SMOKING AND PERIODONTAL DISEASES

The smoking is highly prevalent and can be considered an epidemic inboth developed and developing nations, tobacco users among men and womenare 42.4% and 14.2%, respectively. According to the Global Adult Tobacco Survey 2 (GATS 2), every third adult in rural areas and every fifth adult in urban areas use tobacco in some form or the other. The gas phase of tobacco smoke contains carbon monoxide, ammonia, formaldehyde, hydrogen cyanide, and toxic compounds such as benzo(a)pyrene and dimethylnitrosamine.

❖ **The particulate phase** of tobacco smoke includes nicotine, tar &benzene.

• Nicotine is quickly absorbed in the lungs and it reaches the brain within 10–19 seconds.

• Nicotine causes a rise inblood pressure, increased heart and respiratory rates, and peripheral vasoconstriction.

• Smokers have reduced gingival inflammation and bleeding on probing.

• In smokers, even shallow periodontal pockets are colonized by periodontal pathogens.

✓ **When is considered as a Smoker or Nonsmoker?**

• **Smokers** have smoked ≥100 cigarettes in their lifetime and currently smoke.

• **Former smokers** have smoked ≥100 cigarettes in their lifetime and do not currently smoke.

• **Nonsmokers** have not smoked ≥100 cigarettes in their lifetime and do not currently smoke.

Smoking is harmful to almost every organ in the body and is associatedwith multiple diseases that reduce life expectancy and quality of life. Diseases associated with smoking include lung cancer, heart disease, stroke,emphysema, bronchitis and cancers of the oral cavity, bladder, kidney,stomach, liver and cervix. Approximately half of long-term smokers will die early as a result of smoking and those who die before the age of 70 years willlose an average of 20 years of life. Most death from smoking are due to lungcancer, chronic obstructive pulmonary diseases and coronary heart diseases.

Smoking is a major risk factor for periodontitis, and it affects the prevalence, extent, and severity of disease. In addition, smoking has an adverse impact on the clinical outcome of nonsurgical and surgical therapy aswell as the long-term success of implant placement.

❖ Effects of smoking on the prevalence and severity of periodontal diseases:

Effects on Gingivitis:

Controlled clinical studies have demonstrated that, in human models of experimental gingivitis, the development of inflammation in response to plaque accumulation is reduced in smokers as compared with nonsmokers. In addition, cross-sectional studies have consistently demonstrated that smokers present with less gingival inflammation than nonsmokers. These data suggestthat smokers have a decreased expression of clinical inflammation in the presence of plaque accumulation as compared with nonsmokers.

Effects on Periodontitis:

Although gingival inflammation in smokers appears to be reduced in response to plaque accumulation as compared with nonsmokers, an overwhelming body of evidence points to smoking as a major risk factor for increasing the prevalence and severity of periodontal destruction. Multiplecross-sectional and longitudinal studies have demonstrated that pocket depth,attachment loss, and alveolar bone loss are more prevalent and severe in patients who smoke as compared with nonsmokers. On average, smokers werefour times as likely to have periodontitis as compared with persons who had never smoked, while former smokers were 1.7 times more likely to have periodontitis than personswho had never smoked.

Smoking has also been shown to affect periodontal disease severity in youngerindividuals. Cigarette smoking is associated with increased severity of generalized periodontitis in young adults and those who smoke are 3.8 timesmore likely to have periodontitis as compared with nonsmokers. In addition, smokers are more than six times as likely as nonsmokers to demonstrate continued attachment loss. Former smokers have less risk for periodontitis than current smokers but more risk than nonsmokers and the risk for periodontitis decreases with the increasing number of years since quitting smoking.

Effects of smoking on the etiology and pathogenesis of periodontal diseases:

The increased prevalence and severity of periodontal destruction associated with smoking suggests that the host–bacterial interactions normally seen with periodontitis are altered, resulting in more extensive periodontal breakdown.This imbalance between bacterial challenge and host response may be causedby changes in the composition of the subgingival biofilm (e.g., increases in the number and virulence of pathogenic organisms, changes in the host response to the bacterial challenge, or a combination of both).

