Copyright © Blackwell Munksgaard Ltd JOURNAL OF PERIODONTAL RESEARCH

doi:10.1111/j.1600-0765.2006.00862.x

# Type 2 diabetes and periodontal indicators: epidemiology in France 2002–2003

Mattout C, Bourgeois D, Bouchard P. Type 2 diabetes and periodontal indicators: epidemiology in France 2002–2003. J Periodont Res 2006; 41: 253–258. © Blackwell Munksgaard 2006

*Background and Objective:* Diabetes and periodontal disease have been associated in the literature. In the present study, the periodontal heath of noninsulindependent diabetic adults was compared with that of a general population of nondiabetic patients.

*Material and Methods:* In France, 2144 adults (age: 35–65 years) were examined for life habits (tobacco, alcohol), biological diagnosis (type II diabetes, arterial hypertension), biometry (weight, size) and biochemistry. Dental and periodontal data included plaque index, gingival index, probing depth, and clinical attachment loss.

*Results:* Descriptive and multifactorial analysis evidenced a more severe periodontal disease in diabetic patients. Moreover, when the plaque index was held constant, the gingival index was more elevated in diabetics. In nondiabetics, age, gender, glycemia, alcohol, and tobacco smoking were identified as significant risk factors for periodontal disease. In contrast, in diabetic subjects, only tobacco smoking was a significant risk factor.

*Conclusion:* In type II diabetics, the diabetes factor is probably more significant than periodontal risk factors, age, and gender.

C. Mattout<sup>1</sup>, D. Bourgeois<sup>2,3</sup>,

# P. Bouchard<sup>4</sup>

<sup>1</sup>Private Periodontal Practice, Marseille, France, <sup>2</sup>Department of Public Health, University of Lyon, France, <sup>3</sup>WHO Global Oral Health Program, Department for Chronic Disease and Health Promotion, World Health Organization, Geneva, Switzerland, <sup>4</sup>Department of Periodontology, University of Paris, France

Catherine Mattout, 224, Avenue du Prado, 13008 Marseille, France Tel: 04 91 32 2832 Fax: 04 91 32 2833 email: contact@gepi-mattout.com

Key words: diabetes; epidemiology; gingival index; periodontal diseases

Accepted for publication October 19, 2005

The association between diabetes and periodontal disease has been reported for more than 40 years (1). Clinical studies have demonstrated a higher prevalence of periodontitis in diabetic patients (2–4); however, very often type I and type II diabetes were not distinguished. Type II diabetes (formerly classified as noninsulin-dependent) is the most prevalent type of diabetes and affects  $\approx 90\%$  of diabetic patients. The number of invidivuals with type II diabetes is increasing as a result of poor eating habits, obesity, and a sedentary way of life in populations with increased life expectancy. Associated complications include microvascular alteration, which may directly affect the periodontium and the host response (5). The prevalence of periodontal disease has been reported as 60% in subjects with type II diabetes and as 36% in patients without diabetes (6).

In a large survey in the USA (NHANES III; 1988–1994), type II diabetes was found to be associated with a higher prevalence of periodontal disease. The extent of disease in the two groups were 11.4% and 5.8% of

teeth with attachment loss, and 4.9% and 1.6% of teeth with pockets (7). The aim of the present study was to compare the periodontal health of adults with type II diabetes to a general population of nondiabetic patients.

## Material and methods

The present study was a part of the First National Periodontal and Systemic Examination Survey (NPASES) in France. The data were obtained from a 2002–2003 national study that was performed to identify strategies for the prevention and control of non-transmissible diseases (8).

The NPASES was performed by two national institutions: (i) the Union Française pour la Santé Bucco-dentaire (UFSBD), a World Health Organization Collaborating Center with expertise in oral health epidemiology, and (ii) the National Health Insurance Welfare, which conducts periodic health evaluation for the adult population. The population examined comprised 2144 adults (age: 35-65 years) living in France from June 2002 to September 2003. The sampling technique used was the method of quotas with a stratification of four degrees: age 35-39, 40-49, 50-59 or 60-65 years; gender; socioprofessional category (executive profession, intermediate profession, office worker, manual worker, retired, unemployed); and geographical region (Fig. 1).

