ELSEVIER
SAUNDERS

Dent Clin N Am 49 (2005) 517–532

THE DENTAL
CLINICS
OF NORTH AMERICA

Epidemiology and Risk Factors of Periodontal Diseases

Jasim M. Albandar, DDS, DMD, PhD[a,b,c,*]

[a]*Department of Periodontology, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia, PA 19140, USA*
[b]*Periodontal Diagnostics Research Laboratory, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia, PA 19140, USA*
[c]*Temple University Graduate School, Room 3A07, 3223 North Broad Street, Philadelphia, PA 19140, USA*

Periodontal diseases and dental caries are the main chronic infectious diseases of the oral cavity and the principal causes of tooth loss in humans. Periodontal diseases include a group of chronic inflammatory diseases that affect the periodontal supporting tissues of teeth and encompass destructive and nondestructive diseases [1]. Gingivitis is inflammation of the soft tissue without apical migration of the junctional epithelium. It is a reversible, nondestructive disease that does not involve loss of periodontal tissues [2]. Periodontitis is inflammation of the periodontium that is accompanied by apical migration of the junctional epithelium, leading to destruction of the connective tissue attachment and alveolar bone loss [3,4].

Chronic periodontitis is the most common form of destructive periodontal diseases [3,5] and shows a slow disease progression characterized by bursts of disease activity separated by quiescent periods of varying durations [6–8]. Aggressive periodontitis encompasses aggressive, rapidly progressive forms of periodontitis [9], which often commence during adolescence and early adulthood, and hence classified as early-onset periodontitis [10–12]. Two other groups of destructive periodontal diseases exist, including periodontitis as a manifestation of systemic diseases [13] and necrotizing periodontal diseases [14].

Periodontal diseases share common etiologic factors and have several predisposing factors [15–17]. Most periodontal diseases are infectious in

* Department of Periodontology, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia, PA 19140.
E-mail address: jasim.albandar@temple.edu

doi:10.1016/j.cden.2005.03.003 *dental.theclinics.com*

nature, initiated as a consequence of dental plaque biofilm formation [18–20]. Another common feature of these diseases is that host factors play an important role in their development [21] and therefore show some similarities in their histopathogenesis. Nevertheless, various types of periodontal diseases also show distinctive differences in etiology and risk predisposition. As a result, host factors may play different roles in the development of various periodontal diseases. In addition, the occurrence, clinical features, and progression pattern of these diseases differ significantly.

Goals of epidemiologic studies of periodontal diseases

Epidemiology is the study of health and disease in populations and the effect of various biologic, demographic, environmental, and lifestyles on these states. Epidemiologic studies are conducted to describe the health status of populations, elucidate the etiology of diseases, identify risk factors, forecast disease occurrence, and assist in disease prevention and control. Epidemiologic research often involves the measurement of parameters of disease and health, the estimation of their occurrence in populations, and the use of population estimates to carry out statistical testing of hypotheses related to disease-associated variables. Population-based measurements may include the estimation of the percentage of the population that currently exhibit the disease (prevalence), the percentage of the population that may acquire the disease during a given time interval (incidence), and the probability of occurrence of the disease in the population during a given time interval in the future (risk).

Population studies of the distribution and risk factors of periodontal diseases offer a unique investigational model that provides power and generalization to observations made among more limited populations. By contributing to a better understanding of the causal relationships between risk factors and occurrence of disease, epidemiologic studies form the basis of the disciplines of ''risk assessment'' and ''disease control.'' Information about the distribution of periodontal diseases in the population, current knowledge about the pathogenesis and methods of control of these diseases, and the population's perception of the disease constitute the foundation for determining periodontal treatment needs and making resource allocation decisions for disease control.

Diagnostic criteria and measurement methods

Gingivitis is identified clinically as inflammation of the gingiva causing one or more soft tissue changes including redness, edema, increase in thickness, ulceration, or bleeding. Several indices have been described for the measurement of gingivitis [2]. More recently, however, diagnostic

criteria and indices that assess the presence, extent, or severity of gingival bleeding [22,23] have been used because they provide more objective methods of assessing the degree of gingival inflammation.

Studies have assessed periodontal health and the severity of periodontitis in populations using a wide range of approaches and methodologies. One common approach has been the assessment of periodontal soft or hard tissue loss, with or without assessment of the degree of inflammation. Probing pocket depth has often been used as a measure of local periodontal tissue inflammation and tissue loss, even though assessments of the status of epithelial attachment have often been overlooked, and the validity of periodontal disease estimates based on this measure may therefore be questioned. Hence, differences in disease parameters measured in different studies may partly explain the differences in study findings.

Indices are measurement instruments used in some studies to assess the periodontal health of populations. Simplifying the assessment method and the potential reduction in measurement errors are important advantages of using periodontal indices. Hence, indices may allow a more straightforward approach to gathering and interpreting data. Russell's Periodontal Index [24] and Ramfjord's Periodontal Disease Index [25] were commonly used in periodontal surveys from the 1950s to 1970s but have seldom been used thereafter because of major validity limitations [26,27]. The Community Periodontal Index of Treatment Needs [28] and its newer incarnation, the Community Periodontal Index [29], are widely used today; however, both indices have been criticized for their limited validity for the assessment of periodontal diseases [27,30].

More recently, the Periodontitis Index [26,31] was described to assess the prevalence and severity of periodontitis in the population. This index can use full-mouth data or partial measurements typically used in large national surveys. The Periodontitis Index classifies subjects as having mild, moderate, or advanced periodontitis or not having periodontitis. Because this index does not combine parameters of different diseases, it does not endure some of the validity limitations found in the other indices mentioned earlier. The index uses measurements of attachment loss and probing depth in the evaluation of the severity of disease. The probing depth is measured to indicate only the depth of the true pocket (ie, the probing depth apical to the cementoenamel junction). In the absence of periodontal inflammation and pocketing, the Periodontitis Index does not regard presence of attachment loss alone as a measure of periodontitis.

