High prevalence of apical periodontitis amongst type 2 diabetic patients

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

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. *International Endodontic Journal*, **38**, 564–569, 2005.

Aim To study the prevalence of AP in patients with and without type 2 diabetes mellitus.

Methodology In a retrospective cohort study, the records of 38 subjects with diabetes and 32 control subjects 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 κ test, analysis of variance and logistic regression.

Results Apical periodontitis in at least one tooth was found in 81.3% of diabetic patients and in 58% of control subjects ($P = 0.040$; OR = 3.2; 95% CI = 1.1–9.4). Amongst diabetic patients 7% of the teeth had AP, whereas in the control subjects 4% of teeth were affected ($P = 0.007$; OR = 1.8; 95% CI = 1.2–2.8).

Conclusions Type 2 diabetes mellitus is significantly associated with an increased prevalence of AP.

Keywords: apical periodontitis, diabetes mellitus, endodontics.

Received 18 January 2005; accepted 26 April 2005

Introduction

Diabetes mellitus is a syndrome characterized by abnormalities in carbohydrate, lipid and protein metabolism that results either from profound or an absolute deficiency of insulin (type 1) or from target tissue resistance to its cellular metabolic effects (type 2) (Vernillo 2003). Diabetes also affects many functions of the immune system and is associated with delayed healing and compromised immune responses (Delamaire *et al.* 1997). The oral complications of uncontrolled diabetes mellitus can include xerostomia, infection, poor healing, increased incidence and severity of caries, candidiasis, gingivitis, periodontal disease and burning mouth syndrome (Little *et al.* 1997). Aggressive forms of periodontal disease have been associated with increased serum glucose levels (Katz 2001) especially, when poorly controlled (Soskolne & Klinger 2001). Recently, high incidence of periodontal attachment loss has been described amongst diabetics (Thomson *et al.* 2004).

On the contrary, apical periodontitis (AP) is primarily a sequela to dental caries caused by infection of the root canal system. Periradicular lesions consecutive to AP result from irritation of the periradicular tissues by polymicrobial irritants from root canals, in teeth with necrotic pulps. Several epidemiological investigations have shown 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 increase with age (Figdor 2002, Jiménez-Pinzón *et al.* 2004). Root canal treatment is the elective treatment for teeth with AP that must be preserved. There is a biological basis to suggest that diabetes mellitus can affect the periapical immune response causing a

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

delayed healing process. Consequently, there could be expected a higher prevalence of periapical lesions and higher rate of post-treatment disease in diabetic patients than in control subjects without diabetes. However, the literature on the pathogenesis, progression and healing of endodontic pathosis in diabetic patients is remarkably sparse. Few studies have investigated clinically in humans the possible association between diabetes mellitus and AP (Falk *et al.* 1989, Bender & Bender 2003, Britto *et al.* 2003, Fouad & Burleson 2003).

The aim of the present study was to investigate the prevalence of AP in root filled teeth and untreated teeth in diabetic patients and control subjects without diabetes.

Materials and methods

Amongst the patients seeking routine dental care at the University of Seville, Faculty of Dentistry, 32 subjects reporting a history of well-controlled type 2 diabetes mellitus diagnosed according to the fasting plasma glucose and 75-g oral glucose tolerance test. An additional 38 patients who were within the age range of the diabetic patients, who reported no history of diabetes and normal glucose tolerance, served as control subjects. The total sample consisted of 70 subjects, 29 males (41.4%) and 41 females (58.6%). The mean and standard deviation for age were 61.0 and 8.4 years, respectively. The scientific committee of the Dental Faculty approved the study and all the patients gave written informed consent.

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

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, 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).

From the full-mouth radiographic survey all teeth, excluding third molars, were recorded. Teeth were categorized as root filled teeth if they had been filled with a radiopaque material in the root canal(s). The following information was recorded on a structured form for each subject: (i) number of teeth present; (ii) number and location of teeth without root fillings (untreated teeth) having identifiable periapical lesions and (iii) 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 were taken to represent the PAI score for multirooted teeth.

