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High prevalence of apical periodontitis amongst smokers in a sample of Spanish adults

J. J. Segura-Egea[1], A. Jiménez-Pinzón[1], J. V. Ríos-Santos[1], E. Velasco-Ortega[1], R. Cisneros-Cabello[2] & M. M. Poyato-Ferrera[1]

[1]Department of Stomatology, School of Dentistry, University of Seville, Seville; and [2]Department of Stomatology, School of Dentistry, European University of Madrid, Madrid, Spain

Abstract

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Aim To study the prevalence of apical periodontitis in smoker and nonsmoker patients.

Methodology In a cross-sectional study, the records of 180 subjects, 109 smokers and 71 nonsmokers, were examined. All participants underwent a full-mouth radiographic survey incorporating 14 periapical radiographs. The periapical region of all teeth, excluding third molars, was examined. Periapical status was assessed using the Periapical Index score. Statistical analyses were conducted using the Cohen's Kappa test, analysis of variance and logistic regression.

Results Apical periodontitis in at least one tooth was found in 74% of smokers and in 41% of nonsmokers ($P < 0.01$; odds ratio = 4.2; 95% C.I. = 2.2–7.9). Amongst smoker patients 5% of the teeth had apical periodontitis, whereas in nonsmoker subjects 3% of teeth were affected ($P = 0.008$; odds ratio = 1.5; 95% C. I. = 1.1–2.1). The percentage of root filled teeth in smoker and nonsmoker patients was 2.5% and 1.5%, respectively ($P < 0.05$; odds ratio = 1.7; C.I. 95% = 1.0–2.6).

Conclusions In this study population, smoking was significantly associated with a greater frequency of root canal treatment and with an increased prevalence of apical periodontitis.

Keywords: apical periodontitis, endodontics, smoking.

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Introduction

Tobacco smoking in Spain is widespread, with one third of the Spanish population more than 16-year-old smoking daily (Infante & Rubio-Colavida 2004). Males in general smoke more than females, although during adolescence and early adulthood the reverse is true (Infante & Rubio-Colavida 2004). In the last few years, tobacco smoking has been stable, in spite of the gender-related changes. The prevalent consumption pattern is of daily use and the average consumption is 15 cigarettes per day (Infante & Rubio-Colavida 2004).

Apical periodontitis (AP) is primarily a sequel to dental caries caused by infection of the root canal system. Several epidemiological investigations have reported a high prevalence for AP ranging from 1.4% (Eriksen *et al.* 1998) to 8.0% (Imfeld 1991) using the tooth as unit. When individuals are used as the unit, the prevalence can be as high as 61.1%, and increases with age (Figdor 2002, Jiménez-Pinzón *et al.* 2004, Ridao-Sacie *et al.* 2007). Root canal treatment is the conservation treatment for teeth with AP. Several cross-sectional studies have attempted to identify risk indicators of AP. The radiographic evidence of root fillings, the presence of several caries lesions, the quality of the dental treatment, the regularity of dental visits and type II diabetes mellitus have been showed to be associated statistically with AP (Kirkevang & Wenzel 2003, Segura-Egea *et al.* 2004, 2005).

The harmful effects of tobacco smoking on periodontal bone have been demonstrated in several cross-sectional and longitudinal studies (Krall *et al.* 1999,

Correspondence: Dr Juan J. Segura-Egea; Cueva de Menga nº 1, portal 3, piso 6º-C, 41020-Seville, Spain (e-mail: segurajj@us.es).

Bergström *et al.* 2000). On this basis, it was assumed that it might be a risk factor for AP, through a negative influence on the apical periodontium of endodontically compromised teeth, facilitating the extension of the process of periapical bone destruction and/or interfering with healing and repair events following endodontic treatment. Consequently, an increased number and/or size of periapical lesions would be expected in smokers (Bergström *et al.* 2004).

Kirkevang & Wenzel (2003) reported for the first time in an epidemiological study on the association between tobacco smoking an AP. Recently, several reports have also studied this association with contradictory results (Bergström *et al.* 2004, Marending *et al.* 2005).

The aim of the present study was to investigate the prevalence of AP in root filled and untreated teeth in Spanish smoker patients and nonsmoker control subjects.

Material and methods

The sample consisted of 180 subjects, aged 37.1 ± 15.7 years, 66 male subjects (36.7%) and 114 female subjects (63.3%), presenting consecutively as new patients seeking routine dental care (not emergency care) at the University of Seville, Faculty of Dentistry. The criteria for inclusion in the study were that the patients should be attending for the first time. Patients younger than 18 years and patients having less than eight remaining teeth were excluded. The scientific committee of the Dental Faculty approved the study and all the patients gave written informed consent.