Because of lack of resources or the desire to simplify the examination process, many epidemiologic studies of periodontal diseases have used partial recording methods to assess the occurrence and severity of disease. Partial recording protocols, however, systematically underestimate periodontal disease prevalence, and the degree of underestimation is influenced by the type of protocol used [22,32].

Epidemiology of periodontal diseases

Gingivitis

Epidemiologic studies show that gingivitis is ubiquitous in populations of children and adults globally. It has been estimated that more than 82% of adolescents in the United States have overt gingivitis and signs of gingival bleeding [33]. Similar or higher prevalence of gingivitis has been reported in children and adolescents in other parts of the world [34–38]. A high prevalence of gingivitis is found also in adults. For instance, it has been estimated that more than half of United States adults have gingival bleeding [26,39,40]. Adjusting for the measurement bias due to the use of partial recording protocols in the United States national surveys (as described by Kingman and Albandar [22]), more than 75% of American adults have gingival bleeding and a high percentage have dental calculus, suggesting poor oral hygiene. Other populations show similar or even higher levels of gingival inflammation [38].

Gingivitis is a reversible condition, which except for indicating the level of oral hygiene in the population, has unclear significance as a predictor of future periodontal tissue loss [41,42]. Nonetheless, a high prevalence and extent of gingival bleeding in the population may be detrimental, which is supported by data showing that young subjects who have overt gingival inflammation may show a higher frequency of future periodontal attachment loss than subjects without inflammation [43].

Chronic periodontitis

Nonaggressive forms of periodontitis (chronic periodontitis) are common worldwide. They may occur in most age groups but are most prevalent among adults and seniors [44]. The estimates of chronic periodontitis in various populations vary significantly, in part because populations differ in their demographics and levels of exposure to various etiologic and risk factors of periodontitis. In addition, studies often have used inconsistent study design and many lack standardization of disease definition and measurement methods. Hence, it may be difficult to draw valid conclusions about the prevalence and severity of disease in different populations based on published reports.

Chronic periodontitis in children and adolescents shows a wide frequency range in various geographic regions and racial/ethnic groups [35,44–46]. Low disease frequencies among young Caucasians in Western Europe and North America and relatively high frequencies in Africa and Latin America have been reported. In the age group 11 to 25 years, it is estimated that the disease affects 1% to 3% in Western Europe, 2% to 5% in North America, 4% to 8% in South America, 5% to 8% in Asia, and 10% to 20% in Africa [35]. The estimates by racial/ethnic groups are 1% to 3% of Caucasians, 5% to 8% of Asians, 5% to 10% of Hispanics and Latin Americans, and 8% to 20% of Africans and African Americans.

Two recent surveys, the National Survey of Employed Adults and Seniors (EAS survey, 1985–1986) and the Third National Health and Nutrition Examination Survey (NHANES III, 1988–1994), have provided invaluable information about the epidemiology of periodontal and other oral diseases in the United States population. The EAS survey used a multistage sampling design using United States business establishments (adults, 18–64 years old) and senior centers (65 years and older) as sampling frames. This survey found that approximately 24% and 14% of employed adults and 68% and 52% of seniors had one or more teeth with $\geq$4 mm and $\geq$5 mm attachment loss, respectively; 14% and 4% of adults and 22% and 8% of seniors had teeth with $\geq$4 mm and $\geq$5 mm probing depth, respectively; and 84% and 89% of adults and seniors had dental calculus, respectively [26].

The NHANES III also used a multistage sampling design but was household based, targeting the civilian noninstitutionalized United States population. Among 30- to 90-year-olds, 33% and 20% had $\geq$4 mm and $\geq$5 mm attachment loss, respectively, and 23% and 9% had $\geq$4 mm and $\geq$5 mm probing depth, respectively [31]. In the age group 65 years and older, more than 80% and 40% had attachment loss of $\geq$3 mm and $\geq$5 mm, respectively, and about 60% and 10% had probing depth of $\geq$3 mm and $\geq$5 mm, respectively [26].

It is important to note that the EAS survey and the NHANES III used partial-mouth examinations whereby only the midbuccal and mesiobuccal tooth surfaces of one maxillary and one mandibular quadrants were examined. Kingman and Albandar [22] showed that this method significantly underestimates the prevalence of attachment loss and suggested a method to adjust for the bias in these estimates. Using this approach to adjust the disease estimates in the EAS survey and the NHANES III, it may be concluded that about 41% and 22% of employed adults aged 18 to 64 years and 100% and 83% of seniors aged 65 years and older had one or more teeth with $\geq$4 mm and $\geq$5 mm attachment loss, respectively (Table 1). Similarly, in the NHANES III, 56% and 32% of subjects 30 to 90 years old had corresponding attachment loss. The estimates in subjects aged 65 years and older were 100% and 60% had $\geq$3 mm and $\geq$5 mm attachment loss, respectively, 90% and 15% had $\geq$3 mm and $\geq$5 mm probing depth, respectively, and 75% and 22% had $\geq$3 mm and $\geq$5 mm gingival recession, respectively. Population studies in American seniors [47–49] have typically used partial recording protocols similar to the one used in recent United States national surveys. Adjusting for the measurement bias due to this design shows that United States seniors have a very high level of chronic periodontitis and tissue loss comparable to the rates reported in the EAS survey and the NHANES III.