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 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 κ was 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.

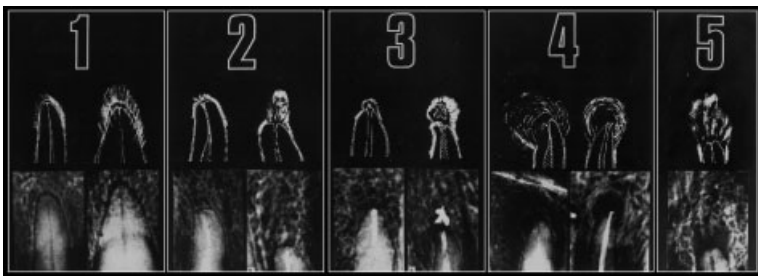

Figure 1 Visual references used for the evaluation of the roots using the PAI score system (Ørstavik *et al.* 1986).

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 κ of 0.77.

PAI score >2 was considered to be a sign of periapical pathology (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 carried out in an SPSS environment (Version 11; SPSS Inc., Chicago, IL, USA). Analysis of variance and logistic regression were used to determine the significance of differences between diabetic and nondiabetic patients for the parameters: number of teeth with AP, number of root filled teeth, number of root filled teeth with AP and number of untreated filled teeth with AP. Data are reported as mean ± standard deviation.

Results

The study group consisted of 32 patients with diabetes, 12 men and 20 women, ranging from 43 to 74 years old (63.1 ± 8.3 years). The control group consisted of 38 subjects without diabetes, 16 men and 22 women, ranging from 46 to 74 years (59.6 ± 7.4 years) ($P > 0.05$). The average number of teeth per patient was 21.6 ± 2.8 and 25.4 ± 4.1 teeth in diabetic and control groups, respectively ($P = 0.025$). Apical periodontitis in one or more teeth was found in 26 diabetic patients (81%) and in 22 control subjects (58%) ($P = 0.040$; OR = 3.2; 95% CI = 1.1–9.4) (Tables 2 and 3). The average number of teeth with AP were 1.5 ± 1.1 and 0.9 ± 1.1 teeth in diabetic and control subjects, respectively ($P > 0.05$). One or more root-filled teeth were found in 31% (10) and 42% (16) of diabetic and control subjects, respectively ($P = 0.25$). Amongst diabetic patients with root filled teeth, seven (70%) had AP affecting at least one treated tooth. In control subjects with root-filled teeth, 10 (63%) had AP affecting at least one treated tooth ($P = 0.34$).

Table 2 Prevalence of AP, root filled teeth (RFT) and root-filled teeth with apical periodontitis (RFT-AP) in diabetic ($n = 32$) and control ($n = 38$) subjects

	AP (%)	RFT (%)	RFT-AP (%)
Diabetic	26 (81)	10 (31)	7 (70)
Control	22 (58)	16 (42)	10 (63)
Total	48 (69)	26 (37)	17 (65)

RFT-AP are out of all RFTs.

Table 3 Logistic regression analyses of the influence of the independent variable 'diabetic status' on the dependent variables AP, RFT and RFT-AP

Dependent variable	*B*	*P*	OR	95% CI
AP	1.148	0.04	3.15	1.05–9.44
RFT	−0.577	0.25	0.56	0.21–1.50
RFT-AP	0.575	0.34	1.78	0.54–5.81

Table 4 Distribution of teeth with AP, RFT, RFT-AP and untreated teeth with apical periodontitis in diabetic and control subjects

	Total teeth	AP	RFT	RFT-AP	UT-AP
Diabetic	692	48 (7)	12 (2)	10 (83)	38 (6)
Control	966	38 (4)	20 (2)	12 (60)	26 (3)
Total	1658	86 (5)	32 (2)	22 (69)	64 (4)
OR					
Control		1.0	1.0	1.0	1.0
Diabetic		1.8**	0.8*	3.3*	2.1**

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

The total number of teeth examined in the study group was 692; 48 (6.9%) had AP (PAI ≥ 3). On the contrary, amongst the 966 teeth examined in the control group only 38 (4%) had AP ($P = 0.007$; OR = 1.8; 95% CI = 1.2–2.8) (Table 4). There was no significant difference between groups in the percentages of PAI scores ≥3 ($P = 0.20$).

