

Root surface and coronal caries in adults with type 2 diabetes mellitus

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Abstract – Objectives: To determine the effect of type 2 diabetes mellitus (DM) on coronal and root surface caries and to investigate some factors suspected of being related to or interacting with DM, that may be associated with coronal and root surface caries. Methods: A stratified cross-sectional study was conducted in 105 type 2 diabetic patients and 103 non-diabetic subjects of the same age and gender. Coronal and root surface caries, exposed root surfaces, periodontal status, stimulated salivary functions, oral hygiene status, oral health behaviors, and counts of mutans streptococci and lactobacilli were measured. Results: Type 2 diabetic patients compared with non-diabetic subjects had a higher prevalence of root surface caries (40.0% versus 18.5%; P = 0.001), a higher number of decayed/filled root surfaces (1.2 ± 0.2 versus 0.5 ± 0.1 ; P < 0.01) and a higher percentage of generalized periodontitis (98.1%) versus 87.4%; P < 0.01); but the prevalence and decayed/filled surface of coronal caries was not significantly different (83.8% versus 72.8% and 8.0 ± 9.4 versus 6.3 ± 7.5 respectively). The factors associated with root surface caries included type 2 DM, a low saliva buffer capacity, more missing teeth, and existing coronal caries; whereas wearing removable dentures, more missing teeth, a high number of lactobacilli, and a low saliva buffer capacity were associated with coronal caries. Conclusion: Type 2 DM is a significant risk factor for root surface, but not for coronal caries. Periodontal disease should be treated early in type 2 diabetic subjects to reduce the risk of subsequent root surface caries.

Diabetes mellitus (DM) is a group of metabolic diseases characterized by hyperglycemia. There are two types of DM. Type 1 develops most frequently in children and adolescents and accounts for 10% of all cases of DM. Type 2, the more common form of DM, is more prevalent among older adults. The World Health Organization (WHO) predicts that between 1995 and 2025, the number of adults with DM will more than double worldwide; from 135 to 300 million. In this period, developing countries will see more than a threefold increase, from 84 to 288 million (1). Due to its systemic nature, DM often leads to impaired general health, including oral health. Both types of DM have been shown to be

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associated with an increased risk of periodontal diseases (2). However, the effects of DM on dental caries are less well known and the study results are contradictory (3–10). The prevalence of dental caries in type 1 or type 2 diabetic subjects compared with normal controls has been reported to be higher, lower, and similar, but some studies did not classify subjects with respect to the DM type or type of caries (6–8).

The relationship between type 2 DM and dental caries is complex. Both have increased prevalence with age. The impact of type 2 DM on caries development may differ between teeth and sites (coronal or root surface). Most of the previous studies have ignored this potential difference by reporting the total caries experience as a whole without specifying the site (6–8, 11). Others have not documented any evidence of the type 2 DM effect on either coronal or root surface caries (3–5). However, all these studies had a rather small sample size and gave no information on concurrent periodontal disease being a known risk factor for root surface caries (3–5). Other potential associated factors for root surface caries such as oral hygiene, oral health behavior, microbial, and salivary factors have not yet been thoroughly examined.

As the prevalence of dental caries, periodontal diseases, and type 2 DM is increasing in Thailand (12–14), this study was conducted to (i) determine the effect of type 2 DM on both coronal and root surface caries and (ii) investigate some factors suspected of being related to, or interacting with, DM that may be associated with coronal and root surface caries.

Material and methods

Study population

This stratified cross-sectional study conducted in southern Thailand was approved by the Ethics Committee of Prince of Songkla University. To test the hypothesis concerning the equality of proportions of root surface caries in people with type 2 diabetes and non-diabetic people, with $\propto = 0.05$ and 80% power, the required sample size was 105 subjects for each group. All participants in this study fulfilled the following inclusion criteria: (a) older than 34 years, (b) to present at least six teeth with crowns, (c) no severe mental or systemic disorder or pregnancy, (d) no sign or symptom of AIDS, and (e) no antibiotic administration during the last 6 months. Informed consent was obtained before oral examination was performed.