Albandar et al [31] assessed the prevalence and severity of chronic periodontitis in the United States adult population. Estimates based on partial recordings showed that from 1988 to 1994, about 35% of United States dentate adults aged 30 years and older had periodontitis. These adults included 22%, 10%, and 3% with mild, moderate, and advanced

Table 1
Percentage of subjects by degree of attachment loss and probing depth assessed in two recent United States national surveys using a partial recording system (random half-mouth and two sites per tooth) and the estimated true prevalence rates using an inflation factor to adjust for measurement bias

	Partial recording system				Full-mouth estimates		
Parameter	EAS 18–64 y	EAS 65+ y	NHANES 30–90 y	Inflation factor	EAS 18–64 y	EAS 65+ y	NHANES 30–90 y
Attachment loss $\geq$4 mm	24.1	68.2	32.7	1.7	41.0	100.0	55.6
Attachment loss $\geq$5 mm	13.6	51.7	19.9	1.6	21.8	82.7	31.8
Probing depth $\geq$4 mm	14.3	22.2	23.1	1.4	20.0	31.1	32.3
Probing depth $\geq$5 mm	4.3	7.6	8.9	1.6	6.9	12.2	14.2
Mild periodontitis	—	—	21.8	1.4	—	—	30.5
Moderate periodontitis	—	—	9.5	1.4	—	—	13.3
Severe periodontitis	—	—	3.1	1.4	—	—	4.3

Data from Albandar JM. Periodontal diseases in North America. Periodontol 2000 2002; 29:31–69; and Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988–1994. J Periodontol 1999;70(1):13–29.

periodontitis, respectively (see Table 1). Adjusting for the measurement error due to the partial recordings suggests that approximately 48% of United States adults had chronic periodontitis, including 31%, 13%, and 4% with mild, moderate, and advanced periodontitis, respectively.

In Europe, a high percentage of adults have moderate probing depth and mild to moderate periodontal attachment loss. It is estimated that between 13% and 54% of 35- to 44-year-olds have 3.5- to 5.5-mm probing depths [50]. These estimates, however, may be conservative because they were derived from poorly documented surveys. A large survey in the United Kingdom found that 42% of 35- to 44-year-olds and 70% of 55- to 64-year-olds had attachment loss $>$3.5 mm [51]. Generally, a higher prevalence of periodontal disease is reported in Western European compared with Eastern European populations.

Credible data from other regions of the world are scarce. Most studies in Central and South America used convenience samples, and the criteria and measures of periodontal disease were not clearly defined. It has been common to use the Community Periodontal Index of Treatment Needs for evaluating periodontal status, and measurement of periodontal attachment loss is seldom reported. Given these inaccuracies, the estimates of disease in this region are uncertain. It has been estimated that approximately 38% to 67% of the population in Central and South America have moderate or advanced chronic periodontitis, including 28% to 52% with moderate disease and 4% to 19% with severe disease [37]. A recent survey used a representative sample of the adult urban population in southern Brazil and showed that 79% and 52% of subjects aged 30 years and older had attachment loss of $\geq$5 and $\geq$7 mm, respectively, and poor oral hygiene and dental calculus were prevalent [52]. Periodontitis is widespread among the

older age groups, with approximately 70% to 100% of adults aged 50 years and older having moderate or advanced chronic periodontitis [37,53].

Studies also show that African populations have poor oral hygiene, abundant calculus, and a high prevalence of moderate probing depth and attachment loss. Disease estimates in this continent, however, may not be accurate due to the scarcity of reliable studies. Among adults aged 30 years and older, the prevalence range of $\geq$4 mm attachment loss is 44% to 84% in Uganda, 79% to 98% in Tanzania, and 91% to 99% in Kenya [27].

The continent of Asia includes more than half of the world's population, with groups of diverse ethnicity and marked differences in wealth and other socioeconomic characteristics. Generally, studies report a very high prevalence of gingival bleeding and dental calculus and a low prevalence of healthy periodontal status irrespective of age cohort or the country's level of development [54]. Periodontal diseases are more prevalent in high-income countries compared with low-income countries, particularly in the younger age groups, whereas these differences are less pronounced in seniors.

Early-onset aggressive periodontitis

Aggressive periodontitis is a disease characterized by severe and rapid loss of periodontal attachment that affects the permanent dentition and may commence at or after the circumpubertal age [1,11]. This nomenclature has been recommended to replace earlier classifications, including early-onset, juvenile, and rapidly progressive periodontitis [10]. For the most part, epidemiologic studies of aggressive periodontitis have many of the same inherent weaknesses of disease definition and study design as studies of chronic periodontitis. Thus, issues such as disease misclassification and inconsistent exclusion criteria of cases make inferences exigent.

Aggressive periodontitis shows a wide range of occurrence frequencies in various geographic regions and demographic groups, and the variance seems particularly large between different racial/ethnic groups. Prevalence estimates in the age group 11 to 25 years are 0.1% to 0.5% in Western Europe, 0.4% to 0.8% in North America, 0.3% to 1.0% in South America, 0.4% to 1.0% in Asia, and 0.5% to 5.0% in Africa [35]. Estimates of disease occurrence by racial/ethnic groups are 0.1% to 0.2% of Caucasians, 0.4% to 1.0% of Asians, 0.5% to 1.0% of Hispanics and Latin Americans, and 1.0% to 3.0% of Africans and African Americans.

Gingival recession

Periodontitis is often followed by recession of the periodontal soft tissues, which results in exposure of the root surfaces of teeth. Gingival recession also may be related to factors other than inflammatory periodontal disease. These include physical trauma, anatomical factors, and other etiological factors. This clinical condition has several negative outcomes, including

increased sensitivity of teeth, esthetic grievance, and susceptibility to root caries. It is important to delineate the prevalence, severity, and risk factors of this condition; however, only a few studies have investigated this.

