CHAPTER 2: SMOKING AS A MAJOR RISK FACTOR FOR ADULT PERIODONTITIS

2.1 SMOKING—A MAJOR RISK FACTOR FOR PERIODONTAL DISEASES

Tobacco use is a potent risk factor for many human diseases and conditions including cancer, cardiovascular and pulmonary diseases and it has a major deleterious effect on population health. Also, tobacco smoking appears to be one of the single most significant environmental factors in the initiation and progression of destructive periodontal disease (Solomon, Priore & Bross, 1968; Haber, 1994; Bergström & Preber, 1994; Grossi et al., 1994; 1995; Kamoi, 1996; Krall et al., 1997; AAP Position Paper, 1996b; 1999). Smoking can affect pathogenesis of the disease in an individual, change periodontal disease patterns in the population and affect periodontal therapy outcomes. A number of studies, mostly from industrialised countries, have confirmed the detrimental effects of tobacco use on periodontal health and disease. The mechanism by which tobacco exerts its influence on oral health has not been fully understood or explained by experiments. Moreover, the temporal sequence of the process is always extremely difficult to explore. However, evidence obtained from cross-sectional risk assessment studies and several longitudinal studies have suggested the causal role of tobacco smoking is the initiation and progression of periodontitis in humans.

2.1.1 Smoking affects the pathogenic chain of adult periodontitis

2.1.1.1 Smoking and periodontal pathogens

Some epidemiological studies have found that smokers exhibited higher accumulation of plaque and may harbour different or more putative periodontal pathogens (Arno et al., 1958). During some earlier periods of time, 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, Bravacos & Rose, 1983; Bergström & Eliasson, 1987; Bergström, 1989). Smoking is more prevalent among people from lower socio-economic groups (Jenkins et al., 1997) who tend to have poorer oral hygiene behaviour and practice and this may be the main reason for higher plaque accumulation. There have also been some discrepancies in reporting calculus formation among smokers and their non-smoking counterparts. Higher levels of calculus in smokers have been reported by some authors (Feldman, Bravacos & Rose, 1983; Linden & Mullally, 1994) but not by others (Bergström, Eliasson & Preber, 1991). Thus, possible higher accumulation of plaque and calculus among smokers may be owing to reasons other than smoking per se and may not be an explanation of the effects of tobacco on the periodontium.

Also, there has been 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 non-smokers (Preber, Bergström & Linder, 1992; Stoltenberg et al., 1993). Another study using the polymerase chain reactions technique to determine the presence of \( \text{A. actinomycetemcomitans, P. gingivalis, P intermedia, B. forsythus and T. denticola} \) also did not support the hypothesis that smoking increases the amount of periodontal pathogens (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 \( \text{A. actinomycetemcomitans, P. gingivalis and B. forsythus} \) among smokers compared to never smokers. Current smokers were 2.3 times more likely to be infected with \( \text{B. forsythus} \) and 3.1 times more likely to be infected with \( \text{A. actinomycetemcomitans} \) than their former or never-smoking counterparts. Moreover, there was a dose–response relationship in relative risk of \( \text{B. forsythus} \); 43\%
of subjects smoking less than ten cigarettes per day were *B. forsythus* positive against 64% of subjects who smoked more than 20 cigarettes per day.

Similar mixed findings have been reported when comparing responses to periodontal therapy of smokers and non-smokers. One study found that non-surgical therapy had similar effect in eliminating periodontal pathogens among smokers and non-smokers (Preber & Bergström, 1985a). On the other hand, some certain bacteria species such as *A. actinomycetemcomitans*, *P. gingivalis* and *B. forsythus* have been found to be less susceptible to therapy among current smokers in other studies (Grossi et al., 1996; Haffajee et al., 1997). These findings may explain a high proportion of smokers among subjects with refractory periodontitis (MacFarlane et al., 1992).

### 2.1.1.2 Effects of smoking on periodontium and the host response

Tobacco smoking could interfere with the pathological chain of periodontitis through several mechanisms. Microbial flora may or may not be affected by smoking *per se*. However, the host response, which is a more important link in the chain, may be substantially affected. Smoking could impair the normal host response in bacterial clearance and the neutralising of infection (Seymour, 1991). Nicotine in tobacco smoke may cause alterations in the oral environment and tissues themselves, which result in destruction of the periodontium (Lamster, 1992; Barbour et al., 1997).

