original article

Wien Klin Wochenschr 2016 · 128:260–265
DOI 10.1007/s00508-015-0910-8

Wiener klinische Wochenschrift
The Central European Journal of Medicine

Influence of tobacco smoking on dental periapical condition in a sample of Croatian adults

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Received: 18 March 2015 / Accepted: 19 November 2015 / Published online: 11 December 2015

Summary
Background Since current evidences support a negative influence of tobacco smoking on the periodontal bone, an increased prevalence or severity of periapical lesions would be expected among smokers. The aim of this study was to investigate the difference in the periapical status of endodontically treated and untreated teeth in current smokers and never-smokers.

Methods The cross-sectional study included 259 subjects, 108 current smokers and 151 never-smokers, presenting as new patients at the Dental Clinic of the Clinical Hospital Centre Rijeka, Croatia. The periapical index (PAI) was used to assess the periapical status of all teeth, excluding third molars.

Results and conclusions Current smokers had higher fraction of teeth with apical periodontitis (AP) than never-smokers (0.13 vs. 0.10; $P=0.025$), while fractions of endodontically treated teeth and endodontically treated teeth with AP did not differ significantly. When overall number of teeth was controlled for, smokers were 16.4 times more likely to have AP than a non-smokers (95% CI: 5.7–47.7; $P<0.001$) and if a person was male, he was 3.1 times more likely to have AP than if the person was female (95% CI: 1.1–8.9; $P=0.039$). The probability of AP increases with increase of age. Smokers will on average have two teeth with AP more than non-smokers, while controlling for gender, age and overall number of teeth.

Keywords Endodontics · Periapical periodontitis · Root canal therapy · Smoking · Tobacco

Abbreviations
AP Apical periodontitis
PAI Periapical index
DM Diabetes mellitus

Introduction

Cigarette smoking in Croatia is a widespread habit. The research conducted by "Ivo Pilar" Institute of Social Science on Croatian subjects established that 39.7 % of men and 32.9 % of women are current smokers. The largest proportion of smokers (16.5 %) consumes 10–20 cigarettes per day [1]. According to the research *The European School Survey Project on Alcohol and Other Drugs* (ESPAD) conducted in 2011, it was established that the prevalence of "smoking in the last 30 days" in Croatian 16-year-olds is 41 %, which is the third largest prevalence among the same age group in 37 European countries [2].

Apical periodontitis (AP) is an inflammatory disorder of periradicular tissues caused by persistent microbial infection of the root canal system of the affected tooth [3]. Since clinical signs and symptoms such as pain, tenderness, swelling and sinus tract formation occur to varying degrees and are only moderately specific [4], diagnosis of AP is based on radiological detection of deviation from

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normal periapical anatomy. A recently conducted cross-sectional study on Croatian subjects has reported the prevalence of AP in 8.5% of examined teeth. It has also found AP in 54% of endodontically treated teeth [5].

Several studies have demonstrated a relationship between smoking and higher incidence of dental caries. The decreased buffering effect and possible lower pH of smokers' saliva as well as the higher number of lactobacilli and *Streptococcus mutans* may favour increased susceptibility to caries in smokers [6–8]. Since AP is commonly an inflammatory sequel to dental caries, it is justified to expect that periapical lesions are more frequent in smokers.

Based on current evidence of harmful effects of tobacco smoking on the periodontal bone [9], it was assumed that smoking might have a negative influence on apical periodontium of endodontically compromised teeth, facilitating the extension of periapical bone destruction and/or interfering with healing after root canal treatment. Consequently, an increased prevalence or severity of periapical lesions would be expected in smokers [10]. Several studies investigated the association between tobacco smoking and AP with contradictory results [10–13].

Since, to our knowledge, the relation between current cigarette smoking and AP in Croatian subjects has not yet been investigated, the aim of the present study was to investigate the difference in the periapical status of endodontically treated and untreated teeth among currently smoking and never-smoked subjects.

