Epidemiology of Periodontal Diseases*

This paper was prepared by the Research, Science and Therapy Committee of the American Academy of Periodontology and is intended for the information of the dental profession. It represents the position of the Academy in regard to the current state of knowledge about the epidemiology of periodontal diseases. This paper, with issues examined from the epidemiological viewpoint, is intended to give practitioners an epidemiological perspective on issues of interest to them. It replaces the version published in 1996. J Periodontol 2005;76:1406-1419.

Epidemiology is the study of health and disease in populations and of how these states are influenced by heredity, biology, physical environment, social environment, and personal behavior. Analytical epidemiology seeks to identify the risk factors associated with a disease, to quantify the strength of those associations, and to estimate whether an association is causal. An understanding of risk factors can lead to theories of causation and then to treatment protocols for clinicians to use with their patients. The essential features of epidemiology as a method of research, when compared to clinical research and case studies, are that 1) groups rather than individuals are the focus of study and 2) persons with and without a particular disease (e.g., periodontal diseases), and with and without the exposure of interest are included, rather than just patients. The study of population groups rather than individuals is to allow for valid estimates while accounting for normal biological variation (e.g., some individuals form plaque readily, others do not). Broadening a study to include those without disease, as well as those with it, provides a reference point against which to quantify risk.

Advances in research over recent years have led to a fundamental change in our understanding of the periodontal diseases. As recently as the mid-1960s, the prevailing model for the epidemiology of periodontal diseases included these precepts: 1) all individuals were considered more or less equally susceptible to severe periodontitis; 2) gingivitis usually progressed to periodontitis with consequent loss of bony support and eventually loss of teeth; and 3) susceptibility to periodontitis increased with age and was the main cause of tooth loss after age 35.1-4 Advances in our understanding of periodontal diseases since that time have led to this old disease model being reevaluated.

This review concentrates on recent research in the epidemiology of chronic periodontitis. It will assess current knowledge on prevalence, incidence, severity, risk factors, and predicting disease risk. The review does not directly address microbial infection and host response mechanisms, modes of disease progression (i.e., bursts or linear), links between periodontitis and systemic diseases, the specifics of less common clinically-recognized conditions such as aggressive periodontitis, and conditions associated with hematological or genetic disorders.

GINGIVITIS: PREVALENCE AND DISTRIBUTION

National survey data show that gingivitis is found in early childhood, is more prevalent and severe in adolescence, and then tends to level off in older age groups.5 The prevalence of gingivitis among schoolchildren in the United States has ranged from 40% to 60% in national surveys.6,7 In the national survey of employed adults in 1985-86, 47% of males and 39% of females aged 18 to 64 exhibited at least one site which bled on probing.8 The mean number of bleeding sites per person was higher in older than in younger males, but this was not seen in females. In the first U.S. national survey of adults in 1960-62, which scored gingivitis visually, 85% of men and 79% of women were found to have some degree of gingivitis.9 In the third National Health and Nutrition Examination Survey (NHANES III, 1988-94), 50% of adults were found to have gingivitis on at least three or four teeth.10 Even allowing for the differences in measurement techniques between the two surveys, there appears to have been an improvement in gingival health over that 25-year period.

Plaque deposits are closely correlated with gingivitis, a relationship long considered one of cause-and-effect. Longitudinal studies among Norwegian professionals and students, among whom oral hygiene was excellent, found no increase in prevalence and severity of gingivitis between the late teen years and age 40.11 In a related study among Sri Lankan tea workers, both oral hygiene and the gingival condition were poorer at all ages.12 Studies among other populations in developing...
countries show that gingivitis, associated with extensive plaque and calculus deposits, is the norm among adults.13,15

