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Bone height changes in individuals with periodontal disease: a 17-year prospective longitudinal study

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Abstract

Objective: The aim of this study was to analyse changes in bone height after 17 years in smokers and non-smokers with periodontal disease, and to compare these with clinical assessment outcome.

Material and Methods: Participants comprised 50 adults with periodontitis and 18 healthy controls from a randomly selected epidemiological sample. Their mean age at the end of the study was 54.2 (SD \pm 3.09) years. The study included radiographic analysis compared with clinical data.

Results: The periodontitis group had significantly (p < 0.001) higher values than their healthy counterparts for plaque index (PLI), gingival index (GI), calculus index (CI), and bleeding on probing (BOP) at baseline and after 17 years. At the end of the follow-up, never-smokers with periodontitis had higher values for PLI (p < 0.05) and ex-smokers and smokers had higher GI and BOP (p < 0.001) than the controls. In all individuals with periodontitis, maxillary molars were most affected. Smokers had more severe marginal bone loss over time. Vertical bone defects were more often seen on the mesial side of teeth (p < 0.05).

Conclusion: Marginal bone level in this prospective study did reveal tooth groups at higher risk for progression of periodontal disease.

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Oral radiological examination of marginal bone level is one way to study progression of periodontal diseases, where marginal alveolar bone is affected, giving rise to horizontal and angular defects. Marginal bone height as a marker for progression of periodontal disease has been recorded in some longitudinal studies (Jansson et al. 2002, Laurell et al. 2003). Hugoson et al. (1998), in their longitudinal study of a Swedish population, found the prevalence for severe periodontal diseases to be 13%. Tobacco smoking, a wellestablished risk factor for periodontal diseases, has a negative effect on marginal bone (Bergström 1989, Haber

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et al. 1993, Papapanou 1996, Norderyd & Hugoson 1998). Bolin et al. (1993), in their longitudinal study of marginal bone changes, showed that marginal bone was more affected in smokers than in non-smokers. Changes in systemic conditions, e.g. diabetes and various hormonal disorders, can also have an effect on marginal bone (Kinane 1999). Moreover, cariological, endodontical, and other changes in oral conditions can cause alveolar bone loss (Kornman & Löe 1993, Grossi et al. 1994, Machtei et al. 1999, Geurs et al. 2003). Kornman & Löe (1993) demonstrated that when multiple factors become involved, the progression of

periodontal disease is not linear. Eickholz et al. (1998), in their study on the validity of radiographic measurement of interproximal bone loss, reported that both vertical and horizontal angulation differences between the central beam and the orthoradial projection will increase the risk of underestimating interproximal bone loss in radiographic examination. Measurement of periodontal disease with radiological examination was evaluated in one study where clinical assessment outcome was compared with radiological examination (Åkesson et al. 1992). The authors concluded that underestimation of bone loss ranged from 9% to 20% in periapical

radiographs, while probing resulted in a 5% error compared with measurements performed during flap surgery. In a longitudinal study (Benn 1990), measurements of bone loss of less than 1mm were assumed to be measurement error. Some studies show that there is a better correction for angulation errors because of foreshortening or elongation when bone loss is expressed as a percentage of root length (Björn 1968, Hausmann et al. 1989). Renvert & Persson (2004), in examining the relationship between clinical and radiographic periodontal data, concluded that alveolar bone loss could be predicted by the number of teeth lost and the proportions of plaque scores. Longitudinal studies are needed to clarify the progression of periodontitis on the marginal bone for both smokers and non-smokers, comparing radiological findings with clinical periodontal parameters for healthy patients.

The aim of this study was to analyse changes in bone height after 17 years in smokers and non-smokers with periodontal disease, and to compare these with clinical assessment outcome.