Information on life habits (tobacco or alcohol use), biological diagnosis (type II diabetes, where diabetes was defined as the current use of antidiabetic drugs; fasting glycemia of > 7.0 mm d/l; and arterial hypertension), biometry (weight and size), and biochemistry (glycemia and cholesterol) were obtained. Tobacco use was categorized as never smoker, former smoker, or current smoker. Alcohol was categorized as never, past, or actual. For biometry, body mass index (BMI) was categorized [according to the formula: weight (in kg)  $\div$  height<sup>2</sup> (in m)] as underweight (BMI < 18.5), normal weight (BMI 18.5-24.9), overweight (BMI 25-29), or obese (BMI ≥30) (9).

Number of healthy, decayed, restored or extracted teeth were determined, and the plaque index (PI) (10), gingival index (GI) (11), probing depth, and clinical attachment loss were recorded. The third molars were excluded (12). The periodontal measurements were assessed at four sites per tooth (mesiobuccal, distobuccal, midbuccal, and midlingual) using a sterile PDT periodontal sensor probe type US (Williams, Professional Dental Technologies Inc., Batesville, AR, USA) and 20 g pressure. Examiners were trained and calibrated (13).

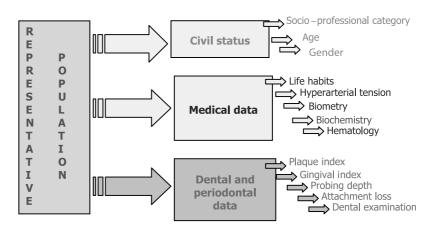


Fig. 1. Study design and recorded data.

The statistical analysis was descriptive (mean, standard deviation, quartile) and analytical. The qualitative and continuous variables were analyzed using chi-square and Student *t*-tests, respectively. A multifactorial analysis tested the relationship between the periodontal disease and type II diabetes.

#### Results

The recorded medical data are summarized in Table 1. Among 2144 adult subjects, 71 were diagnosed with type II diabetes (prevalence = 3.3%). The distribution of gender and tobacco smoking between the two populations was similar (p > 0.05). In contrast, type II diabetic subjects were significantly older (54.5 ± 0.20 vs. 49 ± 1.01, p < 0.01) and more obese (42% vs. 12.4%). Likewise, mean glycemia and arterial hypertension were significantly higher in type II diabetic subjects. Nondiabetic patients consumed more alcohol.

Dental health indicators, adjusted for age, were better in the diabetic group where the subjects presented more healthy teeth and fewer restored teeth (p < 0.01) (Table 2). Except for the mean probing depth, periodontal health indicators, standardized for age,

Table 1. Descriptive analysis of medical data

	Nondiabetic mean or frequency $n = 2073$	Diabetic mean or frequency $n = 71$	<i>p</i> -value <i>t</i> -test or chi-square
Age (years)	49.0 (0.20)	54.5 (1.01)	< 0.001
Gender (males)	48.7%	59.1%	NS
BMI	25.4 (0.09)	29.9 (0.63)	< 0.0001
BMI category			< 0.0001
Underweight	1.8%	0%	
Normal weight	48.6%	15.6%	
Overweight	37.2%	42.2%	
Obese	12.4%	42.2%	
Cholesterol	5.63 (0.02)	5.61 (0.15)	NS
Glycemia	5.36 (0.02)	8.45 (0.35)	< 0.0001
AHT	9.8%	22.5%	0.0005
Tobacco			NS
Never	54.2%	52.1%	
Former	23.8%	16.9%	
Current	22.0%	31%	
Alcohol			< 0.0001
Never	16.0%	39.4%	
Past	1.8%	1.4%	
Actual	82.2%	59.2%	

AHT, arterial hypertension; BMI, body mass index; NS, not significant.