Type 2 diabetic subjects were recruited from the endocrine clinic of Songkhlanagarind Hospital. The prospective list of diabetic patients at the endocrine clinic was examined. On the day of appointment, subjects were approached and screened for eligibility criteria. Non-diabetic subjects were selected from the general practice clinic of the same hospital on the day for regular health check-ups. Nondiabetic subjects had no known history of DM and had a fasting plasma glucose level <110 mg/dl. Selection of the non-diabetic controls was balanced by age–sex stratification with type 2 diabetic individuals.

Clinical examination

Fasting venous blood was collected for the measurement of plasma glucose levels in both groups by an automated analyzer (Hitachi 917; Roche, Mannheim, Germany) using the Hexokinase method (Roche) and glycosylated hemoglobin (HbA1c) was examined in the type 2 diabetic group using the turbidimetric inhibition immunoassay (Hitachi 717; Roche).

After breakfast, the subjects were interviewed using a structured questionnaire for socioeconomic data and behaviors related to oral health such as smoking habit, use of fluoride toothpaste, frequency of tooth brushing, sugar intake and dental visits within 12 months. Diabetes-related variables were extracted from medical records; including diet or oral anti-diabetic and insulin use, duration of the disease in years, and presence of any complications.

After the interview and medical record review, paraffin-stimulated whole saliva was collected into a beaker for 5 min. The saliva secretion rate was calculated as ml/min. The stimulated saliva pH was measured using pH indicator strips (Macherey-Nagel, Düren, Germany). The stimulated saliva buffer capacity was obtained by mixing 1 ml of saliva with 3 ml of 0.005 N HCL and shaking. The sample was left to stand for 10 min and the final pH was then measured with pH indicator strips. The number of mutans streptococci colonies and lactobacilli colonies was examined using the spatula method described by Kohler and Bratthall (15).

Oral examination of all teeth (except third molars) using a plain mouth mirror and standard probe was performed without radiograph assessment. Clinical examinations were performed by one examiner (J.H.) throughout the study. Intraexaminer (1 week apart) kappa statistics for coronal caries, root surface caries, and bleeding on probing were 0.93, 0.87, and 0.79, respectively, while the weight kappa for probing depth, attachment level, plaque index, and calculus index were 0.87, 0.78, 0.89, and 0.81, respectively. The presence and type of prosthesis (removable and non-removable dentures) were recorded. The Simplified Oral Hygiene Index (OHI-S), consisting of plaque and calculus indices, was employed (16). Buccal surfaces of teeth 16, 11, 26, and 31, and the lingual surfaces of teeth 36 and 46 were examined. The mean score of each index was calculated for each subject. The average individual plaque and calculus scores were combined to obtain OHI-S. Bleeding on probing, probing depth, and attachment levels were assessed on six surfaces of each tooth,

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including the mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and distolingual surfaces. The probe was inserted into the bottom of the pocket and gently moved laterally along the pocket wall. Bleeding on probing was measured as present or absent. The probing depth was defined as the distance between the bottom of the pocket and the gingival margin, and the attachment level was defined as the distance between the bottom of the pocket and the cemento-enamel junction (CEJ). Gingival recession was the distance from CEJ to the gingival margin. Coronal caries were assessed using the simplified oral health record form of the WHO (17). Root surface caries were examined according to the criteria of Katz (18). Occlusal, buccal, lingual, mesial, and distal surfaces of each tooth were examined for coronal caries while root surface caries were recorded for all mesial, distal, buccal, and lingual surfaces. Root surfaces with gingival levels below the CEJ were defined as exposed.

Statistical analysis

Data were entered twice to verify accuracy of the entry using EpiData version 2.1 (The EpiData Association, Odense, Denmark). Data was transferred to Stata version 7.0 (Stata Corporation, College Station, TX, USA) for statistical analysis. Each individual's periodontal condition was evaluated in terms of severity and extent. Severity was described for the entire individual site and was categorized on the basis of clinical attachment loss (CAL) as follows: slight = 1–2 mm CAL, moderate = 3–4 CAL, and severe = ≥ 5 mm CAL. Extent was characterized as localized if $\leq 30\%$ of the sites were affected and generalized if $\geq 30\%$ of the sites were affected (19).

The analysis was based on remaining teeth and the third molars were excluded. Thus, the decayed and filled surfaces (DFS) or teeth (DFT) were analysed for both coronal or root surface caries experience. The mean numbers of surfaces, teeth with caries experience, and exposed roots were calculated. The prevalence of root surface and coronal caries was assessed.