Studies measuring host response to the infection have shown a reduction of many defensive functions among smokers. Subjects were found to have a depressed number of helper lymphocytes, which are important to B-lymphocytes function and antibody production. Salivary immunoglobulins A and serum immunoglobulins G also appear at decreased levels among smokers (Bennet & Read, 1982; Barbour et al., 1997). In particular, serum IgG antibodies to *Prevotella intermedia* and *Fusobacterium nucleatum* have been reported to be reduced in smokers (Haber, 1994). Furthermore, tobacco
Smoke and its substances can exert deleterious effects on polymorphonuclear leukocytes (PMN) and other neutrophil functions such as chemotaxis and phagocytosis of oral and peripheral neutrophils so that they cannot efficiently deal with the bacterial infection (Kenney et al., 1977; Eichel & Shahrik, 1969; Selby et al., 1992). There has also been a report of impaired phagocytosis function of neutrophils among smokers with refractory periodontitis (MacFarlane et al., 1992).

In addition, tobacco smoking may modify the production of pro-inflammatory cytokines interleukin-1 (IL-1) and Tumor necrosis factor-alpha (TNF-α), which are considered key regulators of the host response to microbial challenge. Kornman et al. (1997) recently reported a specific periodontitis associated IL-1 genotype, which was correlated with high level of IL-1 production in non-smokers. However, in smokers, severe periodontitis was not correlated with the genotype, which indicates that the genetic control of the host response was evident only when smoking was excluded. This finding further emphasises the importance of smoking in pathogenesis of periodontitis.

Another recent study reported higher levels of TNF-α among smokers in a 5-year study after periodontal therapy (Boström, Linder & Bergström, 1998), which indicates that TNF-α might be a marker for impaired healing in smokers.

Tobacco nicotine can exert cytotoxic effects on the periodontal fibroblast function, which is critical for maintenance of periodontal tissues and for optimal wound healing. It has been reported that nicotine can be stored in and released from the fibroblast (Hanes, Schuster & Lubas, 1991). Nicotine can also inhibit the production of fibroblast fibronectin and collagen and stimulate fibroblast collagenase activity (Tipton & Dabbous, 1995). But, it remains unclear whether these nicotine-exposed fibroblasts have an impaired (Raulin et al., 1988) or enhanced ability (Peacock et al., 1993) to attach to surrounding surfaces. It may be that these in vitro effects on fibroblasts may occur in vivo as well and influence the healing ability of the periodontal tissues. In
addition, tobacco nicotine can stimulate osteoblast alkaline activity and thereby suppress the proliferation of cultured osteoblasts (Fang et al., 1991).

Altogether, the exact mechanism by which tobacco smoking influences the periodontal tissues is still unclear. Biological plausible mechanisms for the effects of smoking on periodontium can be described with supportive evidence. However, research is still required to more thoroughly clarify the pathways by which tobacco smoking and its constituents exert effects on the pathogenesis and treatment outcomes of periodontal disease.

2.1.2 Effects on the disease patterns

2.1.2.1 Cigarette smoking and gingival inflammation

Earlier epidemiological studies concerning the relationship between smoking and gingival inflammation were somewhat contradictory. A series of cross-sectional studies have indicated that smokers may demonstrate lower level of gingivitis to a specific level of plaque than non-smokers (Bergström & Floderus-Myrhed, 1983; Bergström, 1990; Preber & Bergström, 1985b; 1986). This difference was found when both gingival index and dichotomous evaluation of bleeding on probing were used. One explanation for the findings may be the vasoconstrictive effect of smoking on local tissues. In this situation, because of the reduced blood flow this clinical sign of the inflammatory process may be less pronounced and the real periodontal destruction may be masked.