*** Effects of smoking on microbiology:**

Increased complexity of the microbiome and colonization of periodontal pockets by periodontal pathogens:

***Immune– inflammatory response:**

- Altered neutrophil chemotaxis, phagocytosis, and oxidative burst.
- ↑ Tumor necrosis factor-α and prostaglandin E2 in gingival crevicular fluid.
- ↑ Neutrophil collagenase and elastase in gingival crevicular fluid.
- ↑ Production of prostaglandin E2 by monocytes in response to lipopolysaccharide.

***Physiological effect of Smoking:**

- ↓ Gingival blood vessels with ↑ inflammation.
- ↓ Gingival crevicular fluid flow and bleeding on probing with ↑inflammation
- ↓ Subgingival temperature with ↑ Time needed to recover from local anesthesia.

Microbiology:

A study sampled subgingival biofilm from all teeth with the exception of thirdmolars in 272 adult subjects, including 50 current smokers, 98 former smokers and 124 nonsmokers. After screening for 29 different subgingival species, itwas found that members of the orange and red complex species—including *Eikenella nodatum, Fusobacterium nucleatum ss*

vincentii, Prevotella intermedia and *Peptostreptococcus micros*, *Tannerella forsythia, Porphyromonasgingivalis* and *Treponema denticola*- were significantly more prevalent in current smokers than in nonsmokers and former smokers. The increased prevalence of these periodontal pathogens was caused by an increased colonization of shallow sites (pocket depth ≤4 mm), with no differences among smokers, former smokers and nonsmokers in pockets 4 mm or greater.In addition, these pathogenic bacteria were more prevalent in the maxilla than the mandible. These data suggest that smokers have a greater extent of colonization by periodontal pathogens than nonsmokers or former smokers, which may increase the risk of periodontal disease progression.

❖ Immune–inflammatory responses:

The immune response of the host to biofilm accumulation is essentially protective. **In periodontal health**, a balance exists between the bacterial challenge of the biofilm and the immune–inflammatory responses in the gingival tissues with no resulting loss of periodontal support.

By contrast, **periodontitis** is associated with an alteration in the host–bacterial balance that may be initiated by changes in the bacterial composition of the subgingival biofilm, changes in the host responses, other environmental changes, or a combination of these. Smoking exertsa major effect on the immune–inflammatory response that results in an increase in the extent and severity of periodontal destruction. The deleterious effects of smoking appear to result from alterations in the immune– inflammatory response to bacterial challenge.

The neutrophil is an important component of the host response to thebacterial challenge and alterations in **neutrophil number or function** may result in localized and systemic infections. Critical functions of neutrophils include chemotaxis (directed locomotion from the bloodstream to the site of infection), phagocytosis (internalization of foreign particles such as bacteria) and killing via oxidative and nonoxidative mechanisms. Neutrophils obtained

from the peripheral blood, oral cavity, or saliva of smokers or exposed in vitroto whole tobacco smoke or nicotine have demonstrated functional alterations in **chemotaxis, phagocytosis, and the oxidative burst.** In vitro studies of theeffects of tobacco products on neutrophils have shown detrimental effects oncell movement as well as on the oxidative burst.

In addition, **levels of antibody to the periodontal pathogens** essential for phagocytosis and killing of bacteria, specifically **immunoglobulin G2 ,** havebeen reported to be reduced in smokers as compared with nonsmokers with periodontitis, thereby suggesting that smokers may have reduced protection against periodontal bacteria. By contrast, **elevated levels of tumor necrosis factor–α** have been demonstrated in the gingival crevicular fluid of smokersand elevated levels of **prostaglandin E2** , **neutrophil elastase, and matrix metalloproteinase-8** have also been found. These data suggest that smoking alters the response of neutrophils to the bacterial challenge such that there areincreases in the release of tissue-destructive enzymes causing increased periodontal tissue destruction.