Table 2. Descriptive analysis of dental indicators, adjusted	for age
--	---------

	Nondiabetic $n = 2073$		Diabetic $n = 71$		Chi-square	
	n	Mean	n	Mean	type III	
Healthy teeth	1973	15.3 (0.14)	67	17.4 (0.79)	0.01	
Restored teeth	1832	6.7 (0.09)	59	5.30 (0.49)	0.005	
Extracted teeth	1705	4.6 (0.07)	59	4.37 (0.39)	NS	
Plaque index	2062	0.4 (0.01)	71	0.6 (0.06)	0.003	
Gingival index	2062	0.6 (0.01)	71	0.8 (0.07)	0.01	
Probing depth	2061	2.3 (0.01)	71	2.4 (0.05)	NS	
Attachment loss	2061	2.5 (0.01)	71	2.7 (0.07)	0.001	

NS, not significant.

were elevated significantly more in the diabetic group. Significant differences were detected for the PI [ratio of PI in diabetes vs. nondiabetes  $(R_{D/ND}^{PI}) = 0.66$ ], GI (ratio of GI in diabetes vs. nondiabetes  $(R_{D/ND}^{GI}) = 0.75$ ] and attachment loss  $(R_{D/ND}^{AL}) = 0.93$ ). The difference on mean probing depth between the two groups was not significant.

While low PI was similar in the two study populations, a significant difference was observed on elevated PI (PI2 and PI3) (Table 3). The PI ratio for diabetics/nondiabetics was 1.65 for PI3 and 0.71 for PI2. Diabetic men had a significantly higher PI than nondiabetic men (p < 0.001) and the  $R^{PI} =$ ;  $_{D/ND}^{0}$  was 1.68. Its value for the other scores was, respectively, 0.97

 $(\mathbf{R}^{\mathrm{PI}} => \frac{1}{D/ND})$ , 0.48  $(\mathbf{R}^{\mathrm{PI}} => \frac{2}{D/ND})$ , and 1.62  $(\mathbf{R}^{\mathrm{PI}} => \frac{3}{D/ND})$ . Diabetic women had a significantly higher PI than nondiabetic women (p < 0.001), with an  $\mathbf{R}^{\mathrm{PI}} = 0 = 0.44$ . Diabetic women had a higher PI for the higher severity score  $(\mathbf{R}^{\mathrm{PI}} = 3 = 1.52)$ , the ratio for the other categories being  $\approx 1$ .

In the same age group, plaque was more frequently detected at 40–49 years ( $\mathbb{R}^{\text{PI}} =$ ;  ${}^{0}_{40}$  – 49 = 0.35) in diabetic subjects (p < 0.01). The ratio was reversed at 50–59 years ( $\mathbb{R}^{\text{PI}} =$ ;  ${}^{0}_{50}$  – 59 = 1.49) and at 60–64 years ( $\mathbb{R}^{\text{PI}} =$ ;  ${}^{0}_{60}$  – 64 = 1.14). At any age, the PI3 value was higher in diabetics (p < 0.01).

Overall, the majority of subjects (50%) had a GI of 2 (Table 4). While a larger number of nondiabetic women had a GI of 0 or 1, diabetic men pre-

Table 3. Plaque index: percentage of subjects with more elevated score by <sup>sitea</sup>

	Subject $PI = 0$	Subject $PI = 1$	Subject $PI = 2$	Subject $PI = 3$
Diabetic	12.68	26.76	28.17	32.39
Yes 71	12.88	28.03	39.51	19.59
No 2073				
Gender				
Diabetic				
Man 42	16.67	26.19	19.05	38.10
Woman 29	6.90	27.59	41.38	24.14
Nondiabetic				
Man 1009	9.91	26.86	39.74	23.49
Woman 1064	15.70	29.14	39.29	15.88
Age				
Diabetic				
40-49 (21)	4.76	33.33	28.57	33.33
50-59 (20)	15.00	25.00	30.00	30.00
60-64 (27)	14.81	22.22	29.63	33.33
Nondiabetic				
40-49 (698)	13.75	25.64	39.54	21.06
50-59 (556)	10.07	27.34	42.99	19.60
60-64 (361)	13.02	26.04	39.34	21.61

<sup>a</sup> Standardized by age.