The number of root filled teeth in the study and control groups were 12 (2%) and 20 (2%), respectively ($P = 0.62$) (Table 4). Amongst diabetic patients, 10 root filled teeth (83%) had AP, whereas in the control subjects 12 root filled teeth (60%) had AP ($P = 0.17$). Finally, amongst untreated teeth, 38 (6%) and 26 (3%) were associated with AP in diabetic patients and in control subjects, respectively ($P = 0.004$; OR = 2.1; 95% CI = 1.3–3.5).

Discussion

The subjects included in this retrospective cohort 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, Britto *et al.* 2003, Fouad & Burleson 2003). Both the study and the control cohorts consisted of more women than men, however, epidemiological studies reported that gender had no effect on the presence of AP or the frequency of root canal treatment (Ørstavik *et al.* 1986, Jiménez-Pinzón *et al.* 2004). There was no significant difference in the age of the groups.

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, Britto *et al.* 2003, Kirkevang & Wenzel 2003). Moreover, the PAI used for scoring periapical status 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 areas of all the teeth, excluding only third molars, were radiographically evaluated. Thus, the results reproduced 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.

The average number of teeth were lower in diabetic patients (21.6 ± 2.8) than in control subjects (25.4 ± 4.1) ($P = 0.025$). These findings are in accordance with numerous reports, documenting convincingly, that diabetes mellitus, especially when poorly controlled, is associated with significant tooth loss due to the increased incidence and severity of caries and aggressive forms of periodontal disease (Little *et al.* 1997, Katz 2001, Soskolne & Klinger 2001, Fouad 2003, Lagervall *et al.* 2003, Vernillo 2003). On the contrary, Falk *et al.* (1989) did not find significant differences in the number of teeth between diabetic and nondiabetic subjects. However, in the present study, patients with seven or fewer remaining teeth were excluded because they often had periodontal disease (De Cleen *et al.* 1993, Lupi-Pegurier *et al.* 2002). Thus, the influence of periodontal disease has been reduced.

The results of the present study show that the prevalence of AP in diabetic patients is significantly higher than in control subjects ($P = 0.04$; OR = 3.2; 95% CI = 1.1–9.4). This appears to be the first epidemiological study that has demonstrated a significant correlation between diabetes mellitus and AP. In an earlier report (Bender *et al.* 1963) it was proposed that healing of periapical lesions would not occur if diabetes was not controlled and that the lesions will increase in size despite root canal treatment. Later, it has been found that long duration diabetics exhibited teeth with periapical lesions to a greater extent than short duration diabetics and nondiabetic subjects (Falk *et al.* 1989) and it was reported that patients with diabetes mellitus had a disproportionately high percentage of clinically severe pulpal or periodontal infections (Ueta *et al.* 1993).

Recently, in a similar design study (Britto *et al.* 2003) one or more teeth with AP were found in 97% of diabetic patients (81% in the present study) and in 87% of control subjects (58% in the present study) and no significant differences in the prevalence of AP between both groups were reported. However, these investigators excluded teeth with absent or defective coronal restorations, teeth with periradicular tissues near radiolucent anatomic structures and root filled teeth with inadequate root canal treatment. Because of this, their results do not reflect the real periapical status of the subjects studied and the comparison between both groups cannot produce definite conclusions.