Material and Methods

Our study included 50 persons, 26 females and 24 males, with periodontitis and a high plaque/calculus index (CI) and 18 healthy age- and gender-matched controls, originating from a sample of 1676 adults (838 females and 838 males), aged 31-40 years at the start of the study. These 1676 individuals were listed in a registry file of all inhabitants of the Stockholm area born on the 20th of any month between 1945 and 1954 (n = 3273) (Söder et al. 1995). A clinical examination was performed in 1985-1986. Of these 1676 individuals, 289 (17.2%) had periodontal disease with the criterion one site with pocket depth \geq 5 mm, and 144 agreed to participate in a clinical study. From the 144 subjects, a computerized random sample of 50 subjects was collected for this prospective study and followed up to 2002. The control group consisted of 18 individuals, never-smokers who were periodontally healthy in 1985 as well as 2002, age and gender matched with the periodontitis group, and from the same large epidemiological sample. Over a 17-year period, oral health had been examined twice clinically for all participants, and with X-rays twice for periodontitis subjects and once for con-

trols (Wouters et al. 1988). Health questionnaires were included in the investigations. The full-mouth clinical examination comprised determination of the number of remaining teeth excluding third molars, the PLI (Silness & Löe 1964), the CI (Green & Vermillion 1964), the gingival index (GI) (Löe & Sillness 1963), and the number of teeth with pockets $\geq 5 \text{ mm}$. Pocket depth was measured with a Hu-Friedy probe (Hu-Friedy PCPUNC 15, Chicago, IL, USA) and recorded to the nearest higher millimetre at six sites of each tooth. Radiographic data for all remaining teeth were collected with conventional techniques using Ekta Speed periapical radiographs (Ekta Speed Eastman Kodak, Rochester, NY, USA) and Oralix[®] or Gendex[®] Roentgen machines (65 kVp/7.5 mA) with a cone of rectangular section and a film focus distance of approximately 30 cm. Marginal bone height was determined from the radiographs by a computerized measuring system and expressed as a percentage of root length at each measurable interproximal surface, excluding third molars. Vertical bone defects were determined as the distance between the horizontal bone margin and the most apical portion of the defect, at least 2 mm, and measured to the nearest millimeter (Persson et al. 1998). Only vertical defects that were free projected were counted. The marginal bone level was also analysed separately for the different tooth groups: maxillary and mandibular molars, premolars, canines, and incisors. One examiner (S. A.-M.) recorded all radiographs. The study protocol was approved by the Ethics Committee at Huddinge Hospital (Huddinge, Sweden.). All subjects gave their informed consent prior to participating in the study.

Statistical methods

The paired *t*-test was used to compare clinical and X-ray data of these two longitudinal observations, and the unpaired *t*-test was used to compare data between persons who had periodontal disease and their healthy counterparts. Differences between data sets with a probability of less than 0.05 were regarded as significant. Bonferroni correction for mass significance has been made for *p*-values. All data analyses were performed using the Stat View[®] 4.5 statistical package (SAS Institute Inc 1999). Intra-examiner

reproducibility was tested by doubleblinded re-analysis of all radiographs of five participants 6 months after the first analysis, and Spearman's coefficient was used.

Results

Testing of intra-examiner reproducibility showed no significant differences between the first analysis and the reanalysis. Spearman's rho coefficient was 0.78. The mean age for all participants (n = 68) at the 17-year follow-up was 54.2 (SD \pm 3.09) years, for participants periodontitis (n = 50)with 54.3 $(SD \pm 3.12)$ years, and for healthy controls (n = 18) 53.7 (SD \pm 3.06) years. The total number of smokers at the start of this study was 38; after 17 years, 11/ 21 females and 7/17 males continued to smoke. There were five never-smoker females and seven never-smoker males in the periodontitis group. At the beginning of the study, 74% of periodontitis patients had reported visiting their dentist during the last year; at the end of the study, this figure rose to 80%. Of these patients, 24% had visited a dental hygienist at the start of this study and 74% at the end of the study. During the entire study period, 64% of the individuals with periodontitis reported brushing their teeth two times daily and 20% three times daily. In all X-rays, there were 7.0% non-readable surfaces at baseline and after 17 years 5.4%. Results of the full-mouth clinical examination, excluding third molars and including mean values at baseline and after 17 years, for persons with periodontitis and healthy controls are shown in Table 1. For never-smokers, exsmokers, and smokers, significant differences were found for all clinical parameters measured, and compared with healthy controls (Table 2). Longitudinal changes in marginal bone height showed significantly lower values for all tooth groups (Table 3). After 17 years, there were significant differences in bone height between two neighbouring groups of teeth, the molars and premolars in the maxilla, both on the right side (p < 0.05) and on the left side (p < 0.001) (Table 3). Incisors in the upper jaw had lost 4.0% more bone than at baseline and lower incisors 8.0% (*p* < 0.001, respectively) (Table 3). Smokers had greater loss of marginal bone (p < 0.001) after 17 years than healthy controls (Table 4). Individuals