Research dating back to the 1980s has shown that relatively few sites with gingivitis go on to develop periodontitis.16-21 Even so, prevention of gingivitis, in the individual patient or in populations, is still the first step toward preventing periodontitis. On the question of why some gingival sites make the transition from being periodontally healthy to those which bleed on probing, there are indications that more than just plaque accumulations are involved.22 There may be an age relationship; a more pronounced inflammatory response to the same plaque challenge has been reported in older than in younger people.23 A genetically determined response has also been suggested, with non-smoking or former-smoking interleukin (IL)-1 genotype-positive individuals having greater risk of bleeding on probing (BOP) than non-smoking and former-smoking IL-1 negatives.24 For example, in the study just described, when the association between the IL-1 genotype and BOP was tested in a large population which included smokers, the significant associations disappeared because of what the authors called “the overriding effects of smoking.” Smokers also appear to be at higher risk than non-smokers of making the transition from gingiva that do not bleed on probing to those that do.25 There are some differences among researchers about the effects of smoking on gingival bleeding, a subject that will be visited later in this report. Stress has also been associated with increasing IL-1β levels.26 Oral contraceptives have long been considered a risk factor for gingival bleeding27 and some still are.28 But with the lower dosages now generally used, that risk has diminished, and modern oral contraceptives may not affect the inflammatory response of the gingiva to dental plaque.29

**MEASUREMENT OF PERIODONTITIS**

The basic clinical measures for periodontitis, apart from gingival bleeding and radiographic assessment of bone loss, are clinical attachment loss (CAL) and probing depth (PD). The standard protocol used today for measuring CAL and PD with a manual probe was first described more than 45 years ago30 and has not changed much since. Various scaled indexes have been used in the past, but these were “composite” indexes which scored gingivitis and periodontitis on the same scale. Composite indexes are now considered invalid and have thus been discarded. Although CAL, a measure of accumulated past disease at a site rather than current activity, remains a diagnostic “gold standard” for periodontitis,31 the absence of consensus on how best to incorporate CAL and PD into a case definition of periodontitis continues to hamper clinical and epidemiological research. Studies have measured CAL and PD on all teeth, all teeth in two quadrants, the worst teeth in each sextant, and selected index teeth. Measurements have been made on six, four, two, and one sites per tooth. As one illustration of the problems that follow this lack of uniformity, it has been suggested32 that the 1985-86 National Survey of Oral Health in U.S. Employed Adults and Seniors8 may have underestimated the national prevalence of periodontitis because it measured only two sites per tooth (mesiobuccal and mid-buccal) in one maxillary and one mandibular quadrant. Furcation and lingual areas, the places where disease is considered most likely to develop, were not included in the survey protocol. A case definition for periodontitis needs to establish 1) what depth of CAL at any one site constitutes evidence of disease processes; 2) how many such sites need to be present in a mouth to establish disease presence; and 3) how to include probing measurements and BOP in the case definition. The first issue also has to make allowance for examiner variation, which can confuse efforts to measure CAL progression. Even though measurements of probing depth are repeatable to within 1 mm more than 90% of the time,33 the standard deviation of repeated CAL measurements of the same site by an experienced examiner with a manual probe is around 0.8 mm.34 Accordingly, change in attachment level in a clinical study needs to be at least 2 mm (i.e., two to three times the standard deviation) before the investigators can be confident that they are seeing real change rather than measurement error.35,36 CAL progression of at least 3 mm over a given time period has been the criterion for change in other studies.37,38