PI, plaque index.

sented significantly higher elevated values of GI (p < 0.01). About one-third of diabetics in any age category had a maximal GI of 3. In the non-diabetic group, the percentage of subjects with a GI of 3 was significantly lower (p < 0.001) (Table 4).

The relationship between the PI and GI values are presented in Table 5, Fig. 2 and Fig. 3.

Variance analysis (ANOVA) showed a significant difference in the registrated mean value distribution of GI and PI by quartile (p < 0.001). The mean values of GI in diabetics were less elevated for PI Q1, with a ratio of  $R^{GI}$  =  $> \frac{Q1}{D/ND} = 2.22$  (p < 0.01). For the other categories, the mean GI was systematically more elevated in diabetics, with respective ratios  $R^{GI} \Rightarrow \frac{Q3}{D/ND} = 0.63$  and  $R^{GI} \Rightarrow \frac{Q3}{D/ND} = 0.88$ . The reported correlation between GI quartiles and index scores were 16.55% (Q1), 10.23% (Q2), 11.14% (Q3) and 16.45% (Q4) for the nondiabetic group vs. 14.08, 5.63, 7.04 and 28.17 for the diabetic group.

Clinical periodontal indices and associated risk factors are presented in Table 6. For the nondiabetic subjects, the risk factors were age, gender, glycemia, tobacco smoking, and alcohol. For the diabetic subjects, only tobacco smoking was a significant risk factor.

# Discussion

In the present study, the prevalence of type II diabetes was 3.3%, similar to the prevalence of disease in the USA (14). The prevalence of type II diabetes was positively associated with age and was 3.5 times greater in subjects  $\geq 65$  years of age (15). Type II diabetes has been reported to be associated with hyperglycemia and arterial hypertension, and with being overweight (16). Our results are in agreement with these observations.

Numerous studies in the literature have demonstrated that individuals with diabetes tend to have a higher prevalence and more severe forms of periodontitis than nondiabetics. However, in these studies, individuals with type I or II diabetes were not distinguished (16–18).

Table 4. Gingival index: percentage of subjects with more elevated score by sitea

	Subject	Subject	Subject	Subject
	GI = 0	GI = 1	GI = 2	GI = 3
Diabetic				
Yes 71	8.45	15.49	46.48	29.58
No 1673	7.14	23.97	51.91	16.98
Gender				30.95
Diabetic				27.59
Man 42	14.29	16.67	38.10	30.95
Woman 29	0.00	13.79	58.62	27.59
Nondiabetic				
Man 819	6.34	21.11	53.52	19.03
Woman 1064	7.89	26.69	50.38	15.04
Age				
Diabetic				
40-49 (12)	0.00	27.27	45.45	27.27
50-59 (20)	15.00	15.00	30.00	40.00
60-64 (27)	7.41	18.52	40.74	33.33
Nondiabetic				
40-49 (698)	7.31	23.07	51.29	18.34
50-59 (556)	4.50	21.94	56.47	17.09
60-64 (361)	6.65	23.27	49.86	20.22

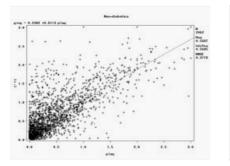
<sup>a</sup> Standardized by age.

GI, gingival index.