2.1.2.2 Cigarette smoking and periodontal destruction

There are many references to the relationship between smoking and periodontal diseases in oral and periodontal research dating back to the mid 20th Century. In a study of Danish marines aged 16 to 28 years, Pindborg (1947) observed a relationship between smoking and the deposition of calculus and gingivitis. Later on, these findings
were confirmed in another study with larger sample of 5,690 persons (Pindborg, 1949), with the additional finding that the effects of smoking on gingival tissue could not be attributed only to the effect on deposition of calculus. Furthermore, Arno et al. (1958) had found a statistically significant relationship between smoking and periodontal diseases even after adjusting for age, oral hygiene and occupation. The authors, using radiographic assessment of alveolar bone levels, later reported a greater bone loss among smokers of the same population (Arno et al., 1959). Yet, little attention had been paid to these findings before the 1980s in periodontal research, probably owing to the conclusion that the effects of smoking on the periodontium were rather indirect, relating to inadequate oral hygiene and higher plaque accumulation seen in smokers.

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. Numerous studies using different means of periodontal destruction measurement such as radiographic bone loss, periodontal pocketing and attachment loss have supported a pathogenic relation between smoking and periodontitis. These studies have reported clear associations between smoking and alveolar bone loss, loss of periodontal attachment and tooth loss. 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 (Bergström & Eliasson, 1987). These conclusions have found support in a number of studies mostly from developed countries using various measuring instruments. Owing to the aims and scope of this project, the main consideration will be paid to data presenting attachment loss as a measure of disease activity in relation with tobacco smoking. Also, all possibly available studies conducted in developing countries will be included irrespective of applied methodology.
Table 3.1: Epidemiological studies with findings on the association between smoking and periodontal health. *(Abbreviations are defined at the end of the table)*

