MF Timmerman
GA van der Weijden

Risk factors for periodontitis

Authors' affiliations:
M.F. Timmerman, G.A. van der Weijden, Department of Periodontology, Academic Centre for Dentistry Amsterdam, Amsterdam, The Netherlands

Correspondence to:
M.F. Timmerman
Department of Periodontology
Academic Centre for Dentistry Amsterdam
Louwesweg 1
1066 EA Amsterdam
The Netherlands
Tel.: +31 205 188 307
Fax: +31 205 188 512
E-mail: m.timmerman@acta.nl

Abstract: This review presents a selected overview of the literature concerning risk factors for periodontitis. That in some individuals gingivitis develops into periodontitis is still a matter of extensive research. Cross-sectional studies of clinical and microbiological factors can be meaningful. Longitudinal studies of the natural history allow analysis of potential factors and conditions that may have an impact on the disease process. At present, several possible risk factors for the initiation and progression of periodontitis have been identified: age, gender, plaque, calculus, existing attachment loss. A consistent finding appears to be genetic predisposition for the development of the disease. In terms of microbiology, several micro-organisms have been identified. The results of the Java Project on natural development of Periodontal Disease clearly pinpoint *Actinobacillus actinimycetemcomitans* as being associated with the onset of disease. The presence of subgingival calculus was found to be associated with onset and dental plaque with progression of disease. Consistent with literature males are more susceptible to disease. The presence of pockets ≥5 mm appear to be a useful tool, since it was found to be a prognostic factor for disease progression.

Key words: epidemiology; periodontal pathogens; periodontitis; risk factors

Introduction

A major objective of dental care is extending the life span of the dentition either by prevention or by treatment of dental diseases. The mean number of teeth present per person is, therefore, an important parameter in the assessment of the longevity of the dentition (1). It has been well accepted that the number of teeth decreases with age and that caries and periodontal disease are the main causes of tooth loss. The relative impact of

Dates:
Accepted 14 November 2005

To cite this article:
Int J Dent Hygiene **4**, 2006; 2–7
Timmerman MF, van der Weijden GA.
Risk factors for periodontitis

these two disease entities may vary in different population groups and geographic areas (2). Epidemiological surveys conducted throughout the world point to the almost universal distribution of caries and periodontal diseases (3). Most studies have found that periodontitis affects a significant number of individuals before the age of 20 years and affects the majority of the adult population after the age of 35–40 years. Studies report that the prevalence and average severity of periodontitis, increased with age for groups of individuals until virtually all middle-aged people had the disease [for review see Refs (4, 5)]. Data from the studies by Brekhus (6) and Allen (7) led many to suppose that the greatest single reason for tooth loss after the age of 40 years was periodontal disease. Later, this was confirmed in a survey in Winnipeg, MB, by Trott and Cross (8). However, their results also showed that the percentage of teeth lost because of periodontitis was higher than the percentage of patients who lost teeth because of periodontitis. In other words, with regard to periodontitis, relatively many teeth were lost in relatively few patients. While a large proportion of the population is susceptible to periodontitis, it appears that there is a small segment of the population that is susceptible to severe forms of periodontitis. This observation leads to the proposal that there are susceptibility or risk factors that modulate susceptibility to destructive periodontitis. The susceptibility of individuals appears to vary greatly depending upon which risk factors are operative (9).

Susceptibility

In a study of elderly people in Iowa, it was found that the distribution of tooth loss over an 18-month period was highly skewed (10). The same was found in a study over a period of 28 years in Tecumseh (11). In this particular study, 14.4% of the population became edentulous. This group accounted for 94% of all teeth lost during the study period. Among those who remained dentate, 13.8% of the persons lost teeth. They accounted for 60.2% of all teeth that were lost in dentate persons in that period. These data suggest that a minority of the population appears to be susceptible to extensive tooth loss, just as a minority appears susceptible to severe manifestations of caries (12, 13) and periodontal disease (14, 15).

Tooth loss

However, tooth loss in itself is not a disease. The question that arises, is to what extent this skewed distribution reflects social factors, or underlying diseases. Studies seeking reasons for tooth loss have not probed this issue (10, 16–23). A principal finding in these studies is, that periodontal disease is a less important reason for tooth loss at older age as once thought. Many questions on the relative impact of disease, patient attitudes, and treatment philosophy on tooth loss remain unanswered (24).