Patients, materials and methods

The initial sample assessed for eligibility included 522 individuals older than 18 years presenting consecutively as new patients seeking routine dental care at the Dental Clinic of the Clinical Hospital Centre Rijeka, Croatia, during a 1-year period (2013). The study included only patients requiring digital panoramic and periapical radiographs of teeth with periapical pathosis and/or endodontically treated teeth as a part of routine diagnostics and dental treatment planning. Patients having less than eight remaining teeth and patients who had endodontic treatment in the preceding 2 years were excluded. Patients with diabetes mellitus (DM) and former smokers were also excluded from the sample (Fig. 1). The smoking status was self-reported and obtained by interview. After the exclusion criteria were applied the final sample consisted of 259 patients, aged 40.3 + 15.1, with 82 male subjects (31.7%) and 177 female subjects (68.3%). The Ethics Committee of the Rijeka Clinical Hospital Centre approved the study, and participants agreed to participate in the study by signing an informed consent.

Panoramic radiographs were taken by using a digital orthopantomograph machine (J. Morita Corporation, Veraviewepocs 6716, Kyoto, Japan). Images were obtained by compatible software (Mediadent V4, Image Level, Nieuwkerken-waas, Belgium) and laser printed

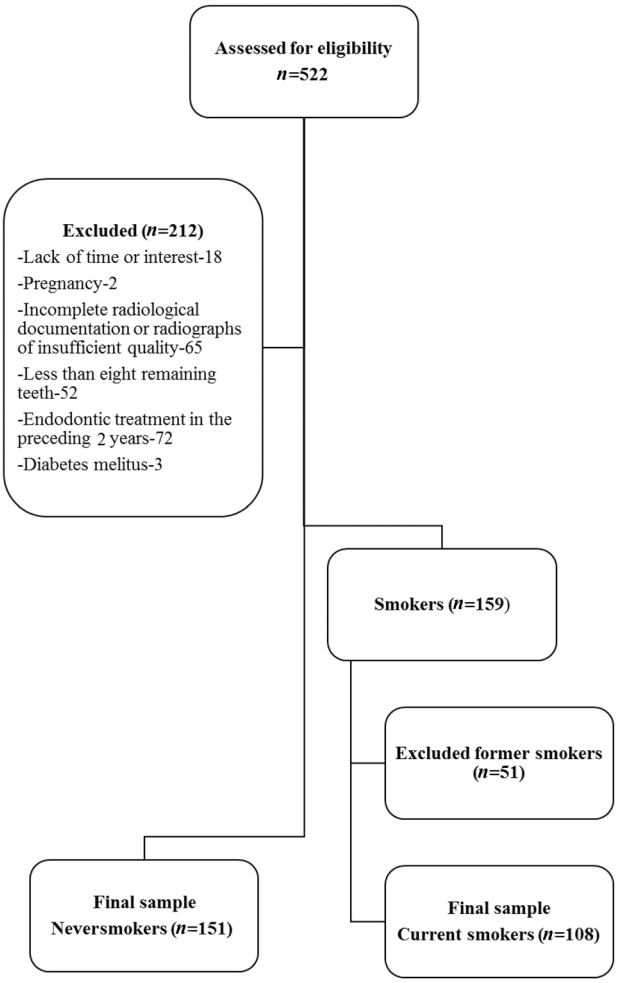

Fig. 1 Flowchart showing patient enrolment in the study

on film. Periapical radiographs of respective teeth were taken using the paralleling technique with a Trophy Elitys x-ray unit (Trophy Radiologie, Marne-la-Vallee, France) and E-Speed film (Carestream Health, Rochester, NY, USA). The films were processed automatically (Durr Dental, Bietigheim-Bissingen, Germany).

From panoramic and periapical radiographs all teeth, excluding third molars, were recorded. Teeth were categorised as endodontically treated if a radio-opaque material was visible in the pulp chamber and/or in the root canals. The method of viewing the radiographs was standardised—they were examined in a darkened room using the negatoscope with magnification (3.5×).