*Table 5*. Mean values of gingival index, according to the distribution of plaque index, by quartile

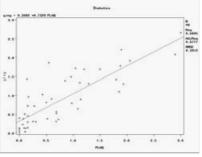
	Mean gingival ind	ex	<i>p</i> -value
Plaque index	Nondiabetic	Diabetic	
Quartile 1 (< 0.068)	0.20 (0.20)	0.09 (0.14)	0.001
Quartile 2 (0.068–0.25)	0.37 (0.19)	0.58 (0.11)	0.001
Quartile 3 (0.25–0.643)	0.63 (0.19)	0.70 (0.14)	NS
Quartile 4 ( $> 0.643$ )	1.22 (0.20)	1.39 (0.10)	0.01

NS, not significant.



*Fig. 2.* The relationship between GI and PI in the non-diabetics (m = 2073).

Only a small number of studies have investigated the relationship between type II diabetes and periodontitis. Five of these studies evaluated Pima Indians in Arizona and included subjects of 5 years and older or 15 years and older



*Fig. 3.* The relationship between GI and PI in the diabetics (m = 71).

(6,19–22). Three other reports included only adults (3,23,24), and reported a poorer periodontal health in diabetics. In 1990, Nelson *et al.* (6) reported a 2.6-fold greater risk of advanced periodontal disease in diabetics. In 1991, Emrich et al. reported that the odds were approximately three times greater for diabetes to have destructive periodontal disease (20). In 1998, Taylor et al. (22) reported that subjects with type II diabetes had a fourfold greater risk for more severe alveolar bone loss. In the NHANES survey, Selwitz et al. reported a large extent of periodontal disease in the group with type II diabetes (7). A more recent study (25) compared the periodontal health of nondiabetics and type II diabetic subjects. GI and attachment loss were elevated significantly more in the diabetics. These results are in agreement with the results of our study.

For a more precise evaluation of the inflammatory response in diabetics, we compared the GI of the two groups. When PI was held constant, GI was elevated more in diabetics. This observation was previously reported for type I (26) and type II diabetes (25), and indicates that with similar hygiene conditions, diabetes promotes pathogenecity of periodontal pathogens by altering the immune system.

Age is reported to be an aggravating factor for periodontal diseases (12) and diabetes (14). However, in our study, age was positively and significantly correlated with the four periodontal indices only in nondiabetics. These results are in accordance with the results of Kun Lu & Chyu Yang in 2004 (25) and some other observations on type I diabetes where age was not a significant factor. Some studies have limited their observations to men (6,25). A homogeneous mixed population was examined in our study. In the nondiabetic group, men had more elevated periodontal indices according to epidemiological studies on a general population (12). In the diabetic group, gender was not a significant factor. In diabetics, the diabetes factors are probably more significant than periodontal risk factors, age, and gender.

In our study, the impact of smoking on periodontal health was significant only in nondiabetics. A similar observation was made for alcohol. The current consumption of alcohol decreased the periodontal indices.

In conclusion, in France, diabetes is a risk factor for chronic periodontal

	Plaque index		Gingival in	dex	Probing depth		Attachment loss	
	Diabetic	Nondiabetic	Diabetic	Nondiabetic	Diabetic	Nondiabetic	Diabetic	Nondiabetic
Intercept	1.1771	-0.471	1.180	-0.256	2.913	1.779	3.428	1.292
Age	-0.001 (NS)	0.007 (p < 0.0001)	0.001 (NS)	0.007 (p < 0.0001)	-0.001 (NS)	0.005 (p < 0.0001)	-0.004 (NS)	0.018 (p < 0.0001)
Glycemia	-0.020 (NS)	0.129 (< 0.0001)	-0.023 (NS)	0.125 (< 0.0001)	-0.022 (NS)	0.054 (< 0.0001)	-0.044 (NS)	0.065 (0.0002)
Gender	(NS)	(< 0.0001)	(NS)	(< 0.0001)	(NS)	(< 0.0001)	(NS)	(< 0.0001)
Woman	0	0	0	0	0	0	0	0
Man	0.264	0.154	-0.004	0.117	-0.050	0.097	0.069	0.159
Tobacco	(p = 0.04)	(p < 0.0001)	(p = 0.03)	(0.005)	(NS)	(p < 0.0001)	(NS)	(p < 0.0001)
Never	0	0	0	0	0	Ō	0	0
Former	-0.459	0.174	-0.397	0.098	-0.223	0.103	0.275	0.240
Current	-0.632	-0.013	-0.584	-0.004	-0.292	0.017	-0.249	0.057
Alcohol	(NS)	(p < 0.0001)	(NS)	(p < 0.0001)	(NS)	(p < 0.0001)	(NS)	(p < 0.0001)
Never	0	0	0	0	0	0	0	0
Actual	-0.179	-0.271	0.094	-0.256	-0.092	-0.143	0.041	-0.232
Past	-0.527	0.123	-0.271	0.040	-0.031	-0.034	-0.602	0.305