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<thead>
<tr>
<th>Authors and Date</th>
<th>Sample/Methodology</th>
<th>Results</th>
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<tr>
<td>Bergström, 1989 Denmark</td>
<td>Case-control study. Total of 155 patients referred to periodontal therapy aged 30, 40 and 50; a random sample from the general population served as controls; full mouth probing assessment; sites with PD≥4 mm were considered diseased.</td>
<td>56% of the patients and 34% of the controls were smokers (OR 2.5); significantly higher extent of periodontally affected teeth in smokers; no differences in terms of plaque and calculus.</td>
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<tr>
<td>Beck et al., 1990 United States</td>
<td>Cross-sectional. Total of 690 community dwelling adults aged 65+. LOA at two sites per tooth, full mouth. Advanced disease: ≥4 sites with LOA≥5 mm and ≥1 site with PD≥4 mm.</td>
<td>OR for having advanced disease. Black subjects: tobacco use: 2.9; Pg&gt;2%: 2.4; Pi&gt;2%: 1.9; last dental visit&gt;3 years: 2.3. Whites subjects: tobacco use: 6.2; Pg(+): 2.4; last dental visits&gt;3 years plus BANA (+): 16.8.</td>
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<tr>
<td>Ismail et al., 1990 United States</td>
<td>Longitudinal, 28 years follow-up. 167 subjects re-examined. Probing assessment at 4 sites per tooth, full mouth. LOA: Mean; Extent of sites and subjects with LOA≥2 mm; ≥3 mm and ≥4 mm.</td>
<td>Significant OR for longitudinal attachment loss: Gender: 2.2. Education: 3.0. Dental visiting patterns: 3.1. Age: 3.9–5.4. Smoking 6.3.</td>
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<td>Goultschin et al., 1990 Israel</td>
<td>Case-control study. Convenient sample of 344 Jerusalem hospital personnel aged 18–74 years. CPI index, smoking history.</td>
<td>Non significant differences in mean sextants with CPI scores 2 and 4. Smokers had significantly higher mean sextants with CPI score 3.</td>
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<thead>
<tr>
<th>Author(s)</th>
<th>Location</th>
<th>Study Design</th>
<th>Population</th>
<th>Methodology</th>
<th>Findings</th>
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<tr>
<td>Haber &amp; Kent, 1992</td>
<td>United States</td>
<td>Cross-sectional</td>
<td>Total of 196 patients with moderate to advanced periodontal diseases in periodontal practices and 209 patients from general practices; probing assessment at 6 sites per tooth and full mouth radiographs; smoking history; patients with no periodontal history were controls; comparison of: the prevalence of smoking among the two patient groups and the severity of periodontitis in current smokers and non-smokers.</td>
<td>75% of the subjects in periodontal practice were smokers and 54% in the general practices (OR 2.6). Frequency of smoking had positive correlation with increasing severity of periodontitis in the periodontal patients.</td>
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<tr>
<td>Locker &amp; Leake, 1993</td>
<td>Canada</td>
<td>Cross-sectional</td>
<td>Total of 907 subjects 50+ years old. Two sites per tooth. Full mouth examination.</td>
<td>Multivariate analysis: age, education, current smoking status, and number of teeth were the most consistent independent variables. OR for severe disease: Age ≥ 75 years old 3.9; low education level 2.0; current smoking 2.9</td>
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<tr>
<td>Grossi et al., 1994</td>
<td>United States</td>
<td>Cross-sectional</td>
<td>Random sample of 1,426 subjects aged 25–74 years in a metropolitan community. Full mouth probing. Multivariate analysis of risk indicators for attachment loss, control for gender, socio-economic status, education and oral hygiene levels. Cigarette exposure: light, moderate, and heavy smokers by pack/years.</td>
<td>Significant OR for severe attachment loss: diabetes 2.0; age 1.9–9.1 Presence of B. forsythus 2.4 Smoking 2.0–4.7. There was a dose response effect of exposure to smoking</td>
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<td>Grossi et al., 1995</td>
<td>United States</td>
<td>Same sample as above (Grossi et al 1994). 1,361 subjects. Assessment of interproximal bone loss from full mouth radiographs. Stepwise logistic regression for the degree of association between bone loss and variables.</td>
<td>OR for severe bone loss: male 1.3; smoking 1.5–7.3; age 2.6–24.1</td>
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<tr>
<th>Study</th>
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<th>Findings</th>
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| Gelskey, Young & Singer, 1998| Canada          | Case-control study    | Dentulous patients aged 35+ attending a dental teaching clinic; 205 cases with moderate to severe periodontitis and 205 controls who were healthy or with gingivitis only. | Moderate to advanced disease as having ≥1 teeth with bone loss ≥3 mm or ≥1 teeth having PD ≥7 mm. OR for having moderate to severe periodontitis after adjustment for age and gender:  
* Current smoking 1.64.  
* Dose-response of cigarette index:  
  + 35–54 yo: 301–500 3.15; >500 7.33  
  + ≥55 yo: 301–500 1.11; >500 2.23 |
| Paidi, Pack & Thomson, 1999  | New Zealand     | Case-control study    | Convenient sample of 240 volunteers divided into current smokers, former smokers and non-smokers. Full mouth, three sites per tooth assessment using NIDR protocol. | Current smokers had more plaque and calculus.  
No difference in bleeding on probing.  
Current smokers had statistically significantly greater prevalence, extent and severity of LOA. Former smokers showed intermediate data. |
| Taani, 1997                  | Jordan          | Case-control study    | Convenience sample of 998 Jordanian adults attending dental clinics. Direct interview. Plaque by Löe and Silness. | Bleeding on probing (CPI 1) was lower among smokers but not significant.  
Calculus (CPI 2) was significantly higher in smokers.  
Non significant differences in mean of sextants with pockets (CPI 3 and 4) between smokers and non-smokers. |
| Kerdvong-bundit & Wikesjo, 2000| Thailand       | Case-control study    | Convenience sample of 120 subjects regularly attending dental clinic aged 31–60 years old. Half was current smokers. Recorded GR, PD and furcation involvement of molar teeth. | Mean GR, PD and LOA were significantly higher among smokers. More smokers had furcation involvement and higher degree of tooth mobility. |

**LOA**: Loss of periodontal attachment  
**PD**: Pocket depth  
**GR**: Gingival recession  
**BOP**: Bleeding on probing  
**NIDR**: The United States National Institute of Dental Research  
**OR**: Odds Ratio  
**CPITN**: Community Periodontal Index for Treatment Needs  
**Pg**: Porphyromonas gingivalis  
**Pi**: Porphyromonas intermedia  
**BANA test**: Hydrolysis of benzoyl-DL-arginine naphthylamide analysis
The above studies have reported a statistical significant association between smoking exposure and periodontal health among different populations. The studies, however, used different methodologies, sampling strategies, assessing instruments and criteria of assessment. These differences may affect the outcomes of the studies and make the comparisons between studies difficult. On the other hand, these differences can add the strength to the conclusion of causality based on Bradford Hill’s criteria.