The periapical status of all teeth was assessed by using the periapical index system (PAI) [14]. A score greater than 2 (PAI > 2) was taken to indicate the presence of AP. For multi-rooted teeth, the root presenting the highest PAI score was used. The radiographs were examined by one observer. Before evaluation, the observer participated in a calibration course for the PAI system, which consisted of 100 radiographic images of teeth (provided by Dr Ørstavik). Each tooth had to be assigned to one of the five PAI scores by using visual references for five categories within the scale. After scoring the teeth, the

results were compared to a silver standard score for 100 reference teeth and the Cohen's kappa was calculated as 0.70. Intra-observer reproducibility was evaluated by the repeat scoring of 30 patients 2 months after the first examination. These patients were randomly selected. Before the second evaluation the observer was recalibrated according to PAI. Cohen's kappa after recalibration was 0.67. The intra-observer agreement test on PAI scores on 30 patients produced a Cohen's Kappa of 0.75. An observer with kappa values for inter- and intra-observer reproducibility of >0.61 is "authorised" to produce valid experimental scores [14, 15].

Statistical methods

Raw data were entered into Excel database, and statistical analyses were carried out using commercial softwares Statistica 12.0 (Statsoft Inc, Tulsa, OK, USA), MedCalc 14.8.1 (MedCalc Software bvba, Belgium) and IBM SPSS 22 (IBM Corp, Armonk, NY, USA). Testing data for normal distribution were performed by Kolmogorov–Smirnov test. Differences in age, average number of teeth, fraction of endodontically treated teeth, fraction of teeth with AP and fraction of endodontically treated teeth with AP between current smokers and never smokers were analysed by *t* test for independent samples. To analyse the differences in the prevalence of persons with AP, endodontic treatment and endodontic treatment associated with AP between current smokers and never smokers chi-square test was used.

A generalised linear model was used to explore the relationship between the presence of AP and smoking, gender, age and endodontic treatment. Two models (Model 1: presence of teeth with AP and Model 2: presence of endodontically treated teeth with AP) and two approaches (binomial distribution with logit link and negative binomial distribution with log lik) were used. Gender (1 = man; 2 = woman), smoking (0 = nonsmoker; 1 = current smoker), age (continuous predictor), fraction of endodontically treated teeth (*N* of endodontically treated teeth/*N* of present teeth; continuous variable) were used as predictors. Overall number of teeth served as offset variable in Model 1 and overall number of endodontically treated teeth in Model 2. Several outcome variables were studied: presence of AP (0 = subject without single tooth with AP; 1 = subject with at least one tooth with AP) in Model 1a, number of teeth with AP in Model 1b, presence of AP in endodontically treated teeth (0 = subject without single tooth with AP on endodontically treated teeth; 1 = subject with at least one tooth with AP on endodontically treated teeth) in Model 2a and the number of endodontically treated teeth with AP in Model 2b. Since the fraction of endodontically treated teeth is too highly correlated with AP in Model 1a and 1b to be meaningfully included in the analyses, it was omitted in those models. Akaike's information criterion (AIC) and Bayesian information criterion (BIC) were used to compare the model's fit and to select the more appropriate model.

Results

The study sample consisted of 108 current smokers (42 %) and 151 never smokers (58 %). The mean age was 38.8 ± 13.6 years for current smokers and 41.9 ± 16.2 years for never smokers ($P = 0.106$). The average number of teeth per patient was 22.9 ± 5.2 and 23.2 ± 4.9 in smoking and never-smoked patients, respectively ($P = 0.636$).

AP in one or more teeth was more often found in smokers (86.1 %) than in never smokers (78.1 %; $P = 0.143$). One or more endodontically treated teeth were found in 89 (82.4 %) and 119 (78.8 %) of smoking and never-smoked subjects, respectively ($P = 0.576$). Among smoking patients with endodontically treated teeth, 67 (72.0 %) had AP affecting at least one treated tooth. Among never smokers with endodontically treated teeth, 93 (78.8 %) had AP affecting at least one treated tooth ($P = 0.328$) (Table 1).

In univariate analysis smokers had a higher fraction of teeth with AP than never smokers (0.13 vs. 0.10; $P = 0.025$), whereas fractions of endodontically treated teeth and endodontically treated teeth with AP did not differ significantly (Table 2).

A current smoker was on average 16.4 times more likely to have AP than a never smoker (95 % CI: 5.7–47.7; $P < 0.001$), and if the person was a man, he was 3.1 times more likely to have AP than if the person was a woman (95 % CI: 1.1–8.9; $P = 0.039$; Table 3). The probability of AP increases with increase in age (Table 3). Age, gender, smoking and fraction of endodontically treated teeth do not predict probability of AP on endodontically treated teeth (Table 4).