Studies in United States adults show that 23% of 30- to 90-year-olds and 50% of 65- to 90-year-olds had $\geq$3 mm gingival recession and that 15% of 65- to 90-year-olds had $\geq$5 mm recession [39]. After adjusting for the bias due to partial recording, these rates correspond to approximately 35%, 75%, and 23%, respectively. High frequencies of gingival recession have also been reported in other populations [27,53]. In an urban South American population aged 14 years and older, 52% and 22% of the subjects had $\geq$3 mm and $\geq$5 mm recession, respectively, and about half of those 40 years and older had $\geq$5 mm recession [53]. These findings suggest that gingival recession is common in various populations.

Etiologic and risk factors of periodontal diseases

Chronic and aggressive periodontitis are multifactorial diseases [15,16,55]. Microorganisms and microbial products in dental plaque are the main etiologic factors responsible for the initiation of the inflammatory reaction leading to periodontal tissue loss [19,20]. Several other local and systemic factors, however, have been shown to play important modifying roles in enhancing the inflammatory or destructive effects of microorganisms.

The significance of different risk factors for the pathogenesis of disease and for the development of tissue loss may vary in different forms of periodontal diseases. Profound progress in risk assessment has been made in recent years, and studies suggest that certain factors possess true risk-modifying effects, whereas others are merely risk indicators or surrogates of other effects. Certain local factors can enhance dental plaque accumulation or influence the composition of the dental plaque biofilm and may thereby potentiate the harmful effects of plaque microorganisms [17,55–58]. In addition, the gingival inflammatory response to plaque accumulation seems to differ substantially among individuals [59].

Age, gender, and socioeconomic status

Epidemiologic studies show that the prevalence and severity of attachment loss increase with age [26,60–63]. It is unclear, however, whether this increase is attributed to an increased risk of destructive periodontitis in older individuals or due to the cumulative effect of time [26,60,64,65]. Several studies also show an association between gender and attachment loss in adults, with men having higher prevalence and severity of periodontal destruction than women [26,31,51,63,66]. Recent data suggest that this finding may be related to gender-dependent genetic predisposing factors [67] or other sociobehavioral factors. Socioeconomic status is an important risk

indicator of periodontal disease, in that individuals with low socioeconomic status have a higher occurrence of attachment loss and probing depth than those with high socioeconomic status [68–72].

Race/ethnicity

The level of attachment loss is also influenced by race/ethnicity, although the exact role of this factor is not fully understood. Certain racial/ethnic groups, particularly subjects of African and Latin American background, have a higher risk of developing periodontal tissue loss than other groups. In the United States population, subjects of African or Mexican heritage have greater attachment loss than Caucasians [31]. The association of periodontal disease with race/ethnicity is significantly attenuated when certain effects such as cigarette smoking and income are accounted for [73]. This effect modification suggests that certain racial/ethnic characteristics are indicators of or confounded by certain other effects. For instance, African Americans generally have lower socioeconomic status than Caucasians. Hence, the increased risk of periodontitis in certain racial/ethnic groups may be partly attributed to socioeconomic, behavioral, and other disparities [74]. On the other hand, there is evidence that increased risk may also be partly related to biologic/genetic predisposition [75–78].

Specific microorganisms

Although there is sufficient evidence that accumulation and maturation of a plaque biofilm is necessary for the initiation and progression of periodontal diseases, studies show that bacterial species colonizing the gingival pocket play variable roles in the pathogenesis of these diseases and may therefore possess different levels of risk of periodontal tissue loss [79]. For instance, the microorganism *Actinobacillus actinomycetemcomitans* has often been identified in the subgingival sites in persons with severe attachment loss or rapid disease progression [80–82]. This bacterium was detected in about 50% of sites with new incidence of radiographic bone loss in young subjects followed over 8 years [83]. Furthermore, specific genetic variants of *A actinomycetemcomitans* possessing significantly enhanced leukotoxin production have been shown to significantly correlate with a higher prevalence of aggressive periodontitis mainly in individuals of African descent [76,78].

There is also evidence that *Porphyromonas gingivalis* is strongly associated with chronic periodontitis [80]. This bacterium is consistently detected in high levels in persons with severe attachment loss, deep probing depth, or with rapidly progressive disease [84,85]. Cross-sectional studies showed that presence of *P gingivalis* and *Tannerella forsythensis* (*Bacteroides forsythus*) in the subgingival flora was associated with significant increased risks for periodontal tissue loss. In one study, the odds ratios of the presence of the two respective species with clinical attachment loss

and severe bone loss were 1.7 and 2.5 [86,87], whereas the reported risks were significantly higher in another study (odds ratios: 12.3 and 10.4, respectively) [88].

Active infections with human cytomegalovirus and other herpesviruses have been proposed as possible risk factors for destructive periodontal diseases, including chronic periodontitis, aggressive periodontitis, and necrotizing periodontal diseases [89]. One study found that presence of herpesviruses in subgingival sites was associated with subgingival colonization of these sites with periodontopathic bacteria and with a threefold to fivefold increased risk of severe chronic periodontitis [90].