In terms of study sample, many studies were based on convenient sampling strategies, which may not be representative of the general population. These samples may bias the results in some way. For example, a sample from a patient contingent may have higher distribution of the disease owing to various reasons, which may lead to underestimation or overestimation of the association.

Nevertheless, studies from developed countries using LOA measurement and radiography as the main assessing instruments have come up with consistent findings. These findings can be sufficient to conclude the role of smoking as a major risk factor for periodontal diseases among populations with high oral hygiene status and access to professional care. On the other hand, studies from developing countries, which were few in quantity, used CPITN as measuring criteria and could not confirm the effects of smoking on periodontium. This reflects the weakness of the CPITN index in presenting true patterns of the disease and in risk assessment. Consequently, the effects of smoking on periodontal health among the two-thirds of the world population in developing countries need to be further investigated in well-organised representative samples.

2.1.3 Effects on periodontal therapy

The effects of smoking on the outcomes of various modalities of periodontal therapy have been extensively reported in numerous studies. According to the recent research, smoking seems to have detrimental effects on wound healing and to contribute to post-operative complications following surgical interventions (Nolan et al., 1985; Preber &
The effects of cigarette smoking may affect the outcomes of surgical as well as non-surgical periodontal therapy. Smoking also may contribute to implant failure (Bain & Moy, 1993; Gorman et al., 1994) whereas Weyant (1994) and Minsk et al. (1996) did not find statistically significant difference in the rate of implant failure in smokers and non-smokers.

Some short-term follow-up studies after non-surgical therapy including scaling and root planing have shown poorer response to periodontal treatment among smokers compared with non-smokers. The reduction in the number of sites with gingival bleeding was significantly less in smokers (Preber & Bergström, 1985b). Probing depth reduction (Preber & Bergström, 1985a) and attachment level gain (Grossi et al., 1996) were also lower among smokers. Newman, Kornman and Holtzman (1994) reported the chances of successful outcomes (greater than 50% reduction in the prevalence of deep pockets) were 50% in smoking patients against 85% for non-smoking counterparts.

In a 12-month follow-up study after surgical therapy, the probing depth reduction was significantly less in smokers compared with non-smokers, 0.8 mm and 1.3 mm respectively (Preber & Bergström, 1990). Two other longitudinal studies following different surgical and non-surgical therapies compared pocket depth reduction and clinical attachment gain in smokers and non-smokers (Ah et al., 1994; Kaldahl et al., 1996). Treatment outcomes were significantly poorer in smokers compared to non-smokers for the entire observation period. These studies also indicated a dose-response relationship, showing a poorer outcome for heavy smokers as compared to light smokers, and for light smokers as compared to non-smokers and former smokers. In addition, Boström, Linder and Bergström (1998) after a 5-year follow-up confirmed the previous findings of lesser pocket depth reduction in smokers and reported significant differences in alveolar bone gain. Smokers did not demonstrate any alveolar bone gain whereas non-smokers and former smokers displayed significant gain over time. Root
coverage following thick free gingival graft was also reportedly affected by heavy smoking (Miller, 1987).

2.1.5  Smoking cessation and periodontal disease patterns

There are no studies directly reporting the effects of smoking cessation on periodontal disease patterns. However, epidemiological and clinical studies have shown that the periodontal status of former smokers is intermediate between smokers and non-smokers (Haber & Kent, 1992; Bolin et al., 1993). These findings suggest that smoking cessation can be beneficial to periodontal health despite the fact that the past destruction owing to smoking cannot be reversed. Paidi, Pack and Thomson (1999), in a cross-sectional study of periodontal health in 240 volunteers, had indicated significant differences in the extent and severity between former and current smokers. Current smokers displayed nearly six times the odds of having one or more sites with LOA≥6 mm whereas former smokers had the odds of 3.45 relative to a non-smoking group. Clinical studies also reported that both never and former smokers responded more favourably to therapy than current smokers and there was no significant difference between never smokers and former smokers with respect of the efficacy of periodontal therapy (Kaldahl et al., 1996; Grossi, Zambon & Machtei, 1997). Therefore, the extrapolation from these studies suggests the important role of smoking cessation in periodontal health and once again supports the causative role of smoking in periodontitis.