The total number of teeth with AP (PAI ≥ 3) was 86, representing 5.2% of the total. The frequency of teeth with AP in other studies varies from 0.6% (Eriksen *et al.* 1995) to 9.8% (Allard & Palmqvist 1986). The range was wide, probably due to the variation amongst populations examined. The frequency of teeth affected with AP amongst diabetic patients (6.9%) was twice ($P = 0.007$; OR = 1.8) than in the control group (3.9%). These results are in agreement with a previous report (Falk *et al.* 1989) that found a group of individuals amongst patients with diabetes who had more periradicular lesions than did those without diabetes. However, OR, confidence intervals and *P*-values arising from analyses in which the tooth is the unit risk serious anticonservatism.

Several animal models have been developed to study the relation between diabetes and periapical lesions. In an animal study, diabetes was induced in rats by using streptozotocin (Kohsaka *et al.* 1996). Diabetic rats showed more severe inflammation in apical periodontal ligament, root resorption and alveolar bone resorption than control rats. Moreover, the lesions in the periradicular area were significantly larger than those in control. Another study (Fouad *et al.* 2002) showed a more severe outcome in a type 1 diabetic mouse model after the inoculation of the exposed pulp with a mixture of facultative and anaerobic bacteria, compared with the control mice. Recently, it has been reported (Iwama *et al.* 2003) that alveolar bone resorption was most severe and periradicular lesions were largest in diabetic rats given a 30% sucrose solution, concluding that the metabolic conditions produced by type 2 diabetes

mellitus enhance the development of periradicular lesions in rats. On the contrary, the results of the present study did not show significant differences in the PAI score (that evaluate the size of the periapical radiolucency) between diabetic and control teeth with AP (PAI scores ≥3). However, it is considered that the diabetic group in this study showed a good degree of glycaemic control.

The percentages of subjects having at least one root filled tooth did not differ significantly in diabetic (31.3%) and control (42.1%) groups, as well as the number of root-filled teeth. In both groups, the prevalence of root canal treatment was low compared with 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 endodontic treatment determined previously in the Spanish population (41%) (Jiménez-Pinzón *et al.* 2004). The results of Britto *et al.* (2003) showed similar features: no association was found between root canal treatment *per se* and diabetic state. Nevertheless, these authors found a very high percentage of root filled teeth both in diabetic (66.7%) and control (91.3%) groups. The reasons for these findings are the same as those stated above, although they can be also attributed to better overall dental care with easier access to the endodontic therapy in the population of the US compared with the Spanish population.

It has been hypothesized that poorly controlled diabetes may also increase the rate of root treatment disease, but this statement is not supported by the results of the present study. The percentages of subjects with at least one root-filled tooth affected with AP did not differ significantly in diabetic (70.0%) and control (62.5%) groups ($P > 0.05$). Taking the tooth as unit, the results were confirmed, although diabetic patients showed a higher percentage of root filled teeth associated with AP (83.3%) than the control subjects (60.0%) ($P > 0.05$). As it has been suggested earlier, the information from the present study confirms that individuals who have root fillings should be considered for further radiographic examination as the radiographic evidence of a root filling was a very crucial risk indicator for AP in the individual (Bergström *et al.* 1987, Kirkevang *et al.* 2001, Jiménez-Pinzón *et al.* 2004).

The results of this study are in agreement with a previous report (Britto *et al.* 2003) that also found 70.0% of diabetic subjects with root filled teeth associated with AP and 71.4% in the control group ($P > 0.05$). On the contrary, in a Swedish subpopulation, female residents with long-duration insulin-dependent diabetes mellitus were found to have an increased prevalence of root filled teeth with periapical lesions than residents with short-duration insulin-dependent diabetes mellitus (Falk *et al.* 1989). Moreover, in an earlier clinical study (Cheraskin & Ringsdorf 1968), radiographic healing of periapical lesions following root canal treatment was closely monitored, showing that in a low glucose group the periapical radiolucencies were reduced by an average of 74% compared with a reduction of only 48% for a high glucose group. Recently, analysing data on nonsurgical endodontic cases, Fouad & Burleson (2003) concluded that patients with diabetes had increased periodontal disease in teeth involved endodontically and had a reduced likelihood of success of root canal treatment, but only in cases with preoperative periradicular lesions. Nevertheless, the same investigator reported that diabetes did not affect the treatment outcome in the total sample (Fouad 2003).