Oral hygiene

Oral hygiene has been consistently associated with higher occurrence of periodontal diseases in various populations [26,64,73]. A study of the natural history of periodontal diseases showed that attachment loss in a Sri Lankan population was significantly associated with dental calculus [91]. In addition, there is evidence that sites with gingival inflammation show a higher progression of attachment loss than sites without gingival inflammation [43,92]. Comprehensive oral hygiene programs are effective in preventing or reducing the level of gingival inflammation in children and adults [93,94]. These programs, however, may not be viable in preventing aggressive periodontitis [95], and it may be difficult to achieve a satisfactory level of oral hygiene in the general population to prevent chronic periodontitis and periodontal tissue loss effectively [51,96].

Smoking

Smoking behaviors have consistently been associated with attachment loss in most studies [55,97]. Smokers have a significantly higher risk of developing chronic periodontal disease [70,73,86,98] and show a higher rate of periodontal destruction over time than nonsmokers [71,99]. There is a dose-effect relationship between cigarette smoking and the severity of periodontal disease such that heavy smokers and those with a longer history of smoking show more severe tissue loss than light smokers [98,100]. Generally, studies show that cigarette smoking is associated with a twofold to sevenfold increased risk of having attachment loss compared with nonsmokers, with a more pronounced risk in young smokers [86,87, 97,99,101,102]. The population risk due to cigarette smoking has been studied in large surveys and it is estimated that in the United States population, approximately 42% and 11% of periodontitis cases may be attributed to current and former cigarette smoking, respectively [98]. A recent survey in Brazilian adults estimated that 12% of periodontitis cases may be attributable to cigarette smoking [103]. Cigar and pipe smoking have been shown to have detrimental effects on periodontal health similar to those attributed to cigarette smoking [104,105].

Systemic diseases

Certain systemic diseases have been associated with an increased risk of attachment loss. The association between diabetes mellitus and periodontal diseases has been studied extensively, and the evidence suggests that diabetics have considerably higher risk of attachment loss than nondiabetics [55,106–108]. There is incomplete information about the relationship of periodontal tissue loss with other systemic diseases and conditions such as osteopenia and osteoporosis [109,110] and arthritis [56,111]. Certain systemic diseases such as AIDS [112] and Down syndrome [113] are significant risk factors for periodontal disease, and a high percentage of cases show moderate to advanced periodontitis.

Gene polymorphisms

Several gene polymorphisms have been investigated, some of which have been shown to be associated with an increased risk of periodontitis [114–117]. Various genetic risk factors, however, may explain only a part of the variance in the occurrence of periodontitis [118]. In addition, significant interactions seem to exist between genetic, environmental, and demographic factors [15,55]. For example, there are data suggesting a modifying effect of smoking and variability in the occurrence of certain genotypes in different racial/ethnic groups. Hence, more studies are needed to better understand the role of these and other factors in the increased susceptibility to destructive periodontal diseases.

Summary

Periodontal surveys enable the determination of the prevalence, extent, and severity of periodontal diseases in populations and can generally be conducted in a reasonable time frame at a relatively moderate cost. The main disadvantage of surveys is that they provide only a ''snapshot in time'' of the occurrence in the population of the disease and of various exposures (potential risk factors) associated with the disease. Therefore, surveys do not provide convincing proof of causal relationships. All inferences derived from these surveys must take into account the potential confounding effects from correlated, noncausal study variables, which can lead to spurious inferences. In addition, the lack of standardization of study design, definition of disease, and methods of disease detection and measurement may significantly limit the use of results of population studies of periodontal diseases. The significance of using clear definitions of diseases and properly described measurement parameters in epidemiologic studies cannot be overemphasized because this practice helps facilitate reproduction and validation of study findings by other independent investigators.

References

[1] Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999;4(1):1–6.
[2] Ciancio SG. Current status of indices of gingivitis. J Clin Periodontol 1986;13(5):375–82.
[3] Flemmig TF. Periodontitis. Ann Periodontol 1999;4(1):32–8.
[4] Suzuki JB. Diagnosis and classification of the periodontal diseases. Dent Clin N Am 1988; 32(2):195–216.
[5] Lindhe J, Ranney R, Lamster I, et al. Consensus report: chronic periodontitis. Ann Periodontol 1999;4(1):38.
[6] Albandar JM. A 6-year study on the pattern of periodontal disease progression. J Clin Periodontol 1990;17(7 Pt 1):467–71.
[7] Albandar JM, Rise J, Gjermo P, et al. Radiographic quantification of alveolar bone level changes. A 2-year longitudinal study in man. J Clin Periodontol 1986;13(3):195–200.
[8] Socransky SS, Haffajee AD, Goodson JM, et al. New concepts of destructive periodontal disease. J Clin Periodontol 1984;11(1):21–32.
[9] Lang N, Bartold M, Cullinan M, et al. Consensus report: aggressive periodontitis. Ann Periodontol 1999;4(1):53.
[10] Albandar JM, Brown LJ, Löe H. Clinical features of early-onset periodontitis. J Am Dent Assoc 1997;128(10):1393–9.
[11] Albandar JM, Brown LJ, Genco RJ, et al. Clinical classification of periodontitis in adolescents and young adults. J Periodontol 1997;68(6):545–55.
[12] Tonetti MS, Mombelli A. Early-onset periodontitis. Ann Periodontol 1999;4(1):39–53.
[13] Lindhe J, Ranney R, Lamster I, et al. Consensus report: periodontitis as a manifestation of systemic diseases. Ann Periodontol 1999;4:64.
[14] Lang N, Soskolne WA, Greenstein G, et al. Consensus report: necrotizing periodontal diseases. Ann Periodontol 1999;4:78.
[15] Albandar JM, Rams TE. Risk factors for periodontitis in children and young persons. Periodontol 2000 2002;29:207–22.
[16] Nunn ME. Understanding the etiology of periodontitis: an overview of periodontal risk factors. Periodontol 2000 2003;32:11–23.
[17] Ezzo PJ, Cutler CW. Microorganisms as risk indicators for periodontal disease. Periodontol 2000 2003;32:24–35.
[18] Socransky SS, Haffajee AD. Dental biofilms: difficult therapeutic targets. Periodontol 2000 2002;28:12–55.
[19] Lindhe J, Hamp SE, Löe H. Plaque-induced periodontal disease in beagle dogs. J Periodontal Res 1975;10:243–55.
[20] Löe H, Theilade E, Jensen SB. Experimental gingivitis in man. J Periodontol 1965;36: 177–87.
[21] Kinane DF, Lappin DF. Clinical, pathological and immunological aspects of periodontal disease. Acta Odontol Scand 2001;59(3):154–60.
[22] Kingman A, Albandar JM. Methodological aspects of epidemiological studies of periodontal diseases. Periodontol 2000 2002;29:11–30.
[23] Newbrun E. Indices to measure gingival bleeding. J Periodontol 1996;67(6):555–61.
[24] Russell AL. A system of classification and scoring for prevalence surveys of periodontal disease. J Dent Res 1956;35:350–9.
[25] Ramfjord SP. Indices for prevalence and incidence of periodontal disease. J Periodontol 1959;30:51–9.
[26] Albandar JM. Periodontal diseases in North America. Periodontol 2000 2002;29:31–69.
[27] Baelum V, Scheutz F. Periodontal diseases in Africa. Periodontol 2000 2002;29:79–103.
[28] Ainamo J, Barmes D, Beagrie G, et al. Development of the World Health Organization (WHO) Community Periodontal Index of Treatment Needs (CPITN). Int Dent J 1982;32: 281–91.