Table 1. Plaque index, gingival index, calculus index, and bleeding on probing in individuals with periodontitis and in periodontally healthy controls at baseline and after 17 years

	Baseline (mean \pm SD)	After 17 years (mean \pm SD)	р
PLI			
Periodontally healthy individuals $(n = 18)$	$0.51 \pm 0.35^{***}$	$0.14 \pm 0.13^{\# \# \#}$	< 0.001
Individuals with periodontitis $(n = 50)$	0.92 ± 0.40	0.82 ± 0.76	NS
GI			
Periodontally healthy individuals $(n = 18)$	$0.97 \pm 0.42^{***}$	$0.07 \pm 0.06^{\# \# \#}$	< 0.001
Individuals with periodontitis $(n = 50)$	2.07 ± 0.51	1.42 ± 0.96	< 0.001
CI			
Periodontally healthy individuals $(n = 18)$	$0.22 \pm 0.50^{***}$	0.05 ± 0.04	NS
Individuals with periodontitis $(n = 50)$	1.00 ± 0.83	0.38 ± 0.47	< 0.001
BOP			
Periodontally healthy individuals $(n = 18)$	$16.33 \pm 24.92^{***}$	$10.07 \pm 8.16^{\#\#}$	NS
Individuals with periodontitis $(n = 50)$	78.08 ± 28.80	41.03 ± 25.46	< 0.001

Significantly different oral clinical data for the two groups (healthy or periodontitis) at baseline; ***p < 0.001. Significantly different oral clinical data for the two groups (healthy or periodontitis) after 17 years; ###p < 0.001. PLI, plaque index; GI, gingival index; NS, not significant; CI, calculus index; BOP, bleeding on probing.

Table 2. Plaque index (PLI), gingival index (GI), calculus index (CI), bleeding on probing (BOP), in individuals with periodontitis (never-smokers, ex-smokers, and smokers) compared with periodontally healthy controls (n = 18) after 17 years.

	Periodontitis group (mean \pm SD)	Control group (mean \pm SD)	р
PLI,	0.73 ± 0.84	0.14 ± 0.13	< 0.05
Never-smokers with periodontitis, $n = 12$			
Ex-smokers with periodontitis, $n = 20$	0.84 ± 0.82	0.14 ± 0.13	< 0.001
Smokers with periodontitis, $n = 18$	0.89 ± 0.82	0.14 ± 0.13	< 0.001
GI,	0.92 ± 0.89	0.07 ± 0.05	< 0.001
Never-smokers with periodontitis, $n = 12$			
-smokers with periodontitis, $n = 20$	1.44 ± 0.83	0.07 ± 0.05	< 0.001
Smokers with periodontitis, $n = 18$	1.80 ± 1.02	0.07 ± 0.05	< 0.001
BOP,	45.27 ± 30.33	11.92 ± 9.76	< 0.001
Never-smokers with periodontitis, $n = 12$			
Ex-smokers with periodontitis, $n = 20$	39.04 ± 24.53	11.92 ± 9.76	< 0.001
Smokers with periodontitis, $n = 18$	39.43 ± 26.39	11.92 ± 9.76	< 0.001

Table 3. Bone height proportion (%) in maxillary and mandibular molars, at pre-molars, canines, and incisors in individuals with periodontitis (n = 50) at baseline and after 17 years