Differences in the prevalence rates of dental caries or the periodontal condition of type 2 diabetic and non-diabetic groups were compared using the chi-squared test. When comparing the median number of dental caries between groups the Mann–Whitney test was employed. As teeth were measured for the presence of coronal or root surface caries nested within subjects, multilevel logistic model, GLMMPQL in R-package (20) was used. Models of the presence of root surface caries and coronal caries were separately developed. Subject's characteristics and possible interactions between the diabetic status and other characteristics were independent variables. The final model was chosen based on the backward elimination process, starting with the full independent variables, followed by subsequent removing of nonsignificant individual independent variables until no other nonsignificant independent variable could be removed. To break down the effect of type 2 DM, first, multilevel linear modeling of the gingival recession in millimeters was developed by using LME in the R-package to check the effect of type 2 DM on exposed root surfaces. In this model, level 1 was the sites, level 2 was the teeth, and level 3 was the subjects. Finally, to examine the effect of type 2 DM on enhancing root surface caries, the data set was confined to teeth with root surface already exposed and the outcome was root surface caries.

Results

One-hundred and five (90% response rate) type 2 diabetic and 103 (70% response rate) non-diabetic subjects consented to the study. The main reason for refusal was not having enough time for the examination. The ratio of type 2 diabetic and non-diabetic subjects was well balanced in all age and sex groups. The type 2 diabetic group had a higher probability of wearing removable dentures, higher plaque index, calculus index, and counts of mutans streptococci and lactobacilli than the non-diabetic group. Saliva flow rate, pH, and buffer capacity were lower in the type 2 diabetic group than in the non-diabetic group (Table 1).

Regarding the oral health outcome, deeper periodontal pockets, more attachment loss, more bleeding on probing and a higher prevalence of generalized chronic periodontitis and severe periodontitis were significantly more common in type 2 diabetic subjects than in non-diabetic subjects (Table 2).

For caries assessment, type 2 diabetic subjects had significantly less remaining teeth, had more exposed root surfaces, and had a higher mean and prevalence of root surface caries than non-diabetic subjects. However, the difference in prevalence and mean of coronal caries was not statistically significant (Table 3).

Characteristics	Type 2 DM ($n = 105$)	Non-DM ($n = 103$)
Age (years)	54.3 ± 8.7	53.3 ± 7.6
Sex (females)	50.5	50.5
Duration of DM (years)	8.7 ± 5.7	_
Poor diabetic control (HbA1c >8.5%)	47.6	_
Diabetic complication	42.9	_
Anti-diabetic treatment (insulin)	15.2	_
Current or ex-smoker ^a	33.3	24.3
Removable denture wearer ^a	32.4	17.5
Use of fluoride toothpaste ^a	75.2	74.8
Tooth brushing at least twice a day	83.8	90.3
Dental visit within 12 months	40.0	49.5
Sugar intake at least once a day	39.1	52.4
Saliva secretion rate (ml/min)	0.8 ± 0.5	1.0 ± 0.6
Saliva pH ^b	7.6 ± 0.5	7.8 ± 0.4
Saliva buffer capacity	6.5 ± 0.5	6.6 ± 0.5
Mutans streptococci >10 ⁵ CFU/ml of saliva	21.0	16.5
Lactobacilli >10 ⁴ CFU/ml of saliva	24.8	15.5
Simplified Oral Hygiene Index ^b	3.5 ± 1.41	3.0 ± 1.4
Plaque Index ^b	1.4 ± 0.1	1.2 ± 0.1
Calculus Index	2.0 ± 0.1	1.8 ± 0.1

Table 1. The distribution of characteristics (percentage or mean ± SD) of type 2 diabetic and non-diabetic subjects

DM, diabetes mellitus; CFU, colony-forming units.

Statistical significance (P < 0.05) by ^achi-squared test and ^bStudent's *t*-test.