Table 1 Prevalence of apical periodontitis (AP) and endodontic treatment in currently smoking and never smoked subjects

Variable	Subjects/*n* (%)		Statistics	Total
	Current smokers group *n* = 108	Never-smoked group *n* = 151		*n* = 259
Individuals with apical periodontitis				
Yes	93 (86.1)	118 (78.1)	$\chi^2 = 2.14$	211 (81.5)
No	15 (13.9)	33 (21.9)	$P = 0.143$	48 (18.5)
Individuals with endodontically treated teeth				
Yes	89 (82.4)	119 (78.8)	$\chi^2 = 0.31$	208 (80.3)
No	19 (17.6)	32 (21.2)	$P = 0.576$	51 (19.7)
AP in individuals with and without endodontic treatment[a]				
Treated	67 (72.0)	93 (78.8)	$\chi^2 = 0.96$	160 (75.8)
Untreated	26 (28.0)	25 (21.2)	$P = 0.328$	51 (24.2)

[a]The percentages are calculated out of the number of all subjects with AP according to the currently smoking group and never-smoked group

Table 2 The difference in the periapical status of teeth in current smokers and never smokers

		n	Mean	Std. deviation	95 % CI	Min	Max	*P*[a]
Fraction of endodontically treated teeth	Never smokers	151	0.11	0.10	0.09–0.12	0	0.43	
	Current smokers	108	0.12	0.11	0.10–0.14	0	0.54	0.246
Fraction of teeth with AP	Never smokers	151	0.10	0.10	0.08–0.11	0	0.45	
	Current smokers	108	0.13	0.11	0.11–0.15	0	0.56	0.025
Fraction of endodontically treated teeth with AP	Never smokers	151	0.06	0.07	0.05–0.07	0	0.39	
	Current smokers	108	0.06	0.07	0.05–0.07	0	0.30	0.832

[a]Independent samples *t* test

Table 3 Risk assessment for AP (0 = subject without single tooth with AP; 1 = subject with at least one tooth with AP) assessed by generalised linear model with binomial distribution and logit link

Parameter	B	Std. error	Sig.[a]	OR	95 % CI
Intercept	−34.125	0.639			
Gender = M	1.123	0.544	0.039	3.075	1.060–8.923
Smoking = yes	2.799	0.543	< 0.001	16.432	5.664–47.672
Age	0.328	0.016	< 0.001	1.388	1.346–1.431

OR odds ratio, *CI* confidence interval, *AIC* Akaike's information criterion, *BIC* Bayesian information criterion
[a]Offset: overall number of teeth
AIC = 495.632; BIC = 509.859

Table 4 Risk assessment for AP on endodontically treated teeth (0 = subject without single tooth with AP on endodontically treated teeth; 1 = subject with at least one tooth with AP on endodontically treated teeth) assessed by generalised linear model with binomial distribution and logit link

Parameter	B	Std. error	Sig.[a]	OR	95 % CI
Intercept	−1.319	0.469			
Gender = M	0.110	0.335	0.742	1.117	0.579–2.153
Smoking = yes	−0.102	0.322	0.751	0.903	0.481–1.696
Age	0.002	0.011	0.824	1.002	0.982–1.024
Fraction of endotreated teeth	−2.249	2.519	0.372	0.106	0.001–14.722

OR odds ratio, *AIC* Akaike's information criterion, *BIC* Bayesian information criterion
[a]Offset: number of endodontically treated teeth
AIC = 261.633; BIC = 279.418

The number of teeth with AP increases with increase in age, and male gender and smoking are also related to increased number of teeth with AP. The difference in the number of teeth with AP is expected to be 2.2 units higher for smokers compared to nonsmokers, while holding the other variables constant in the model (Table 5). The number of AP on endodontically treated teeth increases with increase in age and decreases with increase in fraction of endodontically treated teeth (Table 6).