Risk terminology

A risk factor for periodontal disease is an environmental, behavioural, or biological factor confirmed by temporal sequence, usually longitudinal studies. If present, it directly increases the probability of a disease occurring. If absent, it reduces this probability. Risk factors are part of the causal chain, or expose the host to the causal chain. Once disease occurs, removal of a risk factor may not result in a cure (25, 26). Some risk factors are modifiable, while others cannot (easily) be modified. Factors that cannot be modified are often called 'determinants' or background factors.

The term risk indicator is used to describe plausible correlates of disease identified in cross-sectional studies or case–control studies, while risk factors are best applied to those correlates confirmed in longitudinal studies. Risk indicators are not always confirmed as risk factors in longitudinal studies (27).

The term risk marker is used more in the predictive sense and usually refers to a risk factor. It is associated with an increased probability of disease in the future.

Our knowledge on the aetiology of periodontal diseases and the recognition of the potential importance of susceptibility factors as they affect initiation and progression of periodontitis has changed. This has led to intense study of specific risk factors for destructive periodontal disease.

Putative risk factors

Epidemiological studies show more periodontal disease in older age groups than in younger groups (28–33). This may be the result of cumulative tissue destruction over a lifetime rather than an age-related, intrinsic deficiency or abnormality, which affects periodontal susceptibility. More recent studies suggest that at least in the moderately elderly, the rate of periodontal destruction is the same throughout adulthood (34, 35). When correcting for the oral hygiene status, age appears not of substantial influence on periodontal disease (30). Only at older age (between 75 and 96 years) more pronounced increase of periodontal disease has been reported (36, 37).

A consistent finding in all national surveys in the USA is that periodontal disease is more prevalent in males than in females (31, 38, 39). Risk analyses of periodontitis in other populations are not unanimous about gender as a risk factor (40–43). But, like in the USA, if a significant association is reported, males most often show a higher risk than females. Reasons for this have not been explored in detail, but are thought to be more a matter of differences in behaviour than in genetic background (44).

Socio-economic status historically has been found to be related to gingivitis and poor oral hygiene (38, 39). This does not hold true for periodontitis (31). Both in developing countries (45–47) and in industrialized countries (32, 33) it was found that lower socio-economic status was not associated with severity of periodontitis. It is not clear how other factors like true genetic racial/ethnic influence and cultural factors confound in this multifaceted variable.

Whereas oral hygiene measures correlate well with gingivitis, many cross-sectional studies show a poor correlation between levels of plaque and supragingival calculus and periodontitis (14, 48–56).

Plaque and calculus were discovered for their role in periodontal disease in the cross-sectional studies of the late 1950s and early 1960s (29, 57–59). Controlled studies in Western populations show that the amount of plaque has a low correlation with the amount of attachment loss measured (32, 33, 60–62). Also the predictive value of the amount of plaque for future progression of periodontitis is low (63, 64). On the contrary, in their 28-year longitudinal study on risk factors for tooth loss, Burt *et al.* (11) found that edentulous persons had higher baseline scores for plaque, calculus and gingivitis than age-matched dentate persons. Among dentate the baseline gingivitis score and the baseline number of teeth were risk factors for partial tooth loss. Also periodontal attachment loss ≥4 mm and educational attainment were found to be significant risk factors in the regression analysis. Subgingival calculus as a separate entity has not been studied epidemiologically.

When considering the best predictor for future attachment loss, the amount of existing disease in relation to age seems to be the best choice (64–66). However, this is still an inexact procedure (44).

A few members of the periodontal microflora have been considered as putative pathogens for initiation and progression of periodontal disease. Slots et al. (67) reported, in a retrospective study, a relationship between the presence of *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis* and *Prevotella intermedia* and progression of periodontal disease. Hence, the value of the presence of these three micro-organisms as a predictor for periodontal breakdown was investigated in adult cases with refractory periodontitis during a 12-month evaluation period. Progression of disease (attachment loss ≥2 mm) was not observed at sites without detectable levels of *A. actinomycetemcomitans*, *P. gingivalis* and *P. intermedia* (68). Also microbial epidemiological studies have shed light on the role of specific periodontal micro-organisms in periodontal disease. Carlos *et al.* (69) found the presence of *P. intermedia*, along with gingival bleeding and calculus was correlated with attachment loss in a group of Navajo adolescents aged 14–19 years. An epidemiological study of oral bacteria as risk indicators for periodontitis in older adults reported, that the difference in the prevalence of periodontal disease between blacks and whites was explained, in part, by different prevalences of *P. gingivalis* and *P. intermedia* (70). Grossi *et al.* (32, 33) tested a panel of micro-organisms including *A. actinomycetemcomitans*, *Tannellera forsythensis*, *Campylobacter rectus*, *Capnocytophaga* species, *Eubacterium saburreum*, *Fusobacterium nucleatum*, *P. gingivalis*, and *P. intermedia*. Of these micro-organisms, only *P. gingivalis* and *T. forsythensis* were associated with an increased risk for attachment loss after adjustment for age, plaque, smoking and diabetes. The same two micro-organisms were also identified as risk indicators for periodontal alveolar bone loss.