[29] World Health Organization. Oral health surveys. Basic methods. Geneva (Switzerland): World Health Organization; 1997.
[30] Baelum V, Papapanou PN. CPITN and the epidemiology of periodontal disease. Community Dent Oral Epidemiol 1996;24(6):367–8.
[31] Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988–1994. J Periodontol 1999;70(1):13–29.
[32] Susin C, Kingman A, Albandar JM. Effect of partial recording protocols on estimates of prevalence of periodontal disease. J Periodontol 2005;76:262–7.
[33] Albandar JM, Brown LJ, Brunelle JA, et al. Gingival state and dental calculus in early-onset periodontitis. J Periodontol 1996;67(10):953–9.
[34] Cunha ACP, Chambrone LA. Prevalência de gengivite em criancas. Revista Periodontia 1998;7:1–5.
[35] Albandar JM, Tinoco EM. Global epidemiology of periodontal diseases in children and young persons. Periodontol 2000 2002;29:153–76.
[36] Cunha ACP, Chambrone LA. [Prevalência de gengivite em criancas de um nivel social baixo.] Revista Periodontia 1998;7:6–10 [in Portugese].
[37] Gjermo P, Rosing CK, Susin C, et al. Periodontal diseases in Central and South America. Periodontol 2000 2002;29:70–8.
[38] Petersen PE, Kaka M. Oral health status of children and adults in the Republic of Niger, Africa. Int Dent J 1999;49(3):159–64.
[39] Albandar JM, Kingman A. Gingival recession, gingival bleeding, and dental calculus in adults 30 years of age and older in the United States, 1988–1994. J Periodontol 1999;70(1): 30–43.
[40] Oliver RC, Brown LJ, Löe H. Periodontal diseases in the United States population. J Periodontol 1998;69(2):269–78.
[41] Lang NP, Adler R, Joss A, et al. Absence of bleeding on probing. An indicator of periodontal stability. J Clin Periodontol 1990;17(10):714–21.
[42] Haffajee AD, Socransky SS, Goodson JM. Clinical parameters as predictors of destructive periodontal disease activity. J Clin Periodontol 1983;10(3):257–65.
[43] Albandar JM, Kingman A, Brown LJ, et al. Gingival inflammation and subgingival calculus as determinants of disease progression in early-onset periodontitis. J Clin Periodontol 1998;25(3):231–7.
[44] Albandar JM, Rams TE. Global epidemiology of periodontal diseases: an overview. Periodontol 2000 2002;29:7–10.
[45] Albandar JM. Prevalence of incipient radiographic periodontal lesions in relation to ethnic background and dental care provisions in young adults. J Clin Periodontol 1989;16(10): 625–9.
[46] Löe H, Brown LJ. Early onset periodontitis in the United States of America. J Periodontol 1991;62(10):608–16.
[47] Beck JD, Koch GG, Rozier RG, et al. Prevalence and risk indicators for periodontal attachment loss in a population of older community-dwelling blacks and whites. J Periodontol 1990;61(8):521–8.
[48] Douglass CW, Jette AM, Fox CH, et al. Oral health status of the elderly in New England. J Gerontol 1993;48(2):M39–46.
[49] Fox CH, Jette AM, McGuire SM, et al. Periodontal disease among New England elders. J Periodontol 1994;65(7):676–84.
[50] Sheiham A, Netuveli GS. Periodontal diseases in Europe. Periodontol 2000 2002;29: 104–21.
[51] Morris AJ, Steele J, White DA. The oral cleanliness and periodontal health of UK adults in 1998. Br Dent J 2001;191(4):186–92.
[52] Susin C, Dalla Vecchia CF, Oppermann RV, et al. Periodontal attachment loss in an urban population of Brazilian adults, and the effect of demographic, behavioral and environmental risk indicators. J Periodontol 2004;75(7):1033–41.