All participants underwent a full-mouth radiographic survey consisting of 14 periapical radiographs. All radiographs were taken with a Trophy CCX X-ray unit (Trophy Radiologie–94300, Vincennes, France) using the long-cone paralleling technique, setting of 70 kV, 10 mA, a film-focus distance of 28 cm, and Ultra Speed film (Eastman Kodak, Rochester, NY, USA). The radiographs were processed according to the manufacturer.

From the full-mouth radiographic survey all teeth, excluding third molars, were recorded. Teeth were categorized as root filled if they had been filled with a radiopaque material in the root canal(s), as described previously (De Moor *et al.* 2000, Segura-Egea *et al.* 2005, Sunay *et al.* 2007). The following information was recorded on a structured form for each subject: (a) number of teeth present; (b) number and location of teeth without root fillings (untreated teeth) having identifiable periapical lesions and (c) number and location of root filled teeth. The periapical status was assessed using the 'Periapical Index' (PAI) (Ørstavik *et al.* 1986) (Table 1). Each category used in the PAI represents a step on an ordinal scale of registration of periapical inflammation. The worst score of all roots was taken to represent the PAI score for multirooted teeth.

Table 1 Periapical Index (Ørstavik *et al.* 1986)

Score	Criteria
1	Normal periapical structures
2	Small changes in bone structure
3	Changes in bone structure with some mineral loss
4	Periodontitis with well defined radiolucent area
5	Severe periodontitis with exacerbating features

One observer with 6 years of clinical experience in endodontics examined the radiographs. The method of viewing the radiographs was standardized; films were examined in a darkened room using an illuminated viewer box with magnification (3.5×) whilst mounted in a cardboard slit to block off ambient light emanating from the viewer. Before evaluation, the observer participated in a calibration course for the PAI system, which consisted of 100 radiographic images of teeth, some root filled and some not. Each tooth was assigned to one of the five PAI-scores using visual references (Ørstavik *et al.* 1986) for the five categories within the scale (Fig. 1). After scoring the teeth, the results were compared to a 'gold standard atlas', and Cohen's *Kappa* was calculated as 0.71.

Intra-observer reproducibility was evaluated by the repeat scoring of 50 patients 2 months after the first examination. These patients were randomly selected. Before the second evaluation of the radiographs, the observer was recalibrated in the PAI system by scoring the 100 standard images. The intraobserver agreement test on PAI scores on the 50 patients produced a Cohen's *Kappa* of 0.77.

A score greater than 2 (PAI > 2) was considered to be a sign of periapical patterns (Ørstavik *et al.* 1986). The periapical status of all teeth was assessed.

Raw data were entered into Excel® (Microsoft Corporation, Redmond, WA, USA). The analyses were completed in a SPSS environment (SPSS, Inc, Version 11, Chicago, IL, USA). Analysis of variance and logistic regression were used to determine the significance of differences between smoker and nonsmoker patients for the parameters: number of teeth with AP, number of

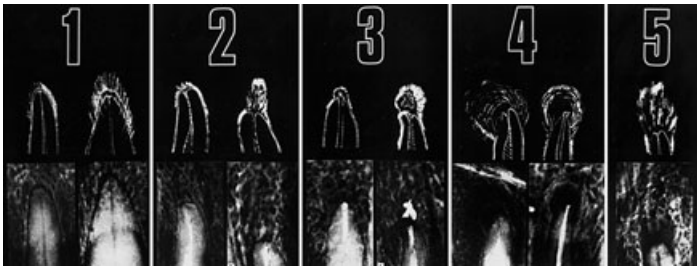

Figure 1 Visual references used for the evaluation of the roots using the Periapical Index-score system (Reproduced from Ørstavik *et al.* 1986, with permission from Blackwell Publishing).

root-filled teeth, number of root-filled teeth with AP, and number of untreated teeth with AP. Data are reported as mean ± SD.

Results

Amongst the study participants, 109 were current smokers (61%) and 71 were nonsmokers (39%). The mean age was 35.0 ± 2.6 years for current smokers and 40.3 ± 3.1 years for nonsmokers. The study population according to age and smoking is presented in Table 2. The average number of teeth per patient was 25.0 ± 3.8 and 24.4 ± 4.5 teeth in smoker and nonsmoker patients, respectively ($P > 0.05$).