Conclusions

The data reported in the present study, taken together with previous reports in human and the animal studies provide findings that suggest some differences in the natural history of periapical lesions in the diabetic patient and strongly support the concept that diabetes acts as a risk factor for AP, increasing its prevalence and may affect the outcome of the root canal treatment. We conclude that type 2 diabetes mellitus is significantly associated with an increased prevalence of AP.

References

Allard U, Palmqvist S (1986) A radiographic survey of periapical conditions in elderly people in a Swedish country population. *Endodontics and Dental Traumatology* **2**, 103–8.

Bender IB, Bender AB (2003) Diabetes mellitus and the dental pulp. *Journal of Endodontics* **29**, 383–9.

Bender IB, Seltzer S, Freedland J (1963) The relationship of systemic diseases to endodontic failures and treatment procedures. *Oral Surgery, Oral Medicine and Oral Pathology* **16**, 1102–15.

Bergström J, Eliasson S, Ahlberg KF (1987) Periapical status in subjects with regular dental care habits. *Community Dentistry and Oral Epidemiology* **15**, 236–9.

Boucher Y, Matossian L, Rilliard F, Machtou P (2002) Radiographic evaluation of the prevalence and technical quality of root canal treatment in a French subpopulation. *International Endodontic Journal* **35**, 229–38.

Britto LR, Katz J, Guelmann M, Heft M (2003) Periradicular radiographic assessment in diabetic and control individuals. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics* **96**, 449–52.

Cheraskin E, Ringsdorf WM Jr (1968) The biology of the endodontic patient: 3. Variability in periapical healing and blood glucose. *Journal of Oral Medicine* **23**, 87–90.

De Cleen MJ, Schuurs AH, Wesselink PR, Wu MK (1993) Periapical status and prevalence of endodontic treatment in an adult Dutch population. *International Endodontic Journal* **26**, 112–9.

Delamaire M, Maugendre D, Moreno M, Le Goff MC, Allannic H, Genetet B (1997) Impaired leukocyte functions in diabetic patients. *Diabetes Medicine* **14**, 29–34.

Eriksen HM, Berset GP, Hansen BF, Bjertness E (1995) Changes in endodontic status 1973–93 among 35-year-olds in Oslo, Norway. *International Endodontic Journal* **28**, 129–32.

Eriksen HM, Bjertness E, Ørstavik D (1998) Prevalence and quality of endodontic treatment in an urban adult population in Norway. *Endodontics and Dental Traumatology* **4**, 122–6.

Falk H, Hugoson A, Thorstensson H (1989) Number of teeth, prevalence of caries and periapical lesions in insulin-dependent diabetics. *Scandinavian Journal of Dental Research* **97**, 198–206.

Figdor D (2002) Apical periodontitis: a very prevalent problem. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics* **94**, 651–2.

Fouad AF (2003) Diabetes mellitus as a modulating factor of endodontic infections. *Journal of Dental Education* **67**, 459–67.

Fouad AF, Burleson J (2003) The effect of diabetes mellitus on endodontic treatment outcome: data from and electronic patient record. *Journal of the American Dental Association* **134**, 43–51.

Fouad A, Barry J, Russo J, Radolf J, Zhu Q (2002) Periapical lesion progression with controlled microbial inoculation in a type 1 diabetic mouse model. *Journal of Endodontics* **28**, 8–16.

Imfeld TN (1991) Prevalence and quality of endodontic treatment in an elderly urban population of Switzerland. *Journal of Endodontics* **17**, 604–7.