Discussion

As is apparent from all these studies, variables that have been suggested as possible risk factors for periodontal disease are not unanimously found to be so. The complex, multifaceted structure of these variables and their confounding influence on the multifactorial disease process of periodontitis may be the reason for the difficulty to assess the quality and quantity of the effects of these factors. A problem of performing studies in Western populations is that there is always some form of treatment effect involved. Ethical considerations do not allow for abstention of therapeutic measures in such a population. The effect of treatment influences the results of these studies to an extent that cannot be controlled for.

In a unique 15-year longitudinal epidemiological study in a young population deprived of regular dental care at a tea estate on Western Java, Indonesia, the role of a number of risk factors in the natural history of untreated periodontitis was investigated (43, 71–74). For this longitudinal prospective study, a village with approximately 2000 inhabitants at the Malabar/Poerbasari tea estate on Western Java, Indonesia, was selected. At the baseline evaluation in 1987 all inhabitants

($N = 255$) in the age range 15–25 years participated in this investigation. The subjects were deprived of regular dental care and had not been exposed to preventive dental care programs. Emergency dental treatment consisting of extraction of teeth was provided by a general physician. Therefore, this population was suitable for study of the natural development and progression of periodontitis. It consisted mostly of tea labourers, receiving basic medical care, employed by a government-owned tea estate, PTP XIII. Besides clinical assessment subject demographics and microbiological data were also obtained. In this population, it was found that the loss of attachment was associated with sibship. This confirms the assumption, that there is a genetic background to periodontal disease. Halfway through the study at the 7-year assessment, age, amount of subgingival calculus and subgingival presence of *A. actinomycetemcomitans* were found to be risk factors for occurrence of disease. At the 15-year evaluation, all subjects had developed periodontitis. This observation impeded with the possibility to evaluate the role risk factors. It, however, opened the opportunity to analyse prognostic factors for disease progression (75). These were the number of sites with Probing Depth ≥5 mm, the number of sites with recession and male gender. Interestingly, the prospective predictive value of dental plaque was found to be minor, which is in agreement with a recent analytical review of the epidemiology of periodontitis (76). However, retrospectively plaque was found to be a highly reliable factor, associated with disease activity and attachment loss. Thus, when considering sites with ongoing inflammation, the presence of dental plaque increases risk for future loss of attachment at these particular sites.

Conclusion

This review presents a selected overview the literature concerning risk factors for periodontitis. A consistent finding appears to be genetic predisposition for the development of the disease. In terms of microbiology, several micro-organisms have been identified. The results of the Java Project on natural development of Periodontal Disease clearly pinpoint *A. actinomycetemcomitans* as being associated with the onset of disease. As a local factor the presence of subgingival calculus was found to be associated with the onset and dental plaque with the progression of disease. Consistent with the existing literature, males are more susceptible to disease. In practice, this implicates the need for thorough screening. The presence of pockets ≥5 mm appear to be a useful tool, as it was found to be a prognostic factor for disease progression.

References

1 Sheiham A, Hobdell MH, Cowell CR. Patterns of tooth loss in British populations. Studies on industrial populations. *Br Dent J* 1969; **126:** 255–260.

2 Löe H, Ånerud Å, Boysen H, Smith M. The natural history of periodontal disease in man. The rate of periodontal destruction before 40 years of age. *J Periodontol* 1978; **49:** 607–620.

3 Russell AL. Epidemiology of periodontal disease. *Int Dent J* 1967; **17:** 282–296.

4 Scherp HW. Current concepts in periodontal disease research: epidemiological contributions. *J Am Dent Assoc* 1964; **68:** 667–675.

5 Brown LJ, Löe H. Prevalence, extent, severity and progression of periodontal disease. *Periodontology 2000* 1993; **2:** 57–71.