A high proportion of even young adults have at least one site with 2 mm CAL.39 Because this level of CAL is so common it is not satisfactory to use it to define periodontitis in a cross-sectional study. An approach like the Extent and Severity Index,40 in which “extent” refers to the number of teeth in the mouth with CAL of ≥1 mm and “severity” is the mean CAL for those teeth, might be appropriate in some circumstances, although the CAL cutoff limit of 1 mm needs to be increased for the reasons of examiner reliability discussed above. Some consensus on age-related case definitions for “serious” and “moderate” disease would also assist research. A number of studies have used their own case definitions for “serious” disease, mostly based on combinations of CAL and PD or extent of bone loss,12,41-46 but no uniformity has yet emerged.
The inherent measurement problems have led researchers to look for markers of periodontitis which, if valid and reliable, would decrease our dependence upon clinical measures based on probing for diagnosing disease. As our understanding of periodontitis etiology has deepened, some markers have emerged as likely candidates. The most promising are the inflammatory cytokines that are expressed in gingival crevicular fluid (GCF) as part of the host response to inflammation, a number of which have been associated with active disease.47,48 These cytokines include prostaglandin E₂ (PGE₂), tumor necrosis factor-alpha (TNF-α), IL-1 alpha (IL-1α), IL-1 beta (IL-1β), and others. While it has been documented for some time that these and other constituents of GCF are associated with inflammatory response, actually quantifying these associations and determining the sensitivity of the measures (i.e., the extent to which the quantity of expressed cytokine goes up or down as inflammation goes up and down) is proving more difficult. One cross-sectional study found greater quantities of PGE₂ expressed by persons with gingivitis only than by those with gingivitis plus untreated periodontitis.49 The researchers attributed this finding to the chronic gingivitis present, which, if true, would diminish the value of this test for periodontitis. The enzyme aspartate aminotransferase (AST), which has been identified as present in the GCF of periodontitis patients, also has been studied. Initial tests have been promising,50 and AST can be identified by a simple chairside test. To date, however, these approaches to measure periodontitis by means of inflammatory cytokines in GCF are still being tested. It will be a distinct help to both clinicians and researchers if one or more of them can become established as valid and reliable measures of active periodontitis.

THE PREVALENCE OF PERIODONTAL DISEASES

Prevalence is the number of cases of a disease in a designated population at a given point.51 Our best information on the prevalence of numerous conditions, including periodontal diseases, comes from the results of national surveys of representative samples conducted by the National Center for Health Statistics and the National Institute of Dental and Craniofacial Research, with additional data from smaller scale surveys of specific, non-representative groups. Any prevalence information must be interpreted in light of the population studied and the periodontitis case definition applied.

The old model of periodontal diseases, described earlier, held that susceptibility to periodontal diseases was virtually universal. Today, however, it is well documented that only some 5% to 15% of any population suffers from severe generalized periodontitis, even though moderate disease affects a majority of adults.8,39,52 This clustering of serious disease in a subset of the population has been recorded among well-treated patients20,53-55 as well as in epidemiologic studies of populations which do not receive modern dental care.13-15,56 What epidemiology has demonstrated is that the majority of just about any adult population has chronic periodontitis to some degree, but that mild attachment loss, as measured by CAL of 2 mm or so, is compatible with good health and function for many years.

Periodontitis, viewed for years as primarily the outcome from infection, is now seen as resulting from a complex interplay between bacterial infection and host response, often modified by behavioral factors.57 The host response is now seen as a key factor in the clinical expression of periodontitis,58 with only some 20% of periodontal diseases now attributed to bacterial variance.57 Some 50% of periodontal diseases have been attributed to genetic variance and more than 20% to tobacco use,57 although the role of tobacco has also been estimated as higher.59

Determining the prevalence of periodontitis in the U.S. population, seemingly a straightforward issue, in fact is complicated by the various case definitions used. If periodontitis is defined as the identification of at least one site with CAL of ≥2 mm, around 80% of all adults are affected, and around 90% of those aged 55 to 64.8,39 When the case definition is at least one site with CAL of ≥4 mm, the prevalence in those aged 55 to 64 drops to around 50%. When it is CAL of ≥6 mm, prevalence is less than 20%.39 Using pockets of ≥4 mm as a case definition, 30% of adults had met that criterion on at least three to four teeth.52 When measured at population level and without adjustment for possible confounders, prevalence is greater in African-Americans50 and in Native Americans.61

These national data make it clear that the milder forms of periodontitis are close to universal. The more severe manifestations of the disease, meaning those that lead to tooth loss or at least threaten it, are less prevalent. Even just these few data demonstrate that any prevalence data need the reference markers of the relevant case definition and the age group to which they apply.