Table 2. The percentage of sites with different levels of periodontal parameters among type 2 diabetic and non-diabetic subjects

Parameters	Type 2 DM	Non-DM	<i>P</i> -value ^a
Teeth sites	$(n = 13\ 288)$	(n = 14502)	
Probing depth (mm)			
≤2	67.97	76.38	
3–4	26.41	19.92	
≥5	6.62	3.70	< 0.001
Attachment level (mi	m)		
0	13.94	34.65	
1–2	42.24	36.29	
3–4	28.12	20.36	< 0.001
≥5	15.70	8.71	
Bleeding on probing	34.84	27.89	< 0.001
Individuals	(n = 105)	(n = 103)	
Generalized	98.1	87.4	< 0.01
periodontitis (%)			
Severe	84.8	72.8	< 0.05
periodontitis (%)			

DM, diabetes mellitus.

^aChi-squared test.

There was no association between the two types of caries and the diabetes-related variables including duration of DM, poor diabetic control (HbA1c >8.5%), diabetic complications, and insulin treatment.

Multilevel logistic models were separately computed for risk factors of coronal and root surface caries with the same set of independent variables (Table 4). After adjustment for all independent variables, teeth of type 2 diabetic subjects were independently at greater risk for root surface caries but not for coronal caries than those of non-diabetic subjects. For determining root surface caries, the effects of type 2 DM and other independent variables were not changed after the presence of coronal caries was removed.

After adjustment for smoking, wearing removable dentures, age, and number of missing teeth, type 2 DM was associated with gingival recession (Table 5). Among the teeth with exposed root surface, type 2 DM was significantly associated with root surface caries (odds ratio = 1.82) after adjustment for the same set of independent variables listed in Table 4.

Discussion

This study demonstrated that type 2 diabetic patients when compared with non-diabetic subjects had a lower saliva pH but a higher percentage of current or ex-smokers, a higher frequency of denture wearing, a higher plaque index, a higher simplified oral hygiene index, and more severe periodontitis. In this population, type 2 DM showed evidence of increased exposure of the root surface by gingival recession. Apart from type 2 DM, low saliva buffer capacity, high number of

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Parameters	Type 2 DM ($n = 105$)	Non-DM ($n = 103$)	<i>P</i> -value
Teeth present ^a	21.7 ± 0.5	23.9 ± 0.5	< 0.01
Teeth with exposed root ^a	11.3 ± 0.6	10.0 ± 0.6	0.15
Exposed root surfaces ^a	26.0 ± 1.8	19.4 ± 1.6	< 0.01
Root surface caries (teeth) ^a	1.0 ± 0.2	0.4 ± 0.1	< 0.001
Root surface caries (surfaces) ^a	1.2 ± 0.2	0.5 ± 0.1	< 0.01
Prevalence (%) of root surface caries ^b	40.0	18.5	0.001
Coronal caries (teeth) ^a	3.8 ± 0.2	3.3 ± 0.3	0.25
Coronal caries (surfaces) ^a	8.0 ± 9.4	6.3 ± 7.5	0.09
Prevalence (%) of coronal caries ^b	83.8	72.8	0.06

Table 3. The mean number ± SD and prevalence of root surface and coronal caries among type 2 diabetic and nondiabetic subjects

DM, diabetes mellitus.

^aMann–Whitney statistics.

^bChi-squared test.

Table 4. The associations between the independent variables and coronal or root surface caries analyzed by multilevel logistic model that reach a statistical significance for coronal and/or root surface caries

	Coronal caries	Coronal caries		Root surface caries	
Independent variables	OR (95% CI)	<i>P</i> -value	OR (95% CI)	<i>P</i> -value	
Type 2 DM ¹	1.08 (0.98-1.50)	0.64	2.12 (1.08-4.14)	< 0.05	
Current or ex-smoker ²	0.44 (0.30-0.64)	< 0.001	1.43 (0.69–2.94)	0.38	
Removable dentures ³	1.59 (1.03-2.43)	< 0.05	1.11 (0.50-2.46)	0.80	
Lactobacilli >10 ⁴ CFU/ml of saliva ⁴	1.51 (1.02-2.25)	< 0.05	1.47 (0.69–3.12)	0.32	
Age ^a	0.98 (0.96-0.99)	< 0.05	1.02 (0.98-1.07)	0.24	
Missing teeth ^a	1.09 (1.04–1.13)	< 0.001	1.24 (1.16–1.33)	< 0.001	
Saliva buffer capacity ^a	0.71 (0.51-0.98)	< 0.05	0.44 (0.24–0.81)	< 0.01	
≥ 1 coronal caries lesion ⁵	_	_	2.96 (1.07-8.16)	< 0.05	

Reference level: $1 = \text{non-DM}, 2 = \text{non-smoker}, 3 = \text{no removable denture}, 4 = \text{lactobacilli} \le 10^4 \text{ CFU/ml of saliva}, 5 = \text{no coronal caries}.$

OR, odds ratio; CI, confidence interval.