Discussion

The study included adult patients attending dental service at the Dental Clinic of the Rijeka Clinical Hospital Centre, Croatia for the first time. The recruitment of subjects, exclusion and inclusion criteria were similar to those used in previous studies [12, 16]. In the present study subjects with DM and former smokers were excluded. In several previous studies, a statistically significant association between DM and radiographically diagnosed chronic AP was found, suggesting that diabetes can act as a confounding factor [17, 18].

A time period of 10–20 years was recently suggested for a resolution of oral tissues after heavy tobacco use [19]. The detrimental effect of tobacco is affected by the number of cigarettes the patient currently smokes, the intensity and duration of smoking and/or time since cessation [20]. This suggests that a broad categorisation of tobacco use is inadequate and makes the identification of former smokers as a separate category necessary. Since data considering smoking cessation obtained by an interview were not reliable, former smokers were excluded.

The overall prevalence of AP in the present study was 82 % which is comparable to similar studies conducted on patients referred to institutions, such as universities or specialist dental clinics [21, 22]. The major shortcoming of such study design is that the patients can be more severely diseased and therefore are not representative for the whole population. The prevalence of individuals with at least one endodontically treated tooth was 80 % which is comparable to 76 % of participants in a recently conducted study in Croatia [5].

The main purpose of the study was to investigate the possible influence of smoking on the prevalence of radiographic changes characteristic of AP. It was hypothesised that the periapical tissues of endodontically compromised teeth would be negatively affected by smoking, through influence on repair and healing events following endodontic treatment or disruption of catabolic and anabolic mechanisms and facilitation of periapical bone destruction. Consequently, increased size and/or number of radiologically detectable lesions would be expected in smokers [10]. Furthermore, smokers have increased caries prevalence due to decreased saliva-buffering effect and a higher number of lactobacilli and *Streptococcus mutans*, poorer oral hygiene, different eating habits (presumably consuming higher amount of

Table 5 Predictors of number of teeth with AP assessed by generalised linear model with negative binomial distribution and log link

Parameter	B	Std. error	Sig.[a]	OR	95 % CI
Intercept	−34.060	0.365			
Gender = M	1.543	0.263	<0.001	4.677	2.794–7.830
Smoking = yes	2.146	0.262	<0.001	8.554	5.118–14.298
Age	0.282	0.008	<0.001	1.325	1.304–1.347

OR odds ratio, *AIC* Akaike's information criterion, *BIC* Bayesian information criterion
[a]Offset: overall number of teeth
AIC = 2507.221; BIC = 2521.448

Table 6 Predictors of number of endodontically treated teeth with AP assessed by generalised linear model with negative binomial distribution and log link

Parameter	B	Std. error	Sig.[a]	OR	95 % CI
Intercept	−1.925	0.314			
Gender = M	0.036	0.206	0.861	1.037	0.693–1.552
Smoking = yes	0.009	0.197	0.963	1.009	0.686–1.485
Age	0.015	0.007	0.030	1.015	1.001–1.029
Fraction of endo-treated teeth	−11.399	0.997	< 0.001	1.12E-05	1.59E-06-7.91E-05

OR odds ratio, *AIC* Akaike's information criterion, *BIC* Bayesian information criterion
[a]Offset: number of endodontically treated teeth
AIC = 756.597; BIC = 774.381

sugar) and reduced frequency of dental recall than nonsmokers [6–8, 23–25]. Taken together, the above points can be construed to contribute to the increased prevalence of dental caries and consequently the periapical disease in smokers.

Although other studies found a higher prevalence of edentulism among smokers [26, 27], in the present study there were no significant differences between current smokers and never smokers considering the average number of teeth per patient. In the present study the prevalence of AP in current smokers was higher than in never smokers (86.1 vs. 78.1) although the difference was not statistically significant. However, a significantly higher fraction of teeth with AP was identified in currently smoking patients.