INCIDENCE OF PERIODONTITIS

Incidence is defined as the number of new cases in a population over a given time period.51 In public health practice, “cases” means people, so that when applied to a tuberculosis outbreak, for example, it will mean the
number of new people diagnosed with the condition during a stated time period. In periodontitis, “incidence” is often taken to mean new sites that meet a definition of periodontitis, even if these occur in people who already have other diseased sites. The term is also applied to an increase in CAL or bone loss in a site that has already been recorded as diseased. As with prevalence, measures of periodontitis incidence will vary according to the case definition of the disease. The more severe the extent of CAL or bone loss that is defined as incidence, the lower will be the incidence of periodontitis recorded.62

Longitudinal studies are logistically and intellectually demanding, but are required if incidence is to be measured. Some longitudinal studies have confirmed the results of cross-sectional studies in identifying age as a risk factor for progression of CAL.63,64 Although others37,65 concluded that progression of CAL is more closely related to the extent of baseline CAL than to age. Healthy older people in the Baltimore Longitudinal Study of Aging did not show much attachment loss even over a 10-year period.66

In the Piedmont study in North Carolina, which followed a population sample aged 65 to over 80 for 3 years, baseline findings were that CAL was much more extensive in study participants than it was in younger groups.43 These and subsequent reports from the Piedmont study38,67,68 have made a substantial contribution to our understanding of the natural history of periodontitis. Using 3 mm as an estimate of CAL, it was found that African-Americans were more likely to experience incident CAL than whites, and about half of the whole group experienced at least one site with 3 mm CAL over the first 18 months.38 After 3 years of observations, it was found that only 12% experienced CAL in each of the two 18-month periods.67 It was also found that CAL during the first 18 months was related to subsequent CAL at the subject level, although not at the individual site level, a finding which supports the episodic, randomized model of periodontitis. Past disease predicted subsequent CAL, although not usually at the same site, and a previous disease episode did not put a site at higher risk for a subsequent episode.68

As would be expected, persons with greater degrees of attachment loss at baseline were also more likely to lose teeth over the next 5 years.68

A 15-year longitudinal study of 480 tea workers in Sri Lanka demonstrated a wide range of susceptibility to periodontitis.12 The group studied had virtually no dental treatment, so the data reflected the natural history of periodontitis. Based on tooth loss and interproximal CAL, it was concluded that about 8% demonstrated rapid progression of periodontitis, 81% showed moderate progression, and the remaining 11% showed no progression beyond gingivitis. In the rapid- and moderate-progression groups, periodontitis progressed with age (much more rapidly in the first group), whereas in the non-progressing group age was not a factor. This Sri Lanka study, like the Piedmont study, demonstrated that loss of CAL became severe over time only for a small group of susceptible individuals.

DETERMINANTS OF PERIODONTITIS

A risk factor is an environmental exposure, aspect of behavior, or an inherent characteristic which is associated with a disease.51 The association may or may not be causal, though the use of the term increasingly implies known or suspected causality. The term determinant is often used synonymously with risk factor in the literature, but for clarity is best reserved for risk factors that cannot be modified (e.g., age, previous disease experience). The term risk indicator describes plausible correlates of disease identified in cross-sectional studies, while risk factor is best applied to those correlates confirmed in longitudinal studies. The term risk factor implies a modifiable condition (e.g., smoking, plaque deposits). Risk indicators identified in cross-sectional studies are not always confirmed as risk factors in longitudinal studies.69 The term risk marker is used more in the predictive sense, a factor associated with increased probability of future disease but where causality is usually not implied.

Age

The prevalence and severity of CAL is invariably related directly to age in cross-sectional surveys. Taking the 1985-86 NIDR national survey of employed adults, the proportion of adults with at least one CAL site of ≥2 mm exceeded 70% even in adults aged 35 to 44 years, and was more than 90% in those aged 55 to 64.8 For ≥4 mm loss of attachment, prevalence was 13.8% of 25- to 34-year-olds and 53.6% of 55- to 64-year-olds. PD is also related to age, although less directly. The 1985-86 national survey found that pockets of 4 to 6 mm were present in 13.4% of all adults and were more frequent in older age groups.70 Pockets of ≥7 mm were found in only 0.6% of those examined and were not related to age.