❖ Physiology:

Previous studies have shown that certain clinical signs of inflammation (e.g., gingival redness, gingival bleeding) are less pronounced in smokers than in nonsmokers. This may result from alterations in the vascular response of the gingival tissues. Although no significant differencesin the vascular density of healthy gingiva have been observed between smokers and nonsmokers, the response of the microcirculation to biofilm accumulation appears to be altered in smokers as compared with nonsmokers. With developing inflammation, gingival crevicular fluid flow, bleeding on probing and gingival blood vessels are lower in smokers than in nonsmokers.In addition, the oxygen concentration in healthy gingival tissues appears to belower in smokers than in nonsmokers, although this condition is reversed in the presence of moderate inflammation. Subgingival temperatures are lower in smokers than nonsmokers and recovery from the vasoconstriction caused by local anesthetic administration takes longer time in smokers. These data suggestthat significant alterations are

present in the gingival microvasculature of smokers as compared with nonsmokers and that these changes lead to decreased blood flow and decreased clinical signs of inflammation. This explains the long-observed phenomenon of a transient increase in gingival bleeding when a smoker quits.

❖ Effects of smoking on the response to periodontal therapy:

✓ Nonsurgical:

↓ Clinical response to root surface debridement

↓ Reduction inprobing depth

↓ Gain in clinical attachment levels

↓ Negative impact of smoking with ↑ level of plaque control

✓ Surgery and implants:

↓ Probing depth reduction and ↓ gain in clinical attachment levels after access flap surgery ↑ Deterioration of furcation after surgery, ↓ bone fill, ↑ recession, and ↑ membrane exposure after guided tissue regeneration ↓ Root coverage after grafting procedures for localized gingival recession .↑ Risk for implant failure and peri-implantitis

✓ Maintenance care:

↑ Probing depth and attachment loss during maintenancetherapy

↑ Disease recurrence in smokers

↑ Need for retreatment in smokers

↑ Tooth loss in smokers after surgical therapy

It can be concluded that smokers respond less well to nonsurgical therapy than do nonsmokers. With excellent plaque control, these differences may be minimized, but the emphasis is on truly excellent plaque control. When comparing current smokers with former

smokers and nonsmokers, the former and nonsmoker subjects appear to respond equally well to nonsurgical care,there by reinforcing the need for patients to be informed about the benefits of smoking cessation.

Nonsurgical therapy:

Numerous studies have indicated that current smokers do not respond as well to periodontal therapy as nonsmokers or former smokers do. Most clinical research supports the observation that probing depth reductions are generally greater in nonsmokers than in smokers after nonsurgical periodontal therapy. In addition, gains in clinical attachmentas a result of nonsurgical treatment are less pronounced in smokers than in nonsmokers. When a higher level of oral hygiene was achieved as part of nonsurgical care, the differences in the resolution of (4-6-mm) pocket depth between nonsmokers and smokers became clinically less significant.

Surgical therapy and implants:

The less favorable response of the periodontal tissues to nonsurgical therapy that is observed in current smokers is also observed after surgical therapy. In a longitudinal comparative study of the effects of four different treatment modalities (coronal scaling, root planning, modified Widman flap surgery, and osseous resection surgery), **smokers (with "heavy" defined as ≥20 cigarettes/day and "light" defined as ≤19 cigarettes/day) consistently showed less pocket reduction and less gain in clinical attachment as compared with nonsmokers or former smokers**. These differences were evident immediately after the completion of therapy and continued throughout7-years of supportive periodontal therapy. During the 7 years, deterioration at furcation areas was greater in heavy and light smokers than in former smokers and nonsmokers.

Smoking has also been shown to have a negative impact on the outcomes of guided tissue regeneration and the treatment of infrabony defects by bone grafts. By 12 months after guided