Considering data from cross-sectional studies, several studies revealed a slight, however, significant association with a reported OR between 1.4 and 4.4 between periapical pathosis and cigarette smoking in populations studied [11, 12, 28]. A recent study, analysing data from hypertensive patients, reported a very strong positive association with an OR 16.8 (95 % CI: 4.6–61.3) [13]. Bergstrom et al. did not find significant association between smoking and periapical lesions (OR 0.7; 95 % CI: 0.5–1.1; $P = 0.128$) [10]. In the present study, on average, a smoker was 16.4 times more likely to have AP than a nonsmoker. A strong association between smoking habit and increased number of teeth with AP was also demonstrated and smokers will on average have two more teeth with AP than nonsmokers, while controlling for gender, age and overall number of teeth. The present findings suggest that smoking might facilitate the extension of the periapical bone destruction, making it possible to detect the higher number of the periapical lesions on the conventional radiographs.

Age has previously been identified as risk indicator for AP [11]. This was confirmed in the present study, and basically each decade tends to increase the number of teeth with AP to three.

Gender appears to be related to AP, and present data demonstrated that men were more likely to have AP than women, and more teeth affected. This difference may reflect the greater interest of women in receiving dental care and attendance for check-ups.

Both endodontically treated teeth and endodontically treated teeth with AP appear to be present in similar numbers in current smokers and nonsmokers, and smoking does not seem to be related to AP in endodontically treated teeth. Although several studies reported a positive relationship between endodontically treated teeth and increased probability of AP [10–12], this was not confirmed by the present data. Moreover, when gender, age and smoking were controlled for, a higher fraction of endodontically treated teeth was related to decrease of AP in endodontically treated teeth. It may imply that quality of treatment is a significant predictor and that well-conducted endodontic treatment decreases AP, irrespective of smoking status or gender of patient.

This study has several limitations. It has been demonstrated by Brynolf et al. that apical inflammation is often present in the absence of radiological signs [29]. In the present study, only radiographs were used to assess the presence of AP, which might have resulted in underestimation of periapical pathosis. Similar to previous studies, smoking habit was treated as a dichotomous variable, whereas the influence of the intensity and duration of smoking habit was not investigated [11–13]. Although cross-sectional studies may demonstrate differences in the prevalence of AP, longitudinal studies may show differences between the smoking groups regarding the incidence, that is, the development of new periapical lesions within a time frame. The present study strongly supports the hypothesis that smoking influences the periapical status of teeth, but not endodontically treated teeth. However, since this study was cross-sectional by design, conclusions should be regarded as temporary until confirmed by long-term observations.

Acknowledgments
This study was supported by a grant from the University of Rijeka (818-10-1218), Republic of Croatia.

Ethical standards
All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008.

Informed consent was obtained from all patients for being included in the study.

Conflict of interest

Romana Peršić Bukmir, Marija Jurčević Grgić, Gordana Brumini, Stjepan Spalj, Sonja Pezelj-Ribaric, and Ivana Brekalo Pršo declare that there are no actual or potential conflicts of interest in relation to this article.

