# A Comparison of the periodontal health of two groups of young adult Indonesians and characterization of advanced periodontal disease

Siti Wuryan Prayitno, author



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Abstrak

ABSTRACT
Periodontal disease has been defined as the pathological processes that involve the periodontium. These pathological changes may be limited to one of the constituent parts of the periodontium as in chronic gingivitis, or include several or all of the tissues. Most periodontal diseases are specific to the periodontium, but in some cases they may be manifestations of general disease or diseases of other organs WHO, TRS 207, 1961). Such a definition or description of periodontal disease is somewhat all encompassing. Indeed, some indices developed to measure periodontal disease appear to have considered the condition as a continuum of a single disease entity. Thus, the periodontal index (Russell 1956) uses an increasing scoring system as gingival inflammation worsens, pocketing develops and tooth mobility occurs. Similarly the Periodontal Disease Index (Ramfjord 1959) scores higher as gingivitis develops and loss of attachment occurs. In our present state of knowledge it would appear that periodontal disease is not a single entity with different degrees of severity. The umbrella title periodontal disease encompasses a number of disease states, some of which may be specific infections in a susceptible host. Unfortunately, our knowledge is still incomplete so that the full picture has yet to be unraveled.

A variety of classifications for periodontal disease have been proposed with broad agreement for the terms chronic gingivitis, chronic adult periodontitis, prepubertal periodontitis, chronic juvenile periodontitis, and rapidly progressive (adult) periodontitis (Gotlieb 1923, Butler 1969, Baer 1971, Page et al 1983a,b, Bystrom et al 1983, Cutress 1986). Further subdivisions of these disease states have been suggested, and include descriptive terminology such as localized and generalized (Baer 1971, Page et al 1983a). Additionally, the condition refractory periodontitis has been suggested to describe disease, which has failed to respond to conventional therapy (Gordon et al 1985).

Despite the identification of apparently distinct chronic disease states of the periodontal tissues, it would appear most of the conditions diagnosed by present day clinical techniques fall within the categories of chronic gingivitis or chronic (adult) periodontitis. Perhaps fortunately, the other disease entities are rare (Page et al 1983a,b, Bystrom et al 1983, Cutress 1986).

The one factor common to all the chronic disease states of the periodontal tissues appears to be a bacterial aetiology or at least a bacterial association. As with all "infections" host factors play an important role in the susceptibility and progress of disease. Nevertheless, bacterial deposits are always found in association with periodontal disease states and at present prevention and treatment is aimed at elimination of bacteria from tissues sites. The association of bacterial plaque to periodontal disease was first derived from epidemiological data (Ash et al 1964). Evidence for a direct aetiological role of bacterial plaque, at least in

the initiation of chronic gingivitis was shown by subsequent clinical experimentation (Lae et al 1965).

The specificity of the bacteria in plaque to disease states is still hotly debated. Universal acceptance of one of either the non-specific plaque hypothesis (Miller 1890) or the specific plaque hypothesis (Tanner et al 1979) has not occurred.

Even for chronic gingivitis it appears uncertain whether clinically measurable inflammation occurs in association with a critical plaque mass or a significant increase in the proportions of certain bacterial species for example, Actinomyces species, Fusobacterium nucleatum, Bacteroides rnelaninogenicus and Bacteroides intermedius (Loesche and Syed 1978, Moore et al 1982a, Moore et al 1987).

Similarly in chronic adult periodontitis, there is strong association between Gram-negative anaerobic bacteria and disease (Slots 1977b, Spiegel et al 1979, Tanner et al 1984, Mandell et al 1987). Furthermore certain organisms are frequently reported at sites of disease activity or rapid disease progression and include Bacteroides gingivalis, Fusobacterium nucleatum, Bacteroides forsythus, Elkenella corrodens, Bacteroides intermedius and Actinobacillus actinomycetemcomitans (Tanner et al 1979, 1984). However, these data are derived, for the most part, from extremely small numbers of patients. Moreover, it must be remember that in excess of 300 species and 35 genera of bacteria have been identified from human periodontal sites (Moore 1987). A further 100 or more species are thought to exist, but have as yet proved impossible to isolate and identify.

To date there have been no epidemiological studies of the microflora associated with periodontal disease. In fact, the feasibility of microbiological testing in the field has not even been assessed. The nearest attempts to large-scale oral microbiology surveys have been from clinical trials of oral hygiene products looking for adverse shifts in flora or resistance to antimicrobial agents (Volpe et al 1969a, Stallard et al 1969, Schlott et al 1976).