Bone height (%) Molars 16,17Pre-molars 14,15 89.0 ± 5.6 82.0 ± 7.8 Canines 13,23 91.8 ± 4.1 83.6 ± 8.9 Incisors 12,11,21,22 88.4 ± 7.0 84.4 ± 7.0 Pre-molars 24,25 88.3 ± 4.9 $82.4 \pm 5.3^{***}$ Molars 26,27 89.0 ± 4.7 75.6 ± 10.3	
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	< 0.001
	< 0.001
Molars 36,37 91.2 ± 3.9 84.8 ± 7.9	< 0.001
Pre-molars 34,35 91.9 ± 4.2 86.9 ± 5.2	< 0.001
Canines 33,43 93.5 ± 3.0 87.1 ± 7.5	< 0.001
Incisors 32,31,41,42 89.4 ± 7.7 81.4 ± 9.4	< 0.001
Pre-molars 44,45 90.6 ± 5.5 85.1 ± 5.9	< 0.001
Molars 46,47 90.7 ± 4.6 85.0 ± 3.6	< 0.001

Significantly different values for the two groups after 17 years; ***p < 0.001, **p < 0.05.

who, after 17 years, were current smokers had more bone loss on the lower front teeth. Data of the local marginal bone height for every tooth on the mesial and distal side, for the entire periodontitis group as well as for

healthy controls, showed that molars in the maxilla lost more marginal bone than all other teeth over time, followed by maxillary canines and mandibular incisors (Figs 1a, b). No one participating in this study had lost the canine on the left side of the mandible. The number and position of teeth missing at baseline or teeth lost during the 17 years are presented in Figs 2a, b. In the periodontitis group, 7/50 individuals were treated orthodontically before commencement of the study. In this group, seven pre-molars were lost before baseline. Marginal bone level for these individuals after 17 years was 86.94% $(SD \pm 3.93)$ and 80.69% $(SD \pm 11.00)$ for the other periodontally diseased individuals. Vertical bone defects were present in 6/7 orthodontically treated persons. The expected number of vertical bone defects for this group was calculated to be 5.5. At baseline, individuals with periodontitis had more vertical bone defects on the mesial side, mean 0.9 (SD \pm 1.40), than on the distal side, mean 0.3 (SD \pm 0.73) (p < 0.05). of the teeth. After 17 years, vertical bone defects were also detected significantly (p < 0.05) more on the mesial side, mean 1.0 (SD \pm 1.24), than on the distal side, mean 0.5 (SD \pm 0.67). Four individuals who were current smokers had more than six vertical bone defects at baseline. After 17 years, these four individuals had less than 22 teeth and only one to four vertical defects each.

Discussion

The age of participants in studies expressing periodontal disease progression using marginal bone level assessment is important since the prevalence of periodontal diseases increases with age (Albandar 1990, van der Velden 1991, Persson et al. 1998). The mean age of participants in this cohort at the end of the study was 54.2 (SD \pm 3.09) years. Thus, this study offers a welldefined group of patients in the same age range, thereby minimizing the bias of age. At the end of the study, none of the participants was edentulous. Marked loss of bone did, however, occur with time in all periodontitis patients. After 17 years, the marginal bone level was higher in all healthy persons compared with the periodontitis group at baseline, which is in accordance with the results reported by Papapanou et al. (1989).

They found that the healthiest subgroup between 20 and 64 years of age had a high proportion of tooth surfaces without bone loss.

In our study, the teeth in which progression of periodontitis was most evident were maxillary molars. A similar finding was made by Laurell et al. (2003), who concluded that maxillary molars and lower incisors had a higher risk for periodontal breakdown over time. Mandibular incisors in our study were another of the most affected tooth groups (Figs 1a, b). In the present study, maxillary molars were more frequently missing at the end of the 17-year observation period (Figs 2a, b). Mandibular canines, by contrast, were the most stable tooth group. The canine on the left side of the mandible was the only tooth that no one had lost at the end of the study. Canines have been reported to have the lowest mortality rate of all teeth (Papapanou et al. 1989). Renvert & Persson (2004), in their study on the relationship between clinical and radiographic periodontal data, concluded that the number of lost teeth and the proportions of plaque scores provided significant predictive factors for alveolar bone loss. In a 10-year cohort study, Paulander et al. (2004) found smoking to be a predictive factor for alveolar bone loss. In our study, PLI scores were higher in individuals with the lowest marginal bone level after 17 years, i.e. in smokers. Current smokers had lost more marginal bone after 17 years compared with never-smokers, and ex-smokers with periodontitis and healthy controls (Table 4). Current smokers also had more bone loss located on the lower front teeth. These findings can be explained by the local effects of tobacco as discussed elsewhere (Schmidt et al. 1999, Palmer et al. 2002). In our previous report on self-assessment of periodontal disease, smokers were more aware of their periodontal status than non-smokers (Airila-Månsson et al. 2004). Smoking can have an effect on pre-disposition for infection and inflammation. Smoking causes leucocyte activation, leucocyte endothelial adhesion, and neutrophil entrapment in the microvasculature, which may help initiate local tissue destruction (Schmidt et al. 1999, Palmer et al. 2002).