Apical periodontitis in one or more teeth was found in 81 smoker patients (74%) and in 29 nonsmoker subjects (41%) ($P < 0.01$; odds ratio = 4.2; C.I. 95% = 2.2–7.9) (Table 3). The average number of teeth with AP was 1.2 ± 1.1 and 0.8 ± 1.1 teeth in smokers and nonsmokers, respectively ($P > 0.05$). One or more root-filled teeth were found in 47% (51) and 31% (22) of smoker and nonsmoker subjects, respectively ($P < 0.05$; odds ratio = 2.0; C.I. 95% = 1.0–3.7). Amongst smoker patients with root-filled teeth, 36 (71%) had AP affecting at least one treated tooth. Amongst nonsmokers with root-filled teeth, 12 (55%) had AP affecting at least one treated tooth ($P = 0.19$).

Table 2 Study population according to age and smoking

Age group (year)	Mean age (year)	Smokers *n* (%)	Nonsmokers *n* (%)	Total *n* (%)
18–25	21.8	31 (28)	21 (30)	52 (29)
26–35	30.6	37 (34)	19 (27)	56 (31)
36–45	40.9	19 (17)	7 (10)	26 (14)
46–55	49.8	14 (13)	3 (4)	17 (9)
56–65	61.5	2 (2)	8 (11)	10 (6)
>65	69.0	6 (6)	13 (18)	19 (11)
Total	37.1	109 (100)	71 (100)	180 (100)

Table 3 Prevalence of apical periodontitis (AP), root-filled teeth (RFT), and root-filled teeth with apical periodontitis (RFT-AP) in smoker ($n = 109$) and nonsmoker ($n = 71$) subjects

	AP (%)	RFT (%)	RFT-AP (%)
Smokers	81 (74)	51 (47)	36 (71)
Nonsmokers	29 (41)	22 (31)	12 (55)
Total	110 (61)*	73 (41)**	48 (66)

RFT-AP are out of all RFTs. *$P < 0.01$; **$P < 0.05$.

Univariate logistic regressions were run with number of teeth, endodontic treatment (yes/no), smoking (yes/no), gender (male/female), and age as independent variables, one at a time, and periapical lesions, dichotomized (present/absent), as the dependent variable. The results of the logistic regression for univariate analyses are demonstrated in Table 4. The analysis suggested that smoking and endodontic treatments were factors associated with increased risk for periapical lesions. Age, gender and number of teeth were not associated with an increased risk. In a multivariate analysis including all the above factors, endodontic treatment (odds ratio = 4.1, C.I. 95% = 2.1–8.1) and smoking (odds ratio = 4.2, C.I. 95% = 2.2–7.9) remained significant, whereas all other factors were not statistically significant (Table 5).

The total number of teeth examined in the smokers was 2722; 131 (5%) had AP (PAI ≥ 3). On the contrary, of the 1731 teeth examined in nonsmoker patients only 55 (3%) had AP ($P = 0.008$; odds ratio = 1.5; C.I. 95% = 1.1–2.1) (Table 6). The number of root-filled teeth in smoker and nonsmoker patients was 67 (2.5%) and 26 (1.5%), respectively ($P = 0.03$; odds ratio = 1.7; C.I. 95% = 1.0–2.6). Amongst smokers, 45 root-filled teeth (67%) had AP, whereas in nonsmokers were 15 (58%) the root-filled teeth were associated with AP ($P = 0.4$; odds ratio = 1.5; C.I. 95% = 0.6–3.8). Finally, amongst un-treated teeth, 86 (3.2%) and 40 (2.3%) were associated to AP in smoker and nonsmoker subjects,

Table 4 Logistic regression analyse (univariate analysis) of the influence of the independent variables age, gender (male/female), smoking status (yes/no), root-filled teeth (RFT) (present/absent), and number of teeth, on the dependent variable 'apical periodontitis' (present/absent). Odds ratio was calculated as Exp (B)

Independent variables	B	*P*	Odds ratio	C. I. 95% Inf. limit	C. I. 95% Sup. limit
Age	0.0142	0.1597	1.0143	0.9944	1.0347
Gender	0.0673	0.8325	1.0696	0.5735	1.9947
Smoking	1.4326	0.0000	4.1897	2.2110	7.9392
RFT	1.4085	0.0001	4.0897	2.0664	8.0944
Teeth (*n*)	−0.0714	0.0898	0.9311	0.8573	1.0112

Table 5 Logistic regression analyse (multivariate analysis) of the influence of the independent variables age, gender (male/female), smoking status (yes/no), root-filled teeth (RFT) (present/absent), and number of teeth, on the dependent variable 'apical periodontitis' (present/absent). Odds ratio was calculated as Exp (B)