6 Brekhus PJ. Dental disease and its relation to the loss of human teeth. *J Am Dent Assoc* 1929; **16:** 2237–2247.

7 Allen EF. Statistical study of the primary causes of extractions. *J Dent Res* 1944; **23:** 453–458.

8 Trott JR, Cross HG. An analysis of the principle reasons for tooth extractions in 1813 patients in Manitoba. *Dent Pract* 1966; **17:** 20–27.

9 Genco RJ. Current view of risk factors for periodontal diseases. *J Periodontol* 1996; **67:** 1041–1049.

10 Hunt RJ, Hand JS, Kohout FJ, Beck JD. Incidence of tooth loss among elderly Iowans. *Am J Public Health* 1988; **78:** 1330–1332.

11 Burt BA, Ismail AI, Morrison EC, Beltran ED. Risk factors for tooth loss over a 28-year period. *J Dent Res* 1990; **69:** 1126–1130.

12 US Public Health Service, National Institute of Dental Research. *The prevalence of dental caries in United States children*. Publication No. 82-2245. Bethesda, MD: NIH, 1981.

13 Graves RC, Bohannan HM, Disney JA, Stamm JW, Bader JD, Abernathy JR. Recent dental caries and treatment patterns in US children. *J Public Health Dent* 1986; **46:** 23–29.

14 Löe H, Ånerud Å, Boysen H, Morrison E. Natural history of periodontal disease in man: rapid, moderate and no loss of attachment in Sri Lankan labourers 14 to 46 years of age. *J Clin Periodontol* 1986; **13:** 431–440.

15 US Public Health Service, National Institute of Dental Research. *Oral Health United States Adults*. Publication No. 87–2868. Bethesda, MD: NIH, 1987.

16 Ainamo J, Sarkki L, Kuhalampi ML, Palolampi L, Piirto O. The frequency of periodontal extractions in Finland. *Community Dent Health* 1984; **1:** 165–172.

17 Bouma J, Schaub RM, Van de Poel F. Periodontal status and total tooth extraction in a medium-sized city in the Netherlands. *Community Dent Oral Epidemiol* 1985; **13:** 323–327.

18 Cahen PM, Frank RM, Turlot JC. A survey of the reasons for dental extractions in France. *J Dent Res* 1985; **64:** 1087–1093.

19 Kay EJ, Blinkhorn AS. The reasons underlying the extraction of teeth in Scotland. *Br Dent J* 1986; **160:** 287–290.

20 Bailit HL, Braun R, Maryniuk GA, Camp P. Is periodontal disease the primary cause of tooth extraction in adults? *J Am Dent Assoc* 1987; **114:** 40–45.

21 Manji F, Baelum V, Fejerskov O. Tooth mortality in an adult rural population in Kenya. *J Dent Res* 1988; **67:** 496–500.

22 Chauncey HH, Glass RL, Alman JE. Dental caries. Principal cause of tooth extraction in a sample of US male adults. *Caries Res* 1989; **23:** 200–205.