References

1. Glavak TkalićR, Miletiić G-M, Maričiić J, Wertag A. Substance abuse among the general population in the Republic of Croatia: research report. Zagreb: Ivo Pilar Institute of Social Sciences and Government of the Republic of Croatia—Drug Abuse Combating Office; 2012.
2. Kuzman M, Paviić Šimetin I, Pejnoviić Franeliić I, Pejak M, Hemen M ESPAD-The European school survey project on alcohol and other drugs, 2011. Zagreb: Croatian National Institute of Public Health; 2012.
3. Cohen S, Hargreaves KM. Pathways of the pulp. 9th Edition. St.Louis: Mosby Elsevier; 2006.
4. Hyman JJ, Cohen ME. The predictive value of endodontic diagnostic tests. Oral Surg Oral Med Oral Pathol. 1984;58(3):343–6.
5. Matijeviić J, Čizmekoviić Dadiić T, Prpiić Mehičiić G, Aniić I, Šlaj M, Jukiić Krmek S. Prevalence of apical periodontitis and quality of root canal fillings in population of Zagreb, Croatia: a cross-sectional study. Croat Med J. 2011;52(6):679–87.
6. Axelsson P, Paulander J, Lindhe J. Relationship between smoking and dental status in 35-, 50-, 65-, and 75-year-old individuals. J Clin Periodontal. 1998;25(4):297–305.
7. Jette AM, Feldman HA, Tennstedt SL. Tobacco use: a modifiable risk factor for dental disease among the elderly. Am J Public Health. 1993;83(9):1271–6.
8. Locker D. Smoking and oral health in older adults. Can J Public Health. 1992;83(6):429–32.
9. Bergström J, Eliasson S, Dock J. A 10-year prospective study of tobacco smoking and periodontal health. J Periodontal. 2000;71(8):1338–47.
10. Bergström J, Babcan J, Eliasson S. Tobacco smoking and dental periapical condition. Eur J Oral Sci. 2004;112(2):115–20.
11. Kirkevang LL, Wenzel A. Risk indicators for apical periodontitis. Community Dent Oral Epidemiol. 2003;31(1):59–67.
12. 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(4);41:310–6.
13. Segura-Egea JJ, Castellanos-Cosano L, Velasco-Ortega E, Ríos-Santos JV, Llamas-Carreras JM, Machuca G, López-Frías FJ. Relationship between smoking and endodontic variables in hypertensive patients. J Endod. 2011;37(6):764–7.
14. Ørstavik D, Kerekes K, Eriksen HM. The periapical index: a scoring system for radiographic assessment of apical periodontitis. Endod Dent Traumatol. 1986;2(1):20–34.
15. Landis JR, Koch GG. The measurement of observer agreement for categorical data. Biometrics. 1977 Mar;33(1):159–74.
16. Persic R, Kqiku L, Brumini G, Husetic M, Pezelj-Ribaric S, Brekalo Prso I, Städtler P. Difference in the periapical status of endodontically treated teeth between the samples of Croatian and Austrian adult patients. Croat Med J. 2011;15(6):672–8.
17. Segura-Egea JJ, Jiménez-Pinzón A, Ríos-Santos JV, Velasco-Ortega E, Cisneros-Cabello R, Poyato-Ferrera M. High prevalence of apical periodontitis amongst type 2 diabetic patients. Int Endod J. 2005;38(8):564–9.
18. López-López J, Jané-Salas E, Estrugo-Devesa A, Velasco-Ortega E, Martín-González J, Segura-Egea JJ. Periapical and endodontic status of type 2 diabetic patients in Catalonia, Spain: a cross-sectional study. J Endod. 2011;37(5):598–601.
19. Warnakulasuriya S, Dietrich T, Bornstein MM, Casals Peidró E, Preshaw PM, Walter C, Wennström JL, Bergström J. Oral health risks of tobacco use and effects of cessation. Review. Int Dent J. 2010;60(1):7–30.
20. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. J Periodontal. 2000;71(5):743–51.
21. Tsuneishi M, Yamamoto T, Yamanaka R, Tamaki N, Sakamoto T, Tsuji K, Watanabe T. Radiographic evaluation of periapical status and prevalence of endodontic treatment in an adult Japanese population. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2005;100(5):631–5.
22. Jersa I, Kundzina R. Periapical status and quality of root fillings in a selected adult Riga population. Stomatologija. 2013;15(3):73–7.
23. Talhout R, Opperhuizen A, van Amsterdam JG. Sugars as tobacco ingredient: effects on mainstream smoke composition. Food Chem Toxicol. 2006;44(11):1789–98.
24. Hirsch JM, Livian G, Edward S, Noren JG. Tobacco habits among teenagers in the city of Göteborg, Sweden, and possible association with dental caries. Swed Dent J. 1991;15(3):117–23.
25. Sherwood NE, Hennrikus DJ, Jeffery RW, Lando HA, Murray DM. Smokers with multiple behavioural risk factors: how are they different? Prev Med. 2000;31(4):299–307.
26. Finlayson TL, Brown TT, Fulton BD, Jahedi S. Adult oral health status in California, 1995–2006: demographic factors associated with tooth loss due to disease. J Calif Dent Assoc. 2009;37(8):561–70.
27. Millar WJ, Locker D. Smoking and oral health status. J Can Dent Assoc. 2007;73(2):155.
28. Frisk F, Hakeberg M. Socio-economic risk indicators for apical periodontitis. Acta Odontol Scand. 2006;64(2):123–8.
29. Brynolf I. A histological and roentgenological study of the periapical region of human upper incisors. Odontologisk Revy. 1967;18:1–176.