The older assumption that periodontitis is a disease of aging is no longer tenable.71-75 The current view sees the greater periodontal destruction in the elderly as reflecting lifetime disease accumulation rather than an age-specific condition. A relatively low prevalence of severe (as opposed to moderate) CAL among the
Figure 1

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elderly was first shown in Sweden\textsuperscript{76} and has since been demonstrated elsewhere. Surveys of older people in the United States, Canada, and Australia have found that CAL or PD of 6 mm or more was prevalent in 15\% to 30\% of persons examined.\textsuperscript{44,77-79} In all of these studies, CAL of 4 to 6 mm was common. Higher estimates of periodontal destruction came from a cross-sectional New England study of community-living elderly people.\textsuperscript{80,81} The New England study was of persons aged 70 to 96, older than those in the 1985-86 national survey, and the results could reflect cohort effects (i.e., results specific to the generation studied and which may not be seen in subsequent generations). All of these reports agree that CAL increases with age, but most did not find extensive loss of function in the affected teeth.

Periodontitis seen in youth and early adulthood can probably be classified as aggressive periodontitis,\textsuperscript{82} and some degree of CAL in youth is well documented in population studies.\textsuperscript{83-92} These findings are from well-treated populations as well as those where modern dental care does not exist. It can be hypothesized that the more susceptible members of the population are those in whom periodontitis begins in youth. If that is so, then the relatively low prevalence of severe CAL among many dentate elderly could be partly a survival phenomenon, meaning that those most susceptible to severe periodontitis have already lost teeth. The most rapid disease progression is seen in that relatively small number of persons in whom the disease starts young, and there is some evidence that these individuals have some genetic predisposition to periodontitis.\textsuperscript{89,93,94}

It is uncommon for elderly people with reasonably intact dentition to exhibit sudden bursts of periodontitis.\textsuperscript{73} Tooth retention, good oral hygiene, and periodontal health (exhibited by little gingival inflammation and few deep pockets) are closely associated, regardless of age.\textsuperscript{95,96}

Gender
CAL of all levels of severity is generally more prevalent in males than in females. This has been a consistent finding in all national surveys\textsuperscript{8,9,97} in the United States since the first such survey in 1960-62. Males usually exhibit poorer oral hygiene than females, whether measured as calculus or soft plaque deposits.\textsuperscript{8,9,97,98}

The reasons for these gender differences have not been explored in detail, but are thought to be more related to poorer oral hygiene, less positive attitudes toward oral health, and dental-visit behavior among males than to any genetic factor. There are, of course, certain gender-related temporary syndromes related to hormonal conditions, such as pregnancy-associated gingivitis, as well as puberty-associated gingivitis which can affect children of both sexes.

Socioeconomic Status (SES)
A multitude of disease conditions are associated with socioeconomic status, and cause/effect (e.g., social stress as a contributory cause of heart disease) is plausible.\textsuperscript{99} Generally, those who are better educated, wealthier, and live in more desirable circumstances enjoy better health status than the less educated and poorer segments of society. Periodontal diseases are no different\textsuperscript{100,101} and historically have been related to lower SES.\textsuperscript{9,97} The ill effects of living in deprived circumstances can start early in life.\textsuperscript{102}

Gingivitis and poor oral hygiene are clearly related to lower SES, but the relationship between periodontitis and SES is less direct. For example, the 1985-86 national survey\textsuperscript{8} found that the prevalence of CAL at all levels of severity was not closely related to household income. On the other hand, CAL of \( \geq 4 \) mm and \( \geq 7 \) mm in at least one site were both closely correlated with educational levels.

It is likely that the widely observed relation between SES levels and gingival health is a function of better oral hygiene among the better educated, more positive attitudes toward oral hygiene, and a greater frequency of dental visits among the more dentally aware and those with dental insurance (who are more likely to be white-collar employees; i.e., those with more education). While racial/ethnic differences in periodontal status have been demonstrated many times, it is thought unlikely that these represent true genetic differences. It is more likely that SES, a complex and multifaceted variable that can include a variety of cultural factors, is confounding these relationships.