Independent variables	B	*P*	Odds ratio	C. I. 95% Inf. limit	C. I. 95% Sup. limit
Age	0.0170	0.2216	1.0172	0.9898	1.0453
Gender	0.0825	0.8235	1.0860	0.5262	2.2413
Smoking	1.4923	0.0000	4.4474	2.2066	8.9634
RFT	1.2807	0.0006	3.5991	1.7306	7.4852
Teeth (*n*)	−0.0358	0.5223	0.9649	0.8648	1.0766

Table 6 Distribution of teeth with apical periodontitis (AP), root-filled teeth (RFT), root-filled teeth with apical periodontitis (RFT-AP) and untreated teeth with apical periodontitis (UT-AP) in smokers and nonsmokers

	Total teeth	AP	RFT	RFT-AP	UT-AP
Smokers	2722	131 (4.8)	67 (2.5)	45 (67.2)	86 (3.2)
Nonsmokers	1731	55 (3.2)	26 (1.5)	15 (57.7)	40 (2.3)
Total	4453	186 (4.2)	93 (2.1)	60 (64.5)	126 (2.8)
OR Nonsmokers		1.0	1.0	1.0	1.0
OR Smokers		1.5***	1.7**	1.5*	1.4*

RFT-AP are out of all RFTs.
OR, odds ratio; *$P > 0.05$; **$P < 0.05$; ***$P < 0.01$.

respectively ($P = 0.086$; odds ratio = 1.4; C.I. 95% = 1.0–2.0).

Discussion

The subjects included in this cross-sectional study were adult patients attending for the first time the dental service of the Faculty of Dentistry of Seville (Spain). The recruitment of subjects was the same as those used by others (Kirkevang *et al.* 2000, Bergström *et al.* 2004).

Periapical radiography was used to evaluate the presence of AP. Previous studies have also used periapical radiographs (Imfeld 1991, Kirkevang *et al.* 2001, Boucher *et al.* 2002, Kirkevang & Wenzel 2003). Moreover, the PAI used for scoring periapical status (PAI) was first described for periapical radiographs (Ørstavik *et al.* 1986) and has been widely used in the literature (Eriksen *et al.* 1995, Marques *et al.* 1998, Sidaravicius *et al.* 1999, Kirkevang *et al.* 2001, Boucher *et al.* 2002, Kirkevang & Wenzel 2003, Segura-Egea *et al.* 2004). Periapical regions of all the teeth, excluding only third molars, were radiographically evaluated. Thus, the results report accurately the periapical status of the subjects. Other authors, in similar studies, have excluded teeth with absent or defective coronal restorations, teeth with their periradicular tissues near radiolucent anatomic structures, or root-filled teeth with inadequate root canal treatment (Britto *et al.* 2003). However, these exclusions necessarily alter the results and prevent the determination of the real periapical status of the subjects.

No significant differences in the number of teeth between smokers and nonsmokers were found. On the contrary, other investigators found a higher prevalence

of edentulism amongst smokers (Millar & Locker 2007). The study carried out by Lang *et al.* (1997) concluded that the number of missing teeth was a good indicator of oral health status. As a result, it can be considered that the oral health status of smokers and nonsmokers was comparable.

The total number of teeth with AP (PAI ≥ 3) was 186, representing 4.2% of the total. The frequency of teeth with AP in other studies varies from 0.6% (Eriksen *et al.* 1995) to 12% (Kabak & Abbott 2005). Recently, Sunay *et al.* (2007) have reported that, on teeth examined in a sample of Turkish adults, 4.2% had visible periapical radiolucencies. The range is wide, probably due to the variation amongst populations examined.

According to the risk assessment performed, root canal treatment and smoking were potential risk factors identified. Root-filled teeth were significantly more frequently affected by periapical lesions as non-treated teeth ($P < 0.01$; odds ratio = 4.1; C.I. 95% = 2.1–8.1). These observations confirm previous observations of the relation between AP and root canal treatment (Jiménez-Pinzón *et al.* 2004).

The main purpose of the present study was to investigate the possible influence of smoking on the prevalence of AP. The results, as well as the regression analysis, show that the prevalence of AP in smokers is significantly higher than in control subjects ($P < 0.01$; odds ratio = 4.2; C. I. 95% = 2.2–7.9). The frequency of teeth affected with AP amongst smokers (5%) was significantly higher ($P = 0.008$; odds ratio = 1.5; C.I. 95% = 1.1–2.1) than in nonsmokers (3%). These results are in agreement with the results reported by Kirkevang & Wenzel (2003) who found an statistical association between smoking and AP ($P = 0.05$; odds ratio = 1.64; C. I. 95% = 1.0–2.8). Moreover, current evidence would indicate that smoking is a significant risk factor in inflammation of the marginal periodontium (Bergström *et al.* 2000, Johnson & Hill 2004, Labriola *et al.* 2005) and therefore it may be hypothesized that it would have a similar effect on the apical periodontium. Recently, Kirkevang *et al.* (2007) have also reported that smoking is a statistically significant risk factor for AP when assessed separately (odds ratio = 1.9; C.I. 95% = 1.3–2.8) but had a reduced, and nonsignificant, effect on the risk of developing AP when adjusting for age and reduced marginal bone level (odds ratio = 1.3; C. I. 95% = 0.9–2.1). The authors claimed that the strong correlation between smoking and marginal periodontitis found in the studies cited previously may, at least partly, explain why the association between smoking and AP was reduced when both smoking and marginal bone level were entered into the analysis.