2.2  TOBACCO USE AMONG THE VIETNAMESE POPULATION

Asian countries of the Pacific Rim appear to have uniformly high smoking prevalence among men and a low smoking rate among women. In China the smoking rate among men and women were 61.3% and 3.8% respectively, in Indonesia 53% of men and 4% of women smoke (Yang et al., 1999; WHO, 1996). In comparison, the rates of smoking among men and women in the United States were 26% and 24%, and in Australia were 32.3% and 21.9% respectively (MMWR Report, 1996; Hill & White, 1995).
Vietnam, a country with a population of 78 million, appears to have serious smoking-related problems owing to the uncontrolled sale of tobacco. There is a governmental ban on tobacco advertisement in the mass media, but there are no restrictions on tobacco sale and smoking zones. Cigarettes can be obtained and smoked anywhere by anybody with no age restriction. This can seriously affect the uptake of smoking by the youth and can cause environmental hazards.

There are few reliable data on the prevalence of tobacco smoking among the Vietnamese population. In 1994, a Vietnamese Community Health Project supported by the University of California Pacific Rim Research Program conducted a survey of tobacco use in the two largest cities, Hanoi and HoChiMinh city, and two rural areas in Vietnam (Jenkins et al., 1997). Despite the fact that study sites were somewhat convenient, this was the most complete and reliable data on smoking prevalence. The results showed a high smoking rate among Vietnamese men, 72.8% and only 4.3% of women were smokers.

According to the above study, the smoking rate in Vietnamese men seemed to be age related with the peak at the 35–44-year-old group (84.1%) and with lower rates at younger and older age. Men belonging to the following categories: peasant class, living in rural area, having less education and lower income were more likely to smoke; however, difference were not statistically significant. Smoking was rare among women younger than 45 years old and peaked at age 65+. Vietnamese smokers mostly smoked manufactured or rolled cigarettes (73.6%), some 14.9% smoked loose tobacco in a water pipe and 11.4% reported smoking both cigarettes and water pipe. Cigarette smokers used a mean of 9.6 cigarettes per day, while water pipe smokers smoked 17.8 tobacco wads per day, which are equivalent to 4.45 cigarettes.

Other unpublished data showed the figure of around 60–70% of men who were smokers in the South of Vietnam. Some other data can be found in studies conducted among Vietnamese living in the United States and Australia confirming a similarly high
smoking prevalence among men and low smoking rate among women (Wiecha, Lee & Hodgkins, 1998; Nelson, Bui & Samet, 1997; Jenkins et al., 1995; Bermingham et al., 1999).

2.3 SUMMARY

As discussed above, cigarette smoking is considered to be one of the most significant risk factors associated with periodontal disease initiation and progression. In epidemiological and clinical studies, after adjustment for potential confounders such as plaque level and age, smokers still demonstrate greater probing depth, clinical attachment loss and alveolar bone loss. Several studies have found the relation of periodontal disease with the duration of tobacco use, smoking status and amount of tobacco consumption.

The effects of tobacco consumption on periodontal health were well observed in a number of clinical follow-up studies after both surgical and non-surgical periodontal therapy. Smoking appears to have deleterious effects on elimination of bacterial flora, wound healing, periodontal pocket reduction and attachment gain.

However, most of the studies on smoking in periodontology appear to have a number of limitations. First, they may not be representative for the general population because the sampling strategy used was often convenient or purposive selection. The non-representativeness of the samples may bias the findings to some extent. Studies used a number of different assessing instruments and different criteria of defining the disease patterns. Furthermore, studies conducted in developing countries used CPITN as the main instrument, which may not be adequate in assessing the disease itself and the possible effects of smoking on the pathological process. More extensive research is required using more precise measurement and more uniform methodology, especially in developing countries where the disease appears to be less confounded by other factors such as oral hygiene practice and professional dental care.