Iwama A, Nishigaki N, Nakamura K *et al.* (2003) The effect of high sugar intake on the development of periradicular lesions in rats with type 2 diabetes. *Journal of Dental Research* **82**, 322–5.

Jiménez-Pinzón A, Segura-Egea JJ, Poyato-Ferrera M, Velasco-Ortega E, Ríos-Santos JV (2004) Prevalence of apical periodontitis and frequency of root filled teeth in an adult Spanish population. *International Endodontic Journal* **37**, 167–73.

Katz J (2001) Elevated blood glucose levels in patients with severe periodontal disease. *Journal of Clinical Periodontology* **28**, 710–2.

Kirkevang LL, Wenzel A (2003) Risk indicators for apical periodontitis. *Community Dentistry and Oral Epidemiology* **31**, 59–67.

Kirkevang LL, Ørstavik D, Hörsted-Bindslev P, Wenzel A (2000) Periapical status and quality of root fillings and coronal restorations in a Danish population. *International Endodontic Journal* **33**, 509–15.

Kirkevang LL, Hörsted-Bindslev P, Ørstavik D, Wenzel A (2001) Frequency and distribution of endodontically treated teeth and apical periodontitis in an urban Danish population. *International Endodontic Journal* **34**, 198–205.

Kohsaka T, Kumazawa M, Yamasaki M, Nakamura H (1996) Periapical lesions in rats with streptozotocin-induced diabetes. *Journal of Endodontics* **22**, 418–21.

Lagervall M, Jansson L, Bergstrom J (2003) Systemic disorders in patients with periodontal disease. *Journal of Clinical Periodontology* **30**, 293–9.

Little JW, Falace DA, Miller CS, Rhodus NL (1997) Diabetes. In: James W. Little, ed. *Dental Management of the Medically Compromised Patient*, 5th edn. St Louis, MO: Mosby, pp. 387–409.

Lupi-Pegurier L, Bartrand M-F, Muller-Bolla M, Rocca JP, Bolla M (2002) Periapical status, prevalence and quality of endodontic treatment in an adult French population. *International Endodontic Journal* **35**, 690–7.

Marques MD, Moreira B, Eriksen HM (1998) Prevalence of apical periodontitis and results of endodontic treatment in an adult, Portuguese population. *International Endodontic Journal* **31**, 161–5.

Ørstavik D, Kerekes K, Eriksen HM (1986) The periapical index: a scoring system for radiographic assessment of apical periodontitis. *Endodontics and Dental Traumatology* **2**, 20–34.

Segura-Egea JJ, Jiménez-Pinzón A, Poyato-Ferrera M, Velasco-Ortega E, Ríos-Santos JV (2004) Periapical status and quality of root fillings and coronal restorations in an adult Spanish population. *International Endodontic Journal* **37**, 525–30.

Sidaravicius B, Aleksejuniene J, Eriksen HM (1999) Endodontic treatment and prevalence of apical periodontitis in an adult population of Vilnius, Lithuania. *Endodontics and Dental Traumatology* **15**, 210–5.

Soskolne WA, Klinger A (2001) The relationship between periodontal diseases and diabetes: an overwiew. *Annals of Peridontology* **91**, 263–70.

Thomson WM, Slade GD, Beck JD, Elter JR, Spencer AJ, Chalmers JM (2004) Incidence of periodontal attachment loss over 5 years among older South Australians. *Journal of Clinical Periodontology* **31**, 119–25.

Ueta E, Osaki T, Yoneda K, Yamamoto T (1993) Prevalence of diabetes mellitus in odontogenic infections and oral candidiasis: an analysis of neutrophil suppression. *Journal of Oral Pathology and Medicine* **22**, 1768–74.

Vernillo AT (2003) Diabetes mellitus: relevance to dental treatment. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics* **91**, 263–70.