Both the present study and the studies performed by Kirkevang & Wenzel (2004) and Kirkevang & Wenzel (2003) only assessed the periapical condition by radiographic methods and there was no clinical examination carried out, and it is recognized that radiographic evaluation is not a perfect method of assessment as apical inflammation can be present in the absence of radiological signs (Duncan & Pitt Ford 2006). This has been demonstrated in histological evaluation of maxillary teeth (Brynolf 1967). Kirkevang & Wenzel (2003) discussed that in several orthopaedic studies bony healing was slower in smokers than in nonsmokers (Haverstock & Mandracchia 1998, Castillo *et al.* 2005) suggesting this delay in healing may result in an over-representation of disease in the smoking group in their study.

On the contrary, other studies have not found any association (Bergström *et al.* 2004, Marending *et al.* 2005). Bergström *et al.* (2004) in a cross-sectional study retrospectively examined 247 intra-oral radiographs of smokers, nonsmokers and former smokers and compared for incidence of apical disease. Although the mean number of periapical lesions was 6% in smokers, 4% in former smokers and 3% in nonsmokers, the association between smoking and periapical lesions was not significant after controlling for age. This study did not examine a random sample of a general population as had the initial study (Kirkevang & Wenzel 2003), but rather examined a subpopulation of Swedish musicians, who may differ from the general population. As the authors themselves admitted (Bergström *et al.* 2004) the investigation was cross-sectional in design and the conclusion should be regarded as temporary until confirmed by long-term observations.

Marending *et al.* (2005) have studied the impact of many patient-related factors with the outcome of root canal treatment and reported that smoking had negligible impact. Nevertheless, any conclusions from this study are limited owing to the small number of patients in each group, 17 smokers and 31 nonsmokers.

Currently, all the studies carried out to investigate the association between smoking and AP are cross-sectional studies. It is difficult to control for confounding factors in cross-sectional studies particularly when any influence on AP is likely to be multifactorial. Confounding factors such as caries, socio-economic class and regularity of dental care are likely to be better

controlled in the subpopulation than the general population and this may account for the differing results between the studies (Duncan & Pitt Ford 2006).

The percentage of subjects having at least one root filled tooth varied significantly in smokers (47%) and nonsmokers (31%) ($P < 0.05$; odds ratio = 2.0), as well as the number of root filled teeth ($P < 0.05$; odds ratio = 1.7), suggesting that cigarette smoking increases the risk of root canal treatment. This finding is in agreement with the results of the longitudinal study carried out by Krall *et al.* (2006), who reported a significantly dose response relationship between cigarette smoking and the risk of root canal treatment. These authors calculated that, compared with never-smokers, current cigarette smokers were 1.7 times as likely to have root canal treatment ($P < 0.001$).

The prevalence of root canal treatment between smokers and nonsmokers was low compared to previous reports (Imfeld 1991, Sidaravicius *et al.* 1999). However, the frequency of root canal treatment found in this study can be considered normal in comparison with the prevalence of root filled teeth determined previously in the Spanish population (41%) (Jiménez-Pinzón *et al.* 2004).

The number of AP in root filled teeth between both groups did not differ significantly. If smoking had an effect on bone healing, more lesions would be expected around root filled teeth in smokers. That counts against an effect of smoking on bone healing. So, even though the results reported in the present study show a statistical association between smoking and the prevalence of AP, it cannot be ruled out that the presence of confounding factors, such as smokers taking less care of their dentition, their health, visit therefore less frequently the dentist on a regular basis and may develop more caries and therefore more AP. Longitudinal studies are required to make firm conclusions.

Conclusions

The data reported in the present study, taken together with previous reports (Kirkevang & Wenzel 2003, Krall *et al.* 2006, Kirkevang *et al.* 2007) support the concept that smoking is associated with an increase in filled teeth and is associated to an increase in the prevalence of AP, being able to act as a risk factor for AP. However, confounding factors can not be ruled out and longitudinal studies are required to make firm conclusions.

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