Genetics
Our understanding of the genetic role in periodontal diseases has grown remarkably in just a few years; the first report identifying a genetic component in periodontitis appeared as recently as 1997.\textsuperscript{103} Most of the research relating to the strength of genetics as a determinant of disease has been laboratory and clinical studies rather than epidemiology, but that research should still be briefly reviewed here.

The original 1997 report,\textsuperscript{103} using data from patients in private practices, found that a specific genotype of the polymorphic IL-1 gene cluster was associated with more severe periodontitis. This relationship could be demonstrated only in non-smokers, which suggested right away that the genetic factor was not as strong a
risk factor as smoking. The IL-1 gene cluster has received a lot of research attention since then. This is appropriate, given that the proinflammatory cytokine IL-1 is a key regulator of the host response to microbial infection, although IL-1 variability instead of IL-1 is unlikely to be the only genetic factor involved. IL-1 has been identified as a contributory cause of periodontitis among some patient groups and in one epidemiological study. However, not all studies exploring a possible genetic link have found one.

While there seems to be little doubt about a genetic component in periodontitis, the strength of that component is still being determined. At one end, a study among 169 twin pairs concluded that about half of the variance in periodontitis was attributable to heritability. At the other end, there were no differences in tooth loss attributable to IL-1 variation over 10 years in a non-smoking, well-maintained population. A combination of IL-1 genotyping and smoking history may provide a good risk profile for patients and a smoking–genetic interaction may be a contributory factor in severity of periodontitis. The role of IL-1 in regulating host response to infection has been described as clearly present, but not essential. Further research, especially epidemiological studies of people with and without disease, will be necessary before the genetic contribution to the initiation and progression of periodontitis can be specified. With current knowledge, inducing periodontal patients to stop smoking would be a higher priority than genetic testing.

**RISK FACTORS FOR PERIODONTITIS**

**Plaque, Microbiota, and Oral Hygiene**

While there is a clear causal relationship between poor oral hygiene and gingivitis, the relationship of oral hygiene to periodontitis is less straightforward. Oral hygiene can favorably influence the ecology of the microbial flora in shallow-to-moderate pockets, but it does not affect host response. Oral hygiene alone has little effect on subgingival microflora in deep pockets and personal oral hygiene practices among health professionals have been shown to be unrelated to periodontitis in these individuals. The conclusion from older studies, mostly cross-sectional, in populations with poor oral hygiene is that plaque and supragingival calculus accumulations correlate poorly with severe periodontitis. Results from other well-controlled studies also concluded that the quantity of plaque accumulation was, at best, only weakly correlated with periodontitis. Clinical findings from the Karlstad studies in Sweden, however, concluded that CAL in susceptible adults can be halted almost completely when meticulous self-performed plaque control is combined with professional prophylaxis three to six times per year. The prophylaxis in these studies included sub- and supragingival scaling, and root planing.

Studies using qualitative measures of plaque (i.e., microbiota), rather than just plaque quantity, have historically produced mixed results, although modern molecular techniques have clarified the picture more in recent years. Cross-sectional associations between putative periodontopathogens and clinical periodontitis have been reported and their presence in subgingival plaque samples from susceptible patients has predicted CAL over the short term. On the other hand, the presence of specific microorganisms could not predict the development or progression of periodontitis in clinical longitudinal studies for up to 3 years. It has long been thought that Gram-negative anaerobes were the primary pathogens in periodontal pockets, but for many years efforts to identify specific causative microorganisms have been unsuccessful. More recently it has become clearer that in the broad Gram-negative profile found at diseased sites there are several putative pathogens that are consistently found. The predominant group includes Actinomyces actinomycetemcomitans (Aa), Bacteroides forsythus (Bf) (now Tannerella forsythensis), Porphyromonas gingivalis (Pg), Prevotella intermedia (Pi), Fusobacterium nucleatum, Campylobacter rectus, and Treponema denticola. The presence of different clonal types of these bacteria is recognized, and it is not known whether all clonal types are pathogenic. If they are not, that could well account for some of the inconsistent associations found between the bacterial presence in the periodontal crevice and clinical disease.

