Prevalence and progression of untreated periodontal disease in a young Indonesian population

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GENERAL INTRODUCTION

CHAPTER 1
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 (Sheiham et al. 1969). It is generally accepted that the number of teeth decreases with age and that caries and periodontal disease are the main causes of tooth loss, although the relative impact of these two disease entities may vary in different population groups and geographic areas (Löe et al. 1978a). The data from the studies by Brekhus (1929) and Allen (1944) 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, Manitoba, by Trott & Cross (1966). However, their results also showed, that the percentage of teeth lost due to periodontitis was higher than the percentage of patients, who lost teeth due to periodontitis. In other words, with regard to periodontitis, relatively many teeth were lost in relatively few patients.

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 (Hunt et al. 1988). The same was found in a study over a period of 28 years in Tecumseh (Burt et al. 1990). 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 invite to the conclusion, 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 (US Public Health Service 1981, Graves et al 1986) and periodontal disease (Löe et al. 1986, US Public Health Service 1987). However, tooth loss in itself is not a disease. The question that arises, is the extent to which this skewed distribution reflects social factors, as well as the underlying diseases. Studies seeking reasons for tooth loss have not probed this issue (Ainamo et al. 1984, Bouma et al. 1985, Cahen et al. 1985, Kay & Blinkhorn 1986, Bailit et al. 1987, Manji et al. 1988, Hunt et al 1988, Chauncey et al. 1939, Niessen & Weyant 1989). A principal finding in these studies is, that periodontal disease is not as important a reason for tooth loss as once thought, but many questions on the relative impact of disease,
patient attitudes, and treatment philosophy on tooth loss remain unanswered (Weintraub & Burt 1985). Epidemiologic surveys conducted throughout the world point to the almost universal distribution of caries and periodontal diseases (Russel 1967). Most studies have found that periodontitis affects a significant percent of individuals before the age of 20 and affecting 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 Scherp 1964, Brown & Löe 1993). 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 (Genco 1996).

A risk factor for periodontal disease is an environmental, behavioral, or biologic factor confirmed by temporal sequence, usually longitudinal studies, which if present, directly increases the probability of a disease occurring, and if absent, 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 (Last 1988, American Academy of Periodontology 1996). Some risk factors are modifiable, while others cannot be modified or cannot easily be modified. Those factors that cannot be modified are often called ‘determinants’ or background factors. ‘Risk factor’ often implies a modifiable condition. 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 (Beck 1994). The term ‘risk marker’ is used more in the predictive sense and usually refers to a risk factor, which is associated with an increased probability of disease in the future.

Changes in our knowledge of the etiology of periodontal diseases, and the recognition of the potential importance of susceptibility factors as they affect initiation and progression of periodontitis, have led to intense study of specific risk factors for destructive periodontal disease.
Epidemiologic studies show more periodontal disease in older age groups than in younger groups (Marshall-Day et al. 1955, Schei et al. 1959, Abdellatif & Burt 1987, Miller et al. 1987, Grossi et al. 1994, Grossi et al. 1995). This may be caused by 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 ((Holm-Pedersen et al. 1975, Machtei et al. 1994). When considering the oral hygiene status, age is not of substantial influence on periodontal disease (Abdellatif & Burt 1987). Only at older age (between 75 and 96) more severe increase of periodontal disease has been reported (Douglass et al. 1993, Fox et al. 1994). At ages up to 75 years, age by itself does not seem to be an intrinsic risk factor.

A consistent finding in all national surveys in the United States is that periodontal disease is more prevalent in males than in females (U.S. Public Health Service 1965, U.S. Public Health Service 1979, Miller et al. 1987). Risk analyses of periodontitis in other populations are not unanimous about gender as a risk factor (Umeda et al. 1998, Gamonal et al. 1998, Norderyd et al. 1999). But, like in the United States, 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 (Position Paper AAP 1996).

Socio-economic status was historically found to relate to gingivitis and poor oral hygiene (U.S. Public Health Service 1965, U.S. Public Health Service 1979). This does not account for periodontitis (Miller et al. 1987). Both in developing countries (Russell 1962, Waerhaug 1967, Wertheimer et al. 1967) and in industrialized countries (Grossi et al. 1994, Grossi et al. 1995) 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.

has not been studied epidemiologically. 

**Plaque and calculus** were discovered for their role in periodontal disease in the cross-sectional studies of the late fifties and early sixties (Lövdal et al. 1958, Ladavalya & Harris 1959, Schei et al. 1959, Mobley & Smith 1963). Controlled studies in Western populations show, that the amount of plaque has a low correlation with the amount of attachment loss measured (Lindhe et al. 1989, Badersten et al. 1990, Machtet al. 1993, Grossi et al. 1994, Grossi et al. 1995). Also the predictive value of the amount of plaque for future progression of periodontitis is low (Claffey et al. 1990, Haffajee et al. 1991). On the other hand, in their 28-year longitudinal study on risk factors for tooth loss, Burt et al. (1990) found that edentulous persons had higher baseline scores for plaque, calculus and gingivitis, then the age-matched dentate persons. Among dentate persons the baseline gingivitis score and the baseline number of teeth were risk factors for partial tooth loss. In this study periodontal attachment loss ≥4 mm and educational attainment were significant risk factors in the regression analysis. When considering the best predictor for future attachment loss, the amount of existing disease in relation to age seems to be the best choice (Haffajee et al. 1991, Grbic et al. 1991, Grbic & Lamster 1992). However this is still an inexact procedure (Position Paper AAP 1996).

A few members of the **periodontal microflora** have been considered as putative pathogens for initiation and progression of periodontal disease. Slots et al. (1986) 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 3 micro-organisms as a predictor for periodontal breakdown was investigated in adult cases with refractory periodontitis during a 12 months 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* (Wennström et al. 1987). Also microbial epidemiologic studies have shed light on the role of specific periodontal micro-organisms in periodontal disease. Carlos et al. (1988) found the presence of *Prevotella intermedia*, along with gingival bleeding and calculus was correlated with attachment loss in a group of Navajo adolescents aged 14 to 19. An epidemiologic 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* (Beck et al. 1992). Grossi et al. (1994, 1995) tested a panel of microorganisms including *A. actinomyctemcomitans*, *B. forsythus*, *Campylobacter rectus*, *Capnocytophaga* species, *Eubacterium sabureum*, *F. nucleatum*, *P. gingivalis*, and *P. intermedia*. Of these microorganisms, only *P. gingivalis* and *B. forsythus* 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.

As is apparent from all these studies, variables that may be accounted for as 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.

Therefore, a longitudinal study was initiated in 1987 in a young population, which had not received regular dental care, in order to establish the role of possible clinical and microbiological risk factors for initiation and progression of periodontitis. In 1987, all subjects in the age range of 15-25 years, living in a small village on a tea estate on western Java, Indonesia, entered the study. The material presented in this thesis describes data on changes in the clinical periodontal condition of the study population over a 7 years period between 1987 and 1994 and the relationship with clinical parameters and with presence of various periodontal bacteria in the oral cavity both at the start and at the end of the investigation.
REFERENCES


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CHAPTER 1


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