[53] Susin C, Haas AN, Oppermann RV, et al. Gingival recession: epidemiology and risk indicators in a representative urban Brazilian population. J Periodontol 2004;75(10): 1377–86.
[54] Corbet EF, Zee KY, Lo EC. Periodontal diseases in Asia and Oceania. Periodontol 2000 2002;29:122–52.
[55] Albandar JM. Global risk factors and risk indicators for periodontal diseases. Periodontol 2000 2002;29:177–206.
[56] Albandar JM. Some predictors of radiographic alveolar bone height reduction over 6 years. J Periodontal Res 1990;25(3):186–92.
[57] Albandar JM, Buischi YA, Axelsson P. Caries lesions and dental restorations as predisposing factors in the progression of periodontal diseases in adolescents. A 3-year longitudinal study. J Periodontol 1995;66(4):249–54.
[58] Kinane DF. Causation and pathogenesis of periodontal disease. Periodontol 2000 2001;25: 8–20.
[59] Tatakis DN, Trombelli L. Modulation of clinical expression of plaque-induced gingivitis. I. Background review and rationale. J Clin Periodontol 2004;31(4):229–38.
[60] Papapanou PN, Lindhe J, Sterrett JD, et al. Considerations on the contribution of ageing to loss of periodontal tissue support. J Clin Periodontol 1991;18(8):611–5.
[61] Yoneyama T, Okamoto H, Lindhe J, et al. Probing depth, attachment loss and gingival recession. Findings from a clinical examination in Ushiku, Japan. J Clin Periodontol 1988; 15(9):581–91.
[62] Burt BA. Periodontitis and aging: reviewing recent evidence. J Am Dent Assoc 1994;125(3): 273–9.
[63] Corbet EF, Wong MC, Lin HC. Periodontal conditions in adult Southern Chinese. J Dent Res 2001;80(5):1480–5.
[64] 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(1): 13–8.
[65] Ship JA, Beck JD. Ten-year longitudinal study of periodontal attachment loss in healthy adults. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81(3):281–90.
[66] Grossi SG. Effect of estrogen supplementation on periodontal disease. Compend Contin Educ Dent Suppl 1998;22:S30–6.
[67] Reichert S, Stein J, Gautsch A, et al. Gender differences in HLA phenotype frequencies found in German patients with generalized aggressive periodontitis and chronic periodontitis. Oral Microbiol Immunol 2002;17(6):360–8.
[68] Borrell LN, Burt BA, Gillespie BW, et al. Periodontitis in the United States: beyond black and white. J Public Health Dent 2002;62(2):92–101.
[69] Drury TF, Garcia I, Adesanya M. Socioeconomic disparities in adult oral health in the United States. Ann N Y Acad Sci 1999;896:322–4.
[70] Dolan TA, Gilbert GH, Ringelberg ML, et al. Behavioral risk indicators of attachment loss in adult Floridians. J Clin Periodontol 1997;24(4):223–32.
[71] Elter JR, Beck JD, Slade GD, et al. Etiologic models for incident periodontal attachment loss in older adults. J Clin Periodontol 1999;26(2):113–23.
[72] 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(2):96–103.
[73] Hyman JJ, Reid BC. Epidemiologic risk factors for periodontal attachment loss among adults in the United States. J Clin Periodontol 2003;30(3):230–7.
[74] Poulton R, Caspi A, Milne BJ, et al. Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. Lancet 2002;360(9346): 1640–5.
[75] Schenkein HA, Best AM, Gunsolley JC. Influence of race and periodontal clinical status on neutrophil chemotactic responses. J Periodontal Res 1991;26(3 Pt 2):272–5.

[76] Haubek D, Ennibi OK, Abdellaoui L, et al. Attachment loss in Moroccan early onset periodontitis patients and infection with the JP2-type of *Actinobacillus actinomycetemcomitans*. J Clin Periodontol 2002;29(7):657–60.
[77] Albandar JM, DeNardin AM, Adesanya MR, et al. Associations of serum concentrations of IgG, IgA, IgM and interleukin-1beta with early-onset periodontitis classification and race. J Clin Periodontol 2002;29(5):421–6.
[78] DiRienzo JM, Slots J, Sixou M, et al. Specific genetic variants of *Actinobacillus actinomycetemcomitans* correlate with disease and health in a regional population of families with localized juvenile periodontitis. Infect Immun 1994;62(8):3058–65.
[79] Wolff L, Dahlen G, Aeppli D. Bacteria as risk markers for periodontitis. J Periodontol 1994;65(Suppl 5):498–510.
[80] Slots J, Ting M. *Actinobacillus actinomycetemcomitans* and *Porphyromonas gingivalis* in human periodontal disease: occurrence and treatment. Periodontol 2000 1999;20: 82–121.
[81] Slots J. The predominant cultivable organisms in juvenile periodontitis. Scand J Dent Res 1976;84:1–10.
[82] Slots J. Bacterial specificity in adult periodontitis. A summary of recent work. J Clin Periodontol 1986;13(10):912–7.
[83] Aass AM, Rossow I, Preus HR, et al. Incidence of early periodontitis in a group of young individuals during 8 years: associations with selected potential predictors. J Periodontol 1994;65(9):814–9.
[84] Albandar JM, Olsen I, Gjermo P. Associations between six DNA probe-detected periodontal bacteria and alveolar bone loss and other clinical signs of periodontitis. Acta Odontol Scand 1990;48(6):415–23.
[85] Albandar JM, Brown LJ, Löe H. Putative periodontal pathogens in subgingival plaque of young adults with and without early-onset periodontitis. J Periodontol 1997;68(10): 973–81.
[86] Grossi SG, Zambon JJ, Ho AW, et al. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. J Periodontol 1994;65(3):260–7.
[87] 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(1):23–9.
[88] van Winkelhoff AJ. Loos BG, van der Reijden WA, et al. *Porphyromonas gingivalis*, *Bacteroides forsythus* and other putative periodontal pathogens in subjects with and without periodontal destruction. J Clin Periodontol 2002;29(11):1023–8.
[89] Kamma JJ, Slots J. Herpesviral-bacterial interactions in aggressive periodontitis. J Clin Periodontol 2003;30(5):420–6.
[90] Contreras A, Umeda M, Chen C, et al. Relationship between herpesviruses and adult periodontitis and periodontopathic bacteria. J Periodontol 1999;70(5):478–84.
[91] Neely AL, Holford TR, Loe H, et al. The natural history of periodontal disease in man. Risk factors for progression of attachment loss in individuals receiving no oral health care. J Periodontol 2001;72(8):1006–15.
[92] Schatzle M, Loe H, Burgin W, et al. Clinical course of chronic periodontitis. I. Role of gingivitis. J Clin Periodontol 2003;30(10):887–901.
[93] Albandar JM, Buischi YA, Mayer MP, et al. Long-term effect of two preventive programs on the incidence of plaque and gingivitis in adolescents. J Periodontol 1994; 65(6):605–10.
[94] Axelsson P, Lindhe J, Nystrom B. On the prevention of caries and periodontal disease. Results of a 15-year longitudinal study in adults. J Clin Periodontol 1991;18(3):182–9.
[95] Albandar JM, Buischi YA, Oliveira LB, et al. Lack of effect of oral hygiene training on periodontal disease progression over 3 years in adolescents. J Periodontol 1995;66(4): 255–60.
[96] Löe H. Oral hygiene in the prevention of caries and periodontal disease. Int Dent J 2000; 50(3):129–39.

