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شبكة المعلومات الجامعية
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شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم



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SMOKING AND PERIODONTAL DISEASE IN
EGYPTIAN ARABIC SPEAKING PEOPLE IN

EL GHARBIYA GOV. EGYPT

(An epidemiological study)

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SMOKING AND PERIODONTAL DISEASES

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Although periodontal diseases are infections caused by dental plaque (Socransky 1970), risk factors could modify the periodontal response to microbial aggression (Page *et al.* 1985, Grossi *et al.* 1995, Clarke & Hirsch 1995). Tobacco smoking is considered one of these factors (Bergström & Preber 1994).

There have been some discrepancies in reporting plaque and calculus formation among smokers and their non-smoking counterparts. Some epidemiological studies have found that smokers exhibited higher accumulation of plaque and may harbor different or more putative periodontal pathogens (Arno *et al.*, 1958). The effect of smoking on periodontal health was explained as the effect of poor oral cleanliness (Sheiham, 1971). However, many other studies have shown little difference in the level of plaque accumulation between smokers and non-smokers (Feldman *et al.* 1983; Bergström & Eliasson, 1987; Bergström, 1989). On the other hand, higher levels of calculus in smokers have been reported by some authors (Feldman *et al.* 1983; Linden & Mullally, 1994) but not by others (Bergström *et al.* 1991).

There has been also inconsistent evidence of the presence of putative pathogenic bacteria among smoking and non-smoking populations. Some studies have reported no significant differences in presence of periodontal pathogens between smokers and nonsmokers (Preber *et al.* 1992; Støitenberg *et al.*, 1993, Darby *et al.*, 2000). However, Zambon *et al.* (1996), using a fluorescence technique in a cross-sectional study from the Erie County Study population, found significantly higher proportions of *A.*

actinomycescomitans, *P. gingivalis* and *B. forsythus* among smokers compared to never smokers.

The effects of smoking on the outcomes of various modalities of periodontal therapy have been extensively reported in numerous studies. Responses to periodontal treatment, both non-surgical and surgical, appear to be compromised (Preber & Bergstrom 1986, Kaldahl et al. 1996, Scabbia et al. 2001). Treatments in smokers resulted in lesser probing depth reduction and smaller clinical attachment level (CAL) gain. Moreover, smokers were at a higher risk for recurrent diseases during periodontal maintenance care than non-smokers (Scabbia et al. 2001).

Although effects of cigarette smoking on the periodontium have been extensively examined, the changing concept of periodontal diseases in recent years and implementation of risk assessment in periodontology has encouraged research on smoking as an independent risk factor for periodontitis. The data suggest that the effects of smoking on periodontal tissues is a direct one and not owing simply to poor oral hygiene and plaque accumulation (Torrunguang et al. 2005).

More extensive research was required using more precise measurement and more uniform methodology especially in developing countries as Egypt, where the periodontal disease appears to be less confounded by other factors such as oral hygiene practice and professional dental care.

EPIDEMIOLOGY OF PERIODONTAL DISEASES

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 quantify the risk factors associated with a disease, which in turn can lead to theories of causation and produce hypotheses for its prevention and control (Burt 1993, Beck 1994).

The Prevalence of Periodontal Diseases

Prevalence is the number of cases of a disease in a designated population at a given point in time (Last 1988). Any measure of prevalence of periodontitis is dependent on how the disease is defined, i.e., the case-definition of periodontitis. If the disease is defined as the identification of at least one site with clinical attachment loss (CAL) of 2 mm or more, around 80% of all adults are affected, and over 90% of those aged 55 to 64. When the case-definition is at least one site with CAL of 4 mm or more, the prevalence in those aged 55 to 64 drops to 64%. When it is CAL of 7 mm or more, prevalence drops to below 10% (NIDR 1987).

National survey data for the United States suggest that gingivitis prevalence has declined over the last 30 years or so, and that while the prevalence of periodontitis has changed little, its severity has diminished (Capilouto & Douglass 1988, Douglass & Fox 1993). Parallel improvements in periodontal health have been reported in Scandinavia (Hansen *et al.* 1990, Hugoson *et al.* 1992).