23 Niessen LC, Weyant RJ. Causes of tooth loss in a veteran population. *J Public Health Dent* 1989; **49:** 19–23.

24 Weintraub JA, Burt BA. Oral health status in the United States: tooth loss and edentulism. *J Dent Educ* 1985; **49:** 368–378.
25 Last JM. *A Dictionary of Epidemiology*, 2nd edn. New York, NY: Oxford University Press, 1988.
26 American Academy of Periodontology. Consensus report for periodontal diseases: epidemiology and diagnosis. *Ann Periodontol* 1996; **1:** 221–222.
27 Beck JD. Methods of assessing risk for periodontitis and developing multifactorial models. *J Periodontol* 1994; **65(Suppl.):** 468–478.
28 Marshall-Day CD, Stephens RG, Quigley LF Jr. Periodontal disease: prevalence and incidence. *J Periodontol* 1955; **26:** 185–203.
29 Schei O, Waerhaug J, Lövdal A, Arno A. Alveolar bone loss as related to oral hygiene and age. *J Periodontol* 1959; **30:** 7–16.
30 Abdellatif HM, Burt BA. An epidemiological investigation into the relative importance of age and oral hygiene status as determinants of periodontitis. *J Dent Res* 1987; **66:** 13–18.
31 Miller AJ, Brunelle JA, Carlos JP, Brown LJ, Löe H. *Oral Health of United States Adults: National Findings*. Bethesda, MD: National Institute of Dental Research, NIH, publication no. 87–2868, 1987.
32 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.
33 Grossi SG, Genco RJ, Machtei EE et al. Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *J Periodontol* 1995; **66:** 23–29.
34 Holm–Pedersen P, Agerbaek N, Theilade E. Experimental gingivitis in young and elderly individuals. *J Clin Periodontol* 1975; **2:** 14–24.
35 Machtei EE, Dunford RG, Grossi SG, Genco RJ. Cumulative nature of periodontal attachment loss. *J Periodont Res* 1994; **29:** 361–364.
36 Douglass CW, Jette AM, Fox CH et al. Oral health status of the elderly in New England. *Gerodontology* 1993; **48:** 39–46.
37 Fox CH, Jette AM, McGuire SM, Feldman HA, Douglass CW. Periodontal disease among New England elders. *J Periodontol* 1994; **65:** 676–684.
38 US Public Health Service, National Center for Health Statistics. *Periodontal Disease in Adults, United States 1960–1962*. PHS publication no. 1000 Series 11, No 12. Washington, DC: Government Printing Office, 1965.
39 US Public Health Service, National Center for Health Statistics. *Basic Data on Dental Health Examination Findings of Persons 1–75 years; United States, 1971–1974*. DHEW publication no. (PHS) 79–1662, Series 11, No. 214. Washington, DC: Governement Printing Office, 1979.
40 Umeda M, Chen C, Bakker I, Contreras A, Morrison JL, Slots J. Risk indicators for harboring periodontal pathogens. *J Periodontol* 1998; **69:** 1111–1118.
41 Gamonal JA, Lopez NJ, Aranda W. Periodontal conditions and treatment needs, by CPITN, in the 35–44 and 65–74 year-old population in Santiago, Chile. *Int Dent J* 1998; **48:** 96–103.
42 Norderyd O, Hugoson A, Grusovin G. Risk of severe periodontal disease in a Swedish adult population. A longitudinal study. *J Clin Periodontol* 1999; **26:** 608–615.
43 Timmerman MF, Van der Weijden GA, Arief EM et al. Untreated periodontal disease in Indonesian adolescents. Subgingival microbiota in relation to experienced progression of periodontitis. *J Clin Periodontol* 2001; **28:** 617–627.
44 Position Paper of the American Academy of Periodontology. Epidemiology of periodontal diseases. *J Periodontol* 1996; **67:** 935–945.
45 Russell AL. Periodontal disease in well and malnourished populations. *Arch Environ Health* 1962; **5:** 153–157.
46 Waerhaug J. Prevalence of periodontal disease in Ceylon. Association with age, sex, oral hygiene, socio-economic factors, vitamin deficiencies, malnutrition, betel and tobacco consumption and ethnic group. Final report. *Acta Odontol Scand* 1967; **25:** 205–231.
47 Wertheimer FW, Brewster RH, White CL. Periodontal disease and nutrition in Trinidad. *J Periodontol* 1967; **38:** 100–104.
48 Cutress TW, Powell RN, Ball ME. Differing profiles of periodontal disease in two similar South Pacific island populations. *Community Dent Oral Epidemiol* 1982; **10:** 193–203.
49 Baelum V, Fejerskov O, Karring T. Oral hygiene, gingivitis and periodontal breakdown in adult Tanzanians. *J Periodont Res* 1986; **21:** 221–232.
50 Baelum V, Fejerskov O, Manji F. Periodontal diseases in adult Kenyans. *J Clin Periodontol* 1988; **15:** 445–452.
51 Baelum V, Wen–Min L, Fejerskov O, Xia C. Tooth mortality and periodontal conditions in 60–80-year-old Chinese. *Scand J Dent Res* 1988; **96:** 99–107.
52 Ismail AI, Burt BA, Brunelle JA. Prevalence of total tooth loss, dental caries, and periodontal disease in Mexican–American adults: results from the southwestern HHANES. *J Dent Res* 1987; **66:** 1183–1188.
53 Ismail AI, Eklund SA, Burt BA, Calderone JJ. Prevalence of deep periodontal pockets in New Mexico adults aged 27 to 74 years. *J Public Health Dent* 1986; **46:** 199–206.
54 Lembariti BS, Frencken JE, Pilot T. Prevalence and severity of periodontal conditions among adults in urban and rural Morogoro, Tanzania. *Community Dent Oral Epidemiol* 1988; **16:** 240–243.
55 Löe H, Ånerud Å, Boysen H. The natural history of periodontal disease in man: prevalence, severity, and extent of gingival recession. *J Periodontol* 1992; **63:** 489–495.
56 Okamoto H, Yoneyama T, Lindhe J, Haffajee A, Socransky S. Methods of evaluating periodontal disease data in epidemiological research. *J Clin Periodontol* 1988; **15:** 430–439.
57 Lövdal A, Arno A, Waerhaug J. Incidence of clinical manifestations of periodontal disease in the light of oral hygiene and calculus formation. *J Am Dental Assoc* 1958; **56:** 21–33.
58 Ladavalya MRN, Harris R. A study of the gingival and periodontal conditions of a group of people in Chieng Mai Province. *J Periodontol* 1959; **30:** 219–222.
59 Mobley E, Smith SH. Some social and economic factors relating to periodontal disease among young Negroes: No. 1. *J Am Dent Assoc* 1963; **66:** 486–491.
60 Lindhe J, Okamoto H, Yoneyama T, Haffajee A, Socransky SS. Longitudinal changes in periodontal disease in untreated subjects. *J Clin Periodontol* 1989; **16:** 662–670.
61 Badersten A, Nilveus R, Egelberg J. Scores of plaque, bleeding, suppuration and probing depth to predict probing attachment loss. 5 years of observation following nonsurgical periodontal therapy. *J Clin Periodontol* 1990; **17:** 102–107.
62 Machtei EE, Christersson LA, Zambon JJ et al. Alternative methods for screening periodontal disease in adults. *J Clin Periodontol* 1993; **20:** 81–87.
63 Claffey N, Nylund K, Kiger R, Garrett S, Egelberg J. Diagnostic predictability of scores of plaque, bleeding, suppuration and probing depth for probing attachment loss. 3 1/2 years of observation following initial periodontal therapy. *J Clin Periodontol* 1990; **17:** 108–114.