Table 6. Multivaried analysis and estimation of regression coefficients of periodontal indices in diabetics and nondiabetics

NS, not significant.

disease in adults older than 35 years of age. This link can be explained by a direct causal relationship. Hyperglycemia and hyperlipidemia result in metabolic alterations, which may then exacerbate the bacteria-induced inflammatory periodontitis (27). Alterations in the host response include impaired function of neutophilic leukocytes (28), exaggerated response to lipopolysaccharide (29), increased production of tumour necrosis factor- $\alpha$  (TNF- $\alpha$ ) (30), decreased synthesis of collagen, and increased collagenase activity (31). Studies have shown that the advanced glycation end products (AGE), formed as a result of hyperglycemia/hyperlipidemia, can alter the phenotype of an number of cells via their cell-surface receptor (RAGE). This reaction can transform macrophages into destructive cells producing pro-inflammatory cytokines. According to some authors, the activation of RAGE can explain the exaggerated inflammation and the periodontal destruction observed in diabetic patients (31). The relationship between the two pathologies may be bidirectional (32). The control of periodontal infection is essential in diabetic patients (28).

## Acknowledgements

The current project is part of NPASES I, funded by the UFSBD, WHO Col-

laborating Center. The UFSBD supports several research projects, which shared data from the population-based NPASES I. This work was also partially supported by Grants from Pierre Fabre (France). The authors wish to thank P. Hescot, President, J. Desfontaine, and A. M. Farozi, epidemiology coordinators of French Union for Oral Health (UFSBD), and N. Deville of the National Health Insurance.

#### References

- Sheridan RC Jr, Cheraskin E, Flyn AC. Epidemiology of diabetes mellitus II. 100 dental patients. *J Periodontol* 1959;30: 298–323.
- Rylander H, Ramberg H, Lindhe J. Prevalence of periodontal disease in young diabetics. J Clin Periodontol 1986;14:38–43.
- Novaes AB Jr, Pereira ALA, de Moraes N, Novaes AB. Manifestations of insulindependent diabetes mellitus in the periodontium of young Brazilian patients. *J Periodontol* 1991;62:116–122.
- Thorstensson H, Hugoson A. Periodontal disease experience in adult long-duration insulin-dependent diabetics. J Clin Periodontol 1993;20:352–358.
- Löe H. Periodontal disease. The sixth complication of diabetes mellitus. *Diabetes Care* 1993;16:329–334.
- Nelson RG, Shlossman M, Budding LM et al. Periodontal disease and NIDDM in Pima Indians. *Diabetes Care* 1990;13:836– 840.
- Selwitz RH, Albandar JM, Harry HI. Periodontal disease in diagnosed diabetes:

US population 1988–1994. *J Dent Res* 1998;77(Spec. Iss. B): Abstr. 2139.