Since radiography is only two dimensional, it is limited in measuring vertical defects. Vertical defects increase over time, as shown in this study and in other studies (Wouters et al. 1989, Baljoon *Table 4*. Bone height proportion (%) in individuals with periodontitis (n = 50) (never-smokers, ex-smokers, and smokers) compared with periodontly healthy controls (n = 18) after 17 years

	Periodontitis group (mean \pm SD)	Control group (mean \pm SD)	р
Bone height (%)			
Periodontitis never-smokers $(n = 12)$	86.63 ± 4.15	93.83 ± 2.24	< 0.001
Periodontitis ex-smokers $(n = 20)$	85.82 ± 3.72	93.83 ± 2.24	< 0.001
Periodontitis smokers $(n = 18)$	75.65 ± 14.75	93.83 ± 2.24	< 0.001

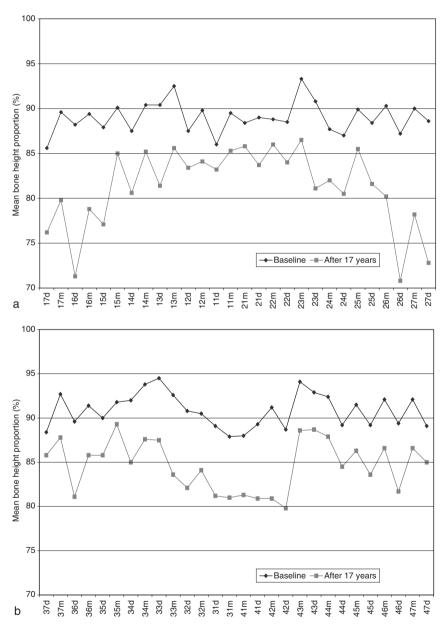


Fig. 1. (a). Maxillary teeth (17-27) mean bone height proportion at baseline and after 17 years. (b) Mandibular teeth (37-47) mean bone height proportion at baseline and after 17 years.

et al. 2003). Tooth fillings in the cervical area may limit the possibility of measuring these defects. Seven individuals in the present study who had been treated orthodontically had lost seven pre-molars probably through planned extractions as part of the treatment. The marginal bone level was higher in

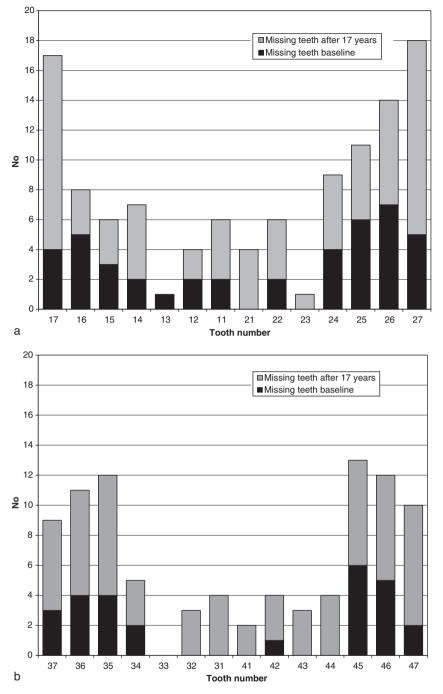


Fig. 2. (a). Maxillary teeth (17-27) missing at baseline and after 17 years. (b) Mandibular teeth (37-47) missing at baseline and after 17 years.

these individuals than in the other participants with periodontitis, and the number of vertical bone defects was similar to the statistically expected value. Axelsson et al. (2004) in their 30-year study, showed that a group of adults participating in a maintenance programme had a very low incidence of caries and periodontitis and a low tooth mortality rate. At the end of our study, the number of individuals with periodontitis who had visited a dental hygienist was three times as high as at the beginning of the study. In general, we found the marginal bone level for every tooth group at the 17-year follow-up to be lower in periodontitis patients with high plaque and gingivitis scores than in healthy controls; moreover, vertical bone defects were increasing in time and were more often seen on the mesial side of teeth.

