult Brazilian population. J Endod 2012;38(03):297–300
4 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(06):831–841
5 de Araújo Nobre MA, Sezinando AM, Fernandes IC, Araújo AC. Influence of smoking habits on the prevalence of dental caries: a register-based cohort study. Eur J Dent 2021;15(04):714–719
6 Segura-Egea JJ, Martín-González J, Castellanos-Cosano L. Endodontic medicine: connections between apical periodontitis and systemic diseases. Int Endod J 2015;48(10):933–951
7 Lehr HA. Microcirculatory dysfunction induced by cigarette smoking. Microcirculation 2000;7(6 Pt 1):367–384
8 Krall EA, Abreu Sosa C, Garcia C, Nunn ME, Caplan DJ, Garcia RI. Cigarette smoking increases the risk of root canal treatment. J Dent Res 2006;85(04):313–317
9 Wong LS, Green HM, Feugate JE, Yadav M, Nothnagel EA, Martins-Green M. Effects of "second-hand" smoke on structure and function of fibroblasts, cells that are critical for tissue repair and remodeling. BMC Cell Biol 2004;5:13. Doi: 10.1186/1471-2121-5-13
10 Johannsen A, Susin C, Gustafsson A. Smoking and inflammation: evidence for a synergistic role in chronic disease. Periodontol 2000 2014;64(01):111–126
11 Dečković Vukres V, Ivičević UA, Mihel S. Survey on the Use of Tobacco in the Adult Population of the Republic of Croatia. Zagreb: Croatian Institute of Public Health; 2016
12 Dai X, Gakidou E, Lopez AD. Evolution of the global smoking epidemic over the past half century: strengthening the evidence base for policy action. Tob Control 2022;31(02):129–137
13 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(05):584–588
14 Correia-Sousa J, Madureira AR, Fontes Carvalho M, Moura Teles A, Pina-Vaz I. Apical periodontitis and related risk factors: cross sectional study. Rev Port Estomatol Med Dent Cir Maxilofac 2015;56(04):226–232
15 Kirkevang LL, Vaeth M, Hörsted-Bindslev P, Bahrami G, Wenzel A. Risk factors for developing apical periodontitis in a general population. Int Endod J 2007;40(04):290–299
16 European Society of Endodontology. Quality guidelines for endodontic treatment: consensus report of the European Society of Endodontology. Int Endod J 2006;39(12):921–930
17 Jindal SK, Malik SK, Dhand R, Gujral JS, Malik AK, Datta BN. Bronchogenic carcinoma in Northern India. Thorax 1982;37 (05):343–347
18 Nagata N, Niikura R, Shimbo T, et al. Alcohol and smoking affect risk of uncomplicated colonic diverticulosis in Japan. PLoS One 2013;8(12):e81137. Doi: 10.1371/journal.pone.0081137

19 Khaled SM, Bulloch AG, Williams JV, Lavorato DH, Patten SB. Major depression is a risk factor for shorter time to first cigarette irrespective of the number of cigarettes smoked per day: evidence from a National Population Health Survey. Nicotine Tob Res 2011; 13(11):1059–1067
20 Heatherton TF, Kozlowski LT, Frecker RC, Fagerström KO. The Fagerström Test for Nicotine Dependence: a revision of the Fagerström Tolerance Questionnaire. Br J Addict 1991;86(09): 1119–1127
21 Figdor D. Apical periodontitis: a very prevalent problem. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94(06): 651–652
22 Alghofaily M, Tordik P, Romberg E, Martinho F, Fouad AF. Healing of apical periodontitis after nonsurgical root canal treatment: the role of statin intake. J Endod 2018;44(09):1355–1360
23 Dawson V, Petersson K, Wolf E, Akerman S. Periapical status of non-root-filled teeth with resin composite, amalgam, or full crown restorations: a cross-sectional study of a Swedish adult population. J Endod 2014;40(09):1303–1308
24 Orstavik D, Kerekes K, Eriksen HM. The periapical index: a scoring system for radiographic assessment of apical periodontitis. Endod Dent Traumatol 1986;2(01):20–34
25 Segura-Egea JJ, Jiménez-Pinzón A, Ríos-Santos JV, Velasco-Ortega E, Cisneros-Cabello R, Poyato-Ferrera MM. High prevalence of apical periodontitis amongst smokers in a sample of Spanish adults. Int Endod J 2008;41(04):310–316
26 Segura-Egea JJ, Castellanos-Cosano L, Velasco-Ortega E, et al. Relationship between smoking and endodontic variables in hypertensive patients. J Endod 2011;37(06):764–767
27 Al-Nazhan SA, Alsaeed SA, Al-Attas HA, Dohaithem AJ, Al-Serhan MS, Al-Maflehi NS. Prevalence of apical periodontitis and quality of root canal treatment in an adult Saudi population. Saudi Med J 2017;38(04):413–421
28 Grossi SG, Zambon J, Machtei EE, et al. Effects of smoking and smoking cessation on healing after mechanical periodontal therapy. J Am Dent Assoc 1997;128(05):599–607
29 Similä T, Virtanen JI. Association between smoking intensity and duration and tooth loss among Finnish middle-aged adults: The Northern Finland Birth Cohort 1966 Project. BMC Public Health 2015;15:1141. Doi: 10.1186/s12889-015-2450-6
30 Mai X, Wactawski-Wende J, Hovey KM, et al. Associations between smoking and tooth loss according to the reason for tooth loss: the Buffalo OsteoPerio Study. J Am Dent Assoc 2013;144(03): 252–265
31 Morse DE, Avlund K, Christensen LB, et al. Smoking and drinking as risk indicators for tooth loss in middle-aged Danes. J Aging Health 2014;26(01):54–71
32 Dietrich T, Walter C, Oluwagbemigun K, et al. Smoking, smoking cessation, and risk of tooth loss: the EPIC-potsdam study. J Dent Res 2015;94(10):1369–1375
33 Gabiec K, Bagińska J, Łaguna W, et al. Factors associated with tooth loss in general population of Bialystok, Poland. Int J Environ Res Public Health 2022;19(04):2369. Doi: 10.3390/ijerph19042369
34 Huumonen S, Ørstavik D. Radiographic follow-up of periapical status after endodontic treatment of teeth with and without apical periodontitis. Clin Oral Investig 2013;17(09):2099–2104
35 Gilbert GH, Duncan RP, Shelton BJ. Social determinants of tooth loss. Health Serv Res 2003;38(6 Pt 2):1843–1862
36 Ng YL, Mann V, Rahbaran S, Lewsey J, Gulabivala K. Outcome of primary root canal treatment: systematic review of the literature – Part 2. Influence of clinical factors. Int Endod J 2008;41(01): 6–31
37 Marquis VL, Dao T, Farzaneh M, Abitbol S, Friedman S. Treatment outcome in endodontics: the Toronto Study. Phase III: initial treatment. J Endod 2006;32(04):299–306
38 Bahrami G, Vaeth M, Kirkevang LL, Wenzel A, Isidor F. The impact of smoking on marginal bone loss in a 10-year prospective longitudinal study. Community Dent Oral Epidemiol 2017;45 (01):59–65
39 Bahrami G, Vaeth M, Wenzel A, Isidor F. Marginal bone level in two Danish cross-sectional population samples in 1997-1998 and 2007-2008. Acta Odontol Scand 2018;76(05):357–363
40 Peršić Bukmir R, Jurčević Grgić M, Brumini G, Spalj S, Pezelj-Ribaric S, Brekalo Pršo I. Influence of tobacco smoking on dental periapical condition in a sample of Croatian adults. Wien Klin Wochenschr 2016;128(7-8):260–265