Gingivitis: Prevalence and Distribution

When assessed in populations, gingivitis is found in early childhood, and is more prevalent and severe in adolescence, after which prevalence tends to level off (Stamm 1986). The prevalence of gingivitis among school children in the United States has ranged from 40% to 60% in national surveys (NCHS 1972, Bhat 1991). In the national survey of employed adults in 1985-86, 47% of males aged 18 to 64 exhibited at least one site which bled on probing and females 39%. The mean number of bleeding sites per person increased with age in males, but not in females (NIDR 1987).

Gingivitis is closely correlated with plaque deposits, a relationship long considered one of cause-and-effect. Studies on the natural history of periodontal diseases in Norway and Sri Lanka found no increase in prevalence and severity of gingivitis between the late teen years and age 40 in Norwegian professionals and students, among whom oral hygiene was excellent (Anerud *et al.* 1979). Among Sri Lankan tea workers, both the gingival condition and oral hygiene were poorer at all ages. Surveys among other third world populations show that gingivitis, associated with extensive plaque and calculus deposits, is the norm among adults (Baelum *et al.* 1986, 1988).

The main interest in gingivitis today is whether it is a precursor to periodontitis, since research suggests that only some sites or patients with gingivitis go on to develop periodontitis (Hugoson & Rylander 1982, Listgarten *et al.* 1985, Haffajee *et al.* 1988).

Measurement of Periodontitis

Periodontitis is defined as inflammation of the gingival tissues together with some loss of both the attachment of the periodontal ligament and bony support (Genco 1990). The standard 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 was first described over 45 years ago (Ramfjord 1959).

Although CAL, a measure of accumulated disease at a site rather than current activity, remains a diagnostic "gold standard" for periodontitis (Goodson 1990), the absence of consensus on how best to use CAL and PD in 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, and the most appropriate use of high-technology diagnostic equipment (e.g., computerized probes and continues to be debated.

As one illustration of this problem, Baer & Lester (1988) suggested that the 1985-86 National Survey of Oral Health in U.S. Employed Adults and Seniors (NIDR 1987) may have underestimated the national prevalence of periodontitis because it measured only two sites per tooth (mesiobuccal and midbuccal) 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. This probably biased the disease estimates downward.

A case-definition for periodontitis needs to establish 1) what depth of CAL at any one site constitutes evidence of disease processes, and 2) how many such sites need to be present to establish disease presence. The standard deviation of repeated CAL measurements of the same site by an experienced examiner with a manual probe is around 0.8 mm (Haffajee & Socransky 1986), so change in attachment level in a clinical study needs to be at least 2 mm (i.e., 2 to 3 times the standard deviation) before the investigators can be confident that they are seeing real change rather than measurement error (Haffajee et al. 1983, Lindhe et al. 1983). CAL progression of at least 3 mm over a given time period has been the criterion for change in other studies (Haffajee et al. 1991, Brown et al. 1994). In epidemiology, use of the 2 mm level to denote CAL in a cross-sectional study can be questioned because it is too common to serve as a disease threshold (NIDR 1987). When the CAL cutoff limit is raised to 4 mm, the distribution of affected individuals becomes sufficiently skewed to discriminate adequately between those with and without CAL of this extent (Burt 1991), but this level can be criticized as being too conservative. Regarding the extent of CAL in a mouth, the issue is how many sites need to be affected in order to give an accurate profile.

An approach like the Extent and Severity Index (Carlos *et al.* 1986), in which "extent" refers to the number of teeth in the mouth with CAL of 1 mm or more, 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 discussed previously.

A number of recent studies have used their own case-definitions, mostly based on combinations of CAL and PD or extent of bone loss (L  e *et al.* 1986, Beck *et al.* 1990, Haffajee *et al.* 1991, Page 1991, Machtei *et al.*