Conclusion

Marginal bone level in this prospective study did reveal tooth groups at higher risk for progression of periodontal disease. Maxillary molars were most affected, with second maxillary molars being lost most frequently, thus having the most marginal bone loss of all teeth. Smokers had more severe marginal bone loss over time.

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References

- Airila-Månsson, S., Söder, B., Jin, L. J., Söder, P. O. & Klinge, B. (2004) Self-reporting of periodontal diseases and clinical assessment outcome in a Swedish urban population of smokers and non-smokers. Acta Odontologica Scandinavica 62, 111–115.
- Åkesson, L., Håkansson, J. & Rohlin, M. (1992) Comparison of panoramic and intraoral radiography and pocket probing for the measurement of the marginal bone level. *Journal* of Clinical Periodontology **19**, 326–332.
- Albandar, J. M. (1990) A 6-year study on the pattern of periodontal disease progression. *Jour*nal of Clinical Periodontology 17, 467–471.
- Axelsson, P., Nyström, B. & Lindhe, J. (2004) The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults. *Journal of Clinical Periodontology* **31**, 749–757.
- Baljoon, M., Natto, S. & Bergström, J. (2003) Occurrence of vertical bone defects in dentally aware individuals. *Acta Odontologica Scandinavica* 61, 47–51.
- Benn, D. K. (1990) A review of the reliability of radiographic measurements in estimating alveolar bone changes. *Journal of Clinical Periodontology* 17, 14–21.
- Bergström, J. (1989) Cigarette smoking as risk factor in chronic periodontal disease. *Community Dentistry and Oral Epidemiology* 17, 245–247.
- Björn, H. (1968) Radiographic assessment of periodontal disease. *International Dental Journal* 18, 611–619.
- Bolin, A., Eklund, G., Frithiof, L. & Lavstedt, S. (1993) The effect of changed smoking habits on marginal alveolar bone loss. A longitudinal study. *Swedisch Dental Journal* 17, 211–216.
- Eickholz, P., Kim, T. S., Benn, D. K. & Staehle, H. J. (1998) Validity of radiographic mea-

surement of interproximal bone loss. Oral Surgery Oral Medicine Oral Pathology Oral Radiology and Endodontics **85**, 99–106.

- Geurs, N. C., Lewis, C. E. & Jeffcoat, M. K. (2003) Osteoporosis and periodontal disease progression. *Periodontol 2000* 32, 105–110.
- Green, J. C. & Vermillion, J. R. (1964) The simplified oral hygiene index. *Journal of the American Dental Association* 68, 7–13.
- Grossi, S. G., Zambon, J. J., Ho, A. W., Koch, G., Dunford, R. G., Machtei, E. E., Norderyd, O. M. & Genco, R. J. (1994) Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *Journal of Periodontology* 65, 260–267.
- Haber, J., Wattles, J., Crowley, M., Mandell, R., Joshipura, K. & Kent, R. L. (1993) Evidence for cigarette smoking as a major risk factor for periodontitis. *Journal of Periodontology* 64, 16–23.
- Hausmann, E., Allen, K., Christersson, L. & Genco, R. J. (1989) Effect of x-ray beam vertical angulation on radiographic alveolar crest level measurement. *Journal of Periodontal Research* 24, 8–19.
- Hugoson, A., Norderyd, O., Slotte, C. & Thorstensson, H. (1998) Oral hygiene and gingivitis in a Swedish adult population 1973, 1983 and 1993. *Journal of Clinical Periodontology* 25, 807–812.
- Jansson, L., Lavstedt, S. & Zimmerman, M. (2002) Prediction of marginal bone loss and tooth loss-a prospective study over 20 years. *Journal of Clinical Periodontology* 29, 672–678.
- Kinane, D. F. (1999) Periodontitis modified by systemic factors. Annals of Periodontology 4, 54–64.
- Kornman, K. S. & Löe, H. (1993) The role of local factors in the etiology of periodontal diseases. *Periodontol 2000* 2, 83–97.
- Laurell, L., Romao, C. & Hugoson, A. (2003) Longitudinal study on the distribution of proximal sites showing significant bone

loss. Journal of Clinical Periodontology **30**, 346–352.