[97] Gelskey SC. Cigarette smoking and periodontitis: methodology to assess the strength of evidence in support of a causal association. Community Dent Oral Epidemiol 1999;27(1): 16–24.
[98] Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. J Periodontol 2000; 71(5):743–51.
[99] Bergstrom J, Eliasson S, Dock J. A 10-year prospective study of tobacco smoking and periodontal health. J Periodontol 2000;71(8):1338–47.
[100] Jette AM, Feldman HA, Tennstedt SL. Tobacco use: a modifiable risk factor for dental disease among the elderly. Am J Public Health 1993;83(9):1271–6.
[101] Bergstrom J. Tobacco smoking and risk for periodontal disease. J Clin Periodontol 2003; 30(2):107–13.
[102] Linden GJ, Mullally BH. Cigarette smoking and periodontal destruction in young adults. J Periodontol 1994;65(7):718–23.
[103] Susin C, Oppermann RV, Haugejorden O, et al. Periodontal attachment loss attributable to cigarette smoking in an urban Brazilian population. J Clin Periodontol 2004;31:951–9.
[104] Krall EA, Garvey AJ, Garcia RI. Alveolar bone loss and tooth loss in male cigar and pipe smokers. J Am Dent Assoc 1999;130(1):57–64.
[105] Albandar JM, Streckfus CF, Adesanya MR, et al. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. J Periodontol 2000;71(12):1874–81.
[106] Lalla E, Lamster IB, Feit M, et al. Blockade of RAGE suppresses periodontitis-associated bone loss in diabetic mice. J Clin Invest 2000;105(8):1117–24.
[107] Taylor GW. Periodontal treatment and its effects on glycemic control: a review of the evidence. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87(3):311–6.
[108] Thomson WM, Slade GD, Beck JD, et al. Incidence of periodontal attachment loss over 5 years among older South Australians. J Clin Periodontol 2004;31(2):119–25.
[109] Payne JB, Reinhardt RA, Nummikoski PV, et al. Longitudinal alveolar bone loss in postmenopausal osteoporotic/osteopenic women. Osteoporos Int 1999;10(1):34–40.
[110] Reinhardt RA, Payne JB, Maze CA, et al. Influence of estrogen and osteopenia/ osteoporosis on clinical periodontitis in postmenopausal women. J Periodontol 1999;70(8): 823–8.
[111] Miranda LA, Fischer RG, Sztajnbok FR, et al. Periodontal conditions in patients with juvenile idiopathic arthritis. J Clin Periodontol 2003;30(11):969–74.
[112] Alpagot T, Duzgunes N, Wolff LF, et al. Risk factors for periodontitis in HIV patients. J Periodontal Res 2004;39(3):149–57.
[113] Agholme MB, Dahllof G, Modeer T. Changes of periodontal status in patients with Down syndrome during a 7-year period. Eur J Oral Sci 1999;107(2):82–8.
[114] Cullinan MP, Westerman B, Hamlet SM, et al. A longitudinal study of interleukin-1 gene polymorphisms and periodontal disease in a general adult population. J Clin Periodontol 2001;28(12):1137–44.
[115] Michalowicz BS, Diehl SR, Gunsolley JC, et al. Evidence of a substantial genetic basis for risk of adult periodontitis. J Periodontol 2000;71(11):1699–707.
[116] Li Y, Xu L, Hasturk H, et al. Localized aggressive periodontitis is linked to human chromosome 1q25. Hum Genet 2004;114(3):291–7.
[117] Noack B, Gorgens H, Hoffmann T, et al. Novel mutations in the cathepsin C gene in patients with pre-pubertal aggressive periodontitis and Papillon-Lefevre syndrome. J Dent Res 2004;83(5):368–70.
[118] Diehl SR, Wang Y, Brooks CN, et al. Linkage disequilibrium of interleukin-1 genetic polymorphisms with early-onset periodontitis. J Periodontol 1999;70(4):418–30.