64 Haffajee AD, Socransky SS, Lindhe J, Kent RL, Okamoto H, Yoneyama T. Clinical risk indicators for periodontal attachment loss. *J Clin Periodontol* 1991; **18:** 117–125.

65 Grbic JT, Lamster IB, Celenti RS, Fine JB. Risk indicators for future clinical attachment loss in adult periodontitis. Patient variables. *J Periodontol* 1991; **62:** 322–329.

66 Grbic JT, Lamster IB. Risk indicators for future clinical attachment loss in adult periodontitis. Tooth and site variables. *J Periodontol* 1992; **63:** 262–269.

67 Slots J, Bragd L, Wikström M, Dáhlen G. The occurrence of *Actinobacillus actinomycetemcomitans*, *Bacteroides gingivalis* and *Bacteroides intermedius* in destructive periodontal disease in adults. *J Clin Periodontol* 1986; **13:** 570–577.

68 Wennström JL, Dahlén G, Svensson J, Nyman S. *Actinobacillus actinomycetemcomitans*, *Bacteroides gingivalis* and *Bacteroides intermedius*: predictors of attachment loss? *Oral Microbiol Immunol* 1987; **2:** 158–162.

69 Carlos JP, Wolfe MD, Zambon JJ, Kingman A. Periodontal disease in adolescents: some clinical and microbiologic correlates of attachment loss. *J Dent Res* 1988; **67:** 1510–1514.

70 Beck JD, Koch GG, Zambon JJ, Genco RJ, Tudor GE. Evaluation of oral bacteria as risk indicators for periodontitis in older adults. *J Periodontol* 1992; **63:** 93–99.

71 Timmerman MF, Van der Weijden GA, Abbas F et al. Untreated periodontal disease in Indonesian adolescents. Clinical and microbiological baseline data. *J Clin Periodontol* 1998; **25:** 215–224.

72 Timmerman MF, Van der Weijden GA, Abbas F et al. Untreated periodontal disease in Indonesian adolescents. Longitudinal clinical data and prospective clinical and microbiological risk assessment. *J Clin Periodontol* 2000; **27:** 932–942.

73 Van der Velden U, Abbas F, Armand S et al. The effect of sibling relationship on the periodontal condition. *J Clin Periodontol* 1993; **20:** 683–690.

74 Van der Velden U, Van Winkelhoff AJ, Abbas F et al. Longitudinal evaluation of the development of periodontal destruction in spouses. *J Clin Periodontol* 1996; **23:** 1014–1019.

75 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:** 291–296.

76 Borrell LN, Papapanou PN. Analytical epidemiology of periodontitis. *J Clin Periodontol* 2005; **32 (Suppl. 6):** 132–158.