- Löe, H. & Sillness, J. (1963) Periodontal disease in pregnancy. (I) Prevalence and severity. Acta Odontologica Scandinavica 21, 533–551.
- Machtei, E. E., Hausmann, E., Dunford, R., Grossi, S., Ho, A., Davis, G., Chandler, J., Zambon, J. & Genco, R. J. (1999) Longitudinal study of predictive factors for periodontal disease and tooth loss. *Journal of Clinical Periodontology* 26, 374–380.
- Norderyd, O. & Hugoson, A. (1998) Risk of severe periodontal disease in a Swedish adult population. A cross-sectional study. *Journal* of Clinical Periodontology 25, 1022–1028.
- Palmer, R. M., Stapleton, J. A., Sutherland, G., Coward, P. Y., Wilson, R. F. & Scott, D. A. (2002) Effect of nicotine replacement and quitting smoking on circulating adhesion molecule profiles (sICAM-1, sCD44v5, sCD44v6). European Journal of Clininical Investigation 32, 852–857.
- Papapanou, P. N. (1996) Periodontal diseases: epidemiology. Annals of Periodontology 1, 1–36.
- Papapanou, P. N., Wennström, J. L. & Gröndahl, K. (1989) A 10-year retrospective study of periodontal disease progression. *Journal of Clinical Periodontology* 16, 403–411.
- Paulander, J., Wennström, J. L., Axelsson, P. & Lindhe, J. (2004) Some risk factors for periodontal bone loss in 50-year-old individuals. *Journal of Clinical Periodontology* 31, 489–496.
- Persson, R. E., Hollender, L. G. & Persson, G. R. (1998) Assessment of alveolar bone levels from intraoral radiographs in subjects between ages 15 and 94 years seeking dental care. *Journal of Clinical Periodontology* 25, 647–654.
- Renvert, S. & Persson, G. R. (2004) Patient-based assessments of clinical periodontal conditions in relation to alveolar bone loss. *Journal of Clinical Periodontology* **31**, 208–213.

- SAS Institute Inc (1999) *Stat View Reference*, 3rd edition. Cary, NC: SAS Institute Inc.
- Schmidt, M. I., Duncan, B. B., Sharrett, A. R., Lindberg, G., Savage, P. J., Offenbacher, S., Azambuja, M. I., Tracy, R. P. & Heiss, G. (1999) Markers of inflammation and prediction of diabetes mellitus in adults (Atherosclerosis Risk in Communities study): a cohort study. *Lancet* 353, 1649–1652.
- Silness, J. & Löe, H. (1964) Periodontal disease in pregnancy II: correlation between oral hygiene and periodontal condition. Acta Odontologica Scandinavica 22, 121–135.
- Söder, B., Jin, L. J., Söder, P. O. & Wikner, S. (1995) Clinical characteristics of destructive periodontitis in a risk group of Swedish urban adults. *Swedish Dental Journal* 19, 9–15.
- van der Velden, U. (1991) The onset age of periodontal destruction. Journal of Clinical Periodontology 18, 380–383.
- Wouters, F. R., Lavstedt, S., Frithiof, L., Söder, P. O., Hellden, L. & Salonen, L. (1988) A computerized system to measure interproximal alveolar bone levels in epidemiologic, radiographic investigations. II. Intra- and inter-examiner variation study. *Acta Odontologica Scandinavica* 46, 33–39.
- Wouters, F. R., Salonen, L. E., Hellden, L. B. & Frithiof, L. (1989) Prevalence of interproximal periodontal intrabony defects in an adult population in Sweden. A radiographic study. *Jour*nal of Clinical Periodontology 16, 144–149.

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