Some of these putative pathogens can become established in young children. While these organisms in the periodontal crevice are closely associated with periodontitis, an important finding is that supragingival plaque can serve as a natural reservoir for them. When the bacterial insult is strong enough to overwhelm host defense, bacteria in supragingival plaque migrate subgingivally to form a subgingival biofilm. Frequent professional supragingival cleaning, added to good personal oral hygiene, has been shown to have a beneficial effect on subgingival microbiota in moderately deep pockets. These findings collectively form an evidence base for close control of supragingival plaque as part of periodontal therapy.

**Tobacco**

Smoking is clearly a risk factor for periodontal diseases, with the risk of periodontitis attributable to...
tobacco, compared to its non-use, in the order of 2.5 to 6.0 or even higher.\textsuperscript{148} Exactly how it acts in the causal chain, however, is still a subject for research. Smoking was first identified as a risk factor from an analysis of data from the 1971-75 National Health and Nutrition Examination Survey in the United States (NHANES I), which showed an association between smoking and periodontal diseases, independent of oral hygiene, age, or other factors.\textsuperscript{149} The evidence to identify smoking as a risk factor for periodontitis has continued to mount since then\textsuperscript{63,122,150-156} and assessments of randomly chosen patient groupings invariably show a higher prevalence of periodontitis among smokers.\textsuperscript{157-159} It has been stated that 90\% of persons with refractory chronic periodontitis are smokers,\textsuperscript{160} and healing following mechanical treatment is slower in smokers.\textsuperscript{161,162} Slower healing could come from the inhibition of growth and attachment of fibroblasts in the periodontal ligament of smokers\textsuperscript{163} and in their slower post-therapy reduction of white blood cells and neutrophils.\textsuperscript{164} Evidence for a non-effect of smoking on periodontitis is thin, although one study was found that reported smokers responding to mechanical treatment just as positively as non-smokers.\textsuperscript{165}

Experimental studies on plaque accumulation in smokers give mixed results, with some showing no difference,\textsuperscript{154,157,166} while others find more plaque and calculus in smokers.\textsuperscript{167,168} Evidence on whether smoking promotes the growth of periodontal pathogens is mixed. Earlier studies showed no difference in prevalence of these bacteria subgingivally,\textsuperscript{169,170} but more recent evidence suggests that smokers may have higher prevalence, rather than counts or proportions, of pathogenic species subgingivally.\textsuperscript{171,172} Smoking appears to promote a favorable habitat for these species in shallow pockets.\textsuperscript{171,173,174}

When discussing gingival bleeding earlier in this report, it was stated that smokers were more at risk of making the transition from non-bleeding to bleeding sites on probing.\textsuperscript{25} However, there is actually more evidence to support the view that smoking suppresses the vascular reaction which follows gingivitis, as well as compromising host response to infection in other ways. In experimental plaque-induced gingivitis, despite the rate of plaque accumulation being equal in smokers and non-smokers, the increase in gingival vascularity in smokers was only half of that seen in the non-smokers.\textsuperscript{175} In effect, this is a masking effect on the signs of inflammation\textsuperscript{148} and may be related to one reported finding of no difference in the risk of gingival recession between smokers and non-smokers with minimal disease.\textsuperscript{176} Further studies have confirmed that smoking suppresses hemorrhagic response as measured by BOP.\textsuperscript{177,178} Others have found no difference in the extent of BOP between smokers and non-smokers despite the smokers having deeper pockets\textsuperscript{179} or more plaque and calculus.\textsuperscript{167} In both instances more gingival bleeding in the smokers would have been expected. While none of this weakens the evidence that smoking is a major risk factor for periodontitis, further study on how smoking affects gingival bleeding is needed.