^aContinuous variable.

Table 5. The associations between the independent variables and gingival recession analyzed by multilevel linear model

Independent variables	Coefficient	Standard error	<i>P</i> -value
Type 2 DM ¹ Current or ex-smoker ²	0.42 0.27	0.40 0.13	<0.001 <0.05
Removable dentures ³	0.04	0.16	0.80
Age ^a	0.03	0.01	< 0.01
Missing teeth ^a	0.12	0.01	< 0.001
Constant	-1.98	0.39	

Reference level: 1 = non-DM, 2 = non-smoker, 3 = no removable denture.

^aContinuous variable.

missing teeth, and existing coronal caries were risk factors for root surface caries. The significant risk factors for coronal caries included removable dentures, a high level of lactobacilli, low saliva buffer capacity, and high number of missing teeth.

Our two groups of subjects shared similar characteristics in relation to most caries risk factors except for a higher frequency of wearing removable dentures and poorer oral hygiene in type 2 diabetic subjects. Greater loss of teeth in type 2 diabetic subjects possibly leads to an increase in the wearing of dentures and subsequently poorer oral hygiene. In addition, a higher prevalence and severity of periodontitis may be a result of poorer oral hygiene care. The pH of saliva in type 2 diabetic subjects was significantly lower than that of non-diabetic subjects, although the range in both groups was within normal limits. No significant difference has been found in any of the previous studies (4, 5). This finding together with the low statistical significance in this study indicates that the pH of saliva is of little or no clinical importance.

A more severe periodontitis among the type 2 DM patients was found and confirmed in a previous report (2). The exact reason behind this association has not been clearly explained; however, alteration in host defenses, vascular changes, change in the oral microflora, and abnormal collagen metabolism are suggested to be major factors (2).

Although type 2 diabetic subjects had fewer remaining teeth, the number of teeth and surfaces with exposed root were higher than that observed in the non-diabetic group. On the other hand, given the more missing teeth in this group, the number of teeth with root surface caries presented during the survey may underestimate the size of the problem.

Previous studies with a small sample size (42–65 subjects per study) reported no difference in the caries rate, both root surface and coronal caries, between type 2 diabetic and non-diabetic subjects (3–5). With a larger sample size as in our study (208 subjects), a higher risk for root surface caries, but not coronal caries, can be demonstrated among type 2 diabetic subjects. Aside from having a large sample size, poorer oral hygiene in our type 2 diabetic subjects may in part facilitate the caries process once the root surface is exposed.

The causal relationship between type 2 DM and the two caries-prone sites (coronal and root surface) can be discussed from two aspects. First, our study confirmed more severe periodontitis in type 2 diabetic than in non-diabetic subjects leading to an increased prevalence of exposure of the tooth roots by gingival recession. There was evidence that exposed root surfaces were more vulnerable to demineralization than enamel (21), suggesting a higher incidence of root surface caries in type 2 diabetic subjects. Secondly, our data demonstrated that once roots of the teeth were exposed to the oral environment, type 2 DM was a risk factor for those roots in developing root surface caries. The higher incidence of root surface caries in type 2 diabetic subjects may be related to a higher glucose level in secretory fluids, e.g., saliva and gingival crevicular fluid (GCF) stimulating the root surface plaque to an increased saccharolytic activity and acid production. Higher glucose levels in saliva have been found in type 1 diabetic patients compared with non-diabetic subjects (22, 23). In the present study, salivary glucose levels of 19 subjects were found to be correlated with glucose in blood (data not shown). A high glucose level in saliva could favor an increase of aciduric and acidogenic bacteria, such as mutans streptococci and lactobacilli associated with root surface caries and coronal caries. The glucose content of GCF, a biologic fluid derived from serum, directly correlates with the glucose concentration in serum, although in comparatively smaller amounts than saliva. On the other hand, any immediate contact with bacteria in the periodontal pocket and the crevicular orifice may increase the glucose fermenting activity of the root surface plaque bacteria and acid production, subsequently leading to root surface caries.