- Bourgeois D *et al.* Epidemiology of periodontal disease in adults in France 2002-2005 (in press).
- WHO. Obesity: Preventing and Managing the Global Epidemic of Obesity. Report of the WHO Consultation of Obesity. Switzerland, Geneva: World Health Organization June 3–5, 1997.
- Silness J, Löe H. Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. *Acta Odont Scand* 1964;24:747–759.
- Löe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odont Scand* 1963;21:533–551.
- Albandar JM. Periodontal diseases in North America. *Periodontol 2000* 2002;29:31–69.
- WHO. Calibration of Examiners for the International Collaborative Study of Oral Health Outcomes. Switzerland, Geneva: World Health Organization, 1994.
- Kenny SJ, Aubert RE, Geissl S. Prevalence and incidence of non-insulindependent diabetes. In: National Diabetes Date Group. *Diabetes in America*, 2nd edn. NIM Publication no. 95–1467. Washington, DC: Government Printing Office, 1995: iv47–iv68.
- Gabir MM, Hanson RL, Dabelea D et al. The 1997 American Diabetes Association and 1999 World Health Organization criteria for hyperglycemia and the diagnosis and prediction of diabetes. *Diabetes Care* 2000;23:1108–1112.
- Sznajder N, Carraro JJ, Rugna S, Sereday M. Periodontal findings in diabetic and non-diabetic patients. *J Periodontol* 1978;49: 445–448.

- Szpunar SM, Ismail AI, Eklund SA. Diabetes and periodontal disease. Analyses of NNHANES I and HHANES. J Dent Res 1989;68(Spec. Issue):383 (Abstr. 1605).
- Bridges RB, Anderson JW, Saxe SR, Gregory K, Bridges SR. Periodontal status of diabetic and non-diabetic men: effects of smoking, glycemic control, and socioeconomic factors. J Periodontol 1996;67:1185–1192.
- Schlossman M, Knowler WC, Pettitt DJ, Genco RJ. Type 2 diabetes mellitus and periodontal disease. J Am Dent Assoc 1990;121:532–536.
- Emrich LJ, Shlossman M, Genco RJ. Periodontal destruction in type II diabetes mellitus. J Periodontol 1991;62:123–130.
- Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M. Glycemic control and alveolar bone loss progression in Type 2 diabetes. *Ann Periodontol* 1998;3:30–39.
- Taylor GW, Burt BA, Becker MP et al. Non-insulin dependant diabetes mellitus and alveolar bone loss progression over 2 years. J Periodontol 1998;69:76–83.

- Morton AA, Williams RW, Watts TLP. Initial study of periodontal status in noninsulin-dependent diabetics in Mauritius. *J Dent Res* 1995;23:343–345.
- 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.
- Kung Lu H, Chyu Yang P. Analyse transversale de différentes variables parodontales de patients diabétiques non insulino-dépendants. *J Parodont Dent* 2004; 24:71–79.
- Cohen DW, Friedman LA, Shapiro J, Kyle GC, Franklin S. Diabetes mellitus and periodontal disease: Two year longitudinal observations. Part I. J Periodontol 1970;41:709–712.
- Miller AJ, Brunelle JA, Carlos JP, Brown LJ, Löe H. Oral Health of United State Adults. Betliesta, MD: National Institutes of Health, 1987: NIH Publication no. 87-2868.
- 28. Grossi SG, Zambon JJ, Ho AW et al. Assessment of risk for periodontal disease.

I. Risk indicators for attachment loss. *J Periodontol* 1994;65:260–267.

- Ryan ME, Ramamurthy S, Golub LM. Matrix metallo-proteinases and their inhibition in periodontal treatment. *Curr Opin Periodontol* 1996;3:85–96.
- Sasaki T, Ramamurthy NS, Yu Z, Golub LM. Tetracycline administration increases protein (presumably pro-collagen) synthesis and secretion in periodontal ligament fibroblasts of streptozotocin-induced diabetic rats. J Periodont Res 1992;27:631– 639.
- Lalla E, Lamster IB, Schmidt AM. Enhanced interaction of advanced glycation end products with their cellular receptor RAGE. Implications for the pathogenesis of accelerated periodontal disease in diabetes. Ann Periodontol 1998;3:13–19.
- Taylor GW, Burt BA, Becker MP et al. Severe periodontitis and risk for poor glycemic control in patients with noninsulin-dependent diabetes mellitus. J Periodontol 1996;67:1085–1093.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.