In other aspects of host response, smoking inhibits granulocyte function\textsuperscript{180} and interactions between smoking and the IL-1 gene cluster have also been indentified.\textsuperscript{181} In that interesting study, no difference in mean CAL could be detected between smokers and non-smokers in those who were genotype-negative, but for those who were genotype-positive, the smokers had considerably greater CAL than non-smokers. Smoking aggravates all tissue-destructive diseases, periodontitis included, by priming the production of TNF-\textalpha,\textsuperscript{182} and it also causes the release of cytokines.\textsuperscript{183} Smoking has been shown to be a stronger risk factor for periodontitis than insulin-dependent diabetes mellitus.\textsuperscript{184} The evidence is clear that smoking is a major risk factor for periodontitis.

**PREDICTING THE RISK OF PERIODONTITIS**

Attempts to identify markers of future disease go back some years. The aim is to identify the presence of some easily measured entity, one that clinicians can readily test for in a patient, that would predict with high reliability the risk of future disease. The research design has to be longitudinal, where participants have measures of suspected predictors measured at baseline (e.g., plaque, subgingival calculus, tobacco use, diabetes, SES, specific cytokines in GCF, psychic stress), and development of new periodontal lesions or progression in existing lesions is noted over a period of time. Cross-sectional assessments are limited in their usefulness. In a longitudinal design, the disease outcome can then be related to the baseline measures. There are many complications in this type of research given the complexity of the host response to periodontal infections, both in conceptualizing the research questions and measuring hypothesized predictors. Models that fit past data often cannot be accurately extended to present conditions.\textsuperscript{185}

The presence of visible plaque and calculus, as one example of a hypothesized marker, was long assumed to predict future CAL or bone loss, but studies have shown that clinical measures of plaque and calculus by themselves do not predict future disease to any useful extent.\textsuperscript{125,186-189} Models that have included the
subgingival presence of specific pathogens such as Aa, Pi, Pg, and Tf with other indicators have shown a moderate degree of predictability.\textsuperscript{134,190-192} Host response needs to be worked into the equation, and it is now recognized that smoking and genetic predisposition are major players in this regard. When smoking and IL-1 genotype status (positive or negative) are included in a predictive model, none of the baseline clinical indicators added significantly to the model for subsequent tooth loss. The baseline clinical indicators performed much better in a model that included IL-1 genotype status in non-smokers.\textsuperscript{193} What this body of research has demonstrated is that multiple predictors work better than any one single predictor by itself, although the nearest we come to a universal predictor is tobacco use.\textsuperscript{21,190,194,195}

Studies have investigated the mind–body connection and measured the role of psychosocial stress in terms of adverse life events or a history of clinical depression. Stress does seem to be associated with progressive periodontitis, whether assessed in a case-control study,\textsuperscript{196} cross-sectionally,\textsuperscript{197} or in a longitudinal design.\textsuperscript{198} Since psychosocial distress is a well-documented risk factor for a number of different diseases,\textsuperscript{99} the identification of its predictive role in periodontitis strengthens the hypothesis that periodontitis is related to systemic diseases.

While risk prediction is still not a precise science in periodontology, enough advances in our knowledge of risk factors have been made to permit development of a risk calculator that is offered to practitioners to help assess a patient’s risk of disease.\textsuperscript{199} Refinement of risk prediction models in the future will give practitioners an ever improving evidence base upon which to select treatment.

**SUMMARY**

1) Data on the prevalence of periodontitis are dependent on how the disease is defined and the age group from which they were taken. Some 5% to 20% of any population suffers from severe, generalized periodontitis, although mild to moderate periodontitis affects a majority of adults. For those who are most susceptible, periodontitis becomes evident in teenage and early adult years rather than the later years.

2) Risk factors for periodontitis include smoking, genetic predisposition, probably psychosocial stress, diabetes, and several uncommon systemic diseases.

3) Improved molecular biology techniques for measuring bacteria and inflammatory cytokines have aided recent research in both epidemiology and clinical studies, and in the future are likely to permit more precise diagnosis in the clinic.

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