High counts of lactobacilli in saliva were associated with coronal caries but not root surface caries. This discrepancy may be due to the fact that the cavities of coronal caries in our samples were more developed and the salivary content of lactobacilli is more reflective of coronal caries than root surface caries.

Salivary buffer capacity is important in maintaining a pH level in saliva and plaque which counteracts dissolution of minerals of the teeth. An inverse relationship between buffer capacity and both types of caries was confirmed in the present study as well as the previous studies (24– 26).

The association between coronal and root surface caries at the subject level has been previously reported (27). In the present study, a significant association was confirmed. Teeth with coronal caries may be brushed less thoroughly than noncarious teeth because of pain. This can lead to poor local hygiene and subsequently root surface caries.

Smoking is known to be a strong risk factor for periodontitis (28). However, the expected association with root surface caries could not be confirmed by our data. On the contrary, current and ex-smoking was significantly negatively associated with coronal caries. This is in contrast to studies that showed that smoking is a risk factor for caries (29-31). These reports suggested that other factors related to smoking, such as behavioral quality and decreased salivary flow rate, may have a confounding effect on smokers' risk for caries. The temporal sequence of smoking and dental caries that could not be evaluated because of the crosssectional nature of the study might be an explanation for the unexpected finding in the present study.

Older age in our subjects was associated with a decreased risk for coronal caries. This is in contrast to the expectation that the risk would accumulate and increase over time. However, the difference can also be explained by a cohort effect. For the past few decades, age-adjusted caries rate in Thailand has tended to increase over time (32), particularly as a result of an increase in the consumption of sugars. The older subjects in the

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present study may subsequently have a lower risk for caries than the younger subjects, leading to a negative association between age and coronal caries.

Periodontal disease and root surface caries prevention should be emphasized for type 2 diabetic subjects. Patients wearing removable dentures and having a high number of missing teeth need to be educated on the benefits of denture care and a regular dental check-up. The precise mechanism of type 2 DM on root surface caries and the role of GCF on dental plaque microorganisms need further investigation.

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References

- 1. World Health Organization. Global burden of diabetes [WWW document]. URL http://www.who.int/ inf-pr-1998/en/pr98–63.html (last accessed 30 April, 2006).
- 2. Diabetes and periodontal diseases. Committee on Research, Science and Therapy. American Academy of Periodontology. J Periodontol 2000;71:664–78.
- 3. Lin BP, Taylor GW, Allen DJ, Ship JA. Dental caries in older adults with diabetes mellitus. Spec Care Dentist 1999;19:8–14.
- Collin HL, Uusitupa M, Niskanen L, Koivisto AM, Markkanen H, Meurman JH. Caries in patients with non-insulin-dependent diabetes mellitus. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85:680–5.
- Narhi TO, Meurman JH, Odont D, Ainamo A, Tilvis R. Oral health in the elderly with non-insulindependent diabetes mellitus. Spec Care Dentist 1996;16:116–22.
- Zielinski MB, Fedele D, Forman LJ, Pomerantz SC. Oral health in the elderly with non-insulin-dependent diabetes mellitus. Spec Care Dentist 2002;22:94– 8.
- 7. Sandberg GE, Sundberg HE, Fjellstrom CA, Wikblad KF. Type 2 diabetes and oral health: a comparison

between diabetic and non-diabetic subjects. Diabetes Res Clin Pract 2000;50:27–34.

- 8. Bacic M, Ciglar I, Granic M, Plancak D, Sutalo J. Dental status in a group of adult diabetic patients. Community Dent Oral Epidemiol 1989;17:313–6.
- 9. Moore PA, Weyant RJ, Etzel KR, Guggenheimer J, Mongelluzzo MB, Myers DE et al. Type 1 diabetes mellitus and oral health: assessment of coronal and root caries. Community Dent Oral Epidemiol 2001;29:183–94.
- Tavares M, Depaola P, Soparkar P, Joshipura K. The prevalence of root caries in a diabetic population. J Dent Res 1991;70:979–83.
- 11. Arrieta-Blanco JJ, Bartolome-Villar B, Jimenez-Martinez E, Saavedra-Vallejo P, Arrieta-Blanco FJ. Buccodental problems in patients with diabetes mellitus (I): index of plaque and dental caries. Med Oral 2003;8:97–109.
- 12. Likitmaskul S, Kiattisathavee P, Chaichanwatanakul K, Punnakanta L, Angsusingha K, Tuchinda C. Increasing prevalence of type 2 diabetes mellitus in Thai children and adolescents associated with increasing prevalence of obesity. J Pediatr Endocrinol Metab 2003;16:71–7.
- 13. Baelum V, Pisuithanakan S, Teanpaisan R, Pithpornchaiyakul W, Pongpaisal S, Papapanou PN et al. Periodontal conditions among adults in southern Thailand. J Periodontal Res 2003;38:156–63.
- 14. Baelum V, Pongpaisal S, Pithpornchaiyakul W, Pisuithanakan S, Teanpaisan R, Papapanou PN et al. Determinants of dental status and caries among adults in southern Thailand. Acta Odontol Scand 2002;60:80–6.
- 15. Kohler B, Bratthall D. Practical method to facilitate estimation of *Streptococcus mutans* levels in saliva. J Clin Microbiol 1979;9:584–8.
- 16. Greene JC, Vermillion JR. The Simplified Oral Hygiene Index. J Am Dent Assoc 1964;68:7–13.
- 17. World Health Organization. Oral health surveys: basic methods. Geneva: World Health Organization; 1997.
- 18. Katz RV. Clinical signs of root caries: measurement issues from an epidemiologic perspective. J Dent Res 1990;69:1211–5.
- 19. Flemmig TF. Periodontitis. Ann Periodontol 1999;4:32–8.
- 20. Pinheiro J, Bates D, DebRoy S, Sarkar D. NLME: linear and nonlinear mixed effects models. R package version 31-53 2004 [WWW document]. http:// www.stats.bris.ac.uk/R/src/contrib/2.0.1-patched/ Recommended/nlme_3.1-5.6.tar.gz (last accessed 30 April, 2006).
- 21. Hoppenbrouwers PM, Driessens FC, Borggreven JM. The vulnerability of unexposed human dental roots to demineralization. J Dent Res 1986;65:955–8.
- 22. Karjalainen KM, Knuuttila ML, Kaar ML. Salivary factors in children and adolescents with insulindependent diabetes mellitus. Pediatr Dent 1996;18:306–11.
- 23. Tenovuo J, Alanen P, Larjava H, Viikari J, Lehtonen OP. Oral health of patients with insulin-dependent diabetes mellitus. Scand J Dent Res 1986;94: 338–46.
- 24. Guivante-Nabet C, Berenholc C, Berdal A. Caries activity and associated risk factors in elderly

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hospitalised population–15-months follow-up in French institutions. Gerodontology 1999;16:47–58.

- 25. Guivante-Nabet C, Tavernier JC, Trevoux M, Berenholc C, Berdal A. Active and inactive caries lesions in a selected elderly institutionalised French population. Int Dent J 1998;48:111–22.
- 26. Fure S, Zickert I. Root surface caries and associated factors. Scand J Dent Res 1990;98:391–400.
- Beck JD, Drake CW. Do root lesions tend to develop in the same people who develop coronal lesions? J Public Health Dent 1997;57:82–8.
- Beck JD, Koch GG, Offenbacher S. Incidence of attachment loss over 3 years in older adults – new and progressing lesions. Community Dent Oral Epidemiol 1995;23:291–6.
- 29. Axelsson P, Paulander J, Lindhe J. Relationship between smoking and dental status in 35-, 50-, 65-, and 75-year-old individuals. J Clin Periodontol 1998;25:297–305.
- Fure S. Ten-year cross-sectional and incidence study of coronal and root caries and some related factors in elderly Swedish individuals. Gerodontology 2004;21:130–40.
- 31. Drake CW, Beck JD, Lawrence HP, Koch GG. Threeyear coronal caries incidence and risk factors in North Carolina elderly. Caries Res 1997;31:1–7.
- 32. The 5th Thailand National Oral Health Survey, 200– 01 [WWW document]. URL http://www.anamai. moph.go.th/fluoride/survey/frame.html (last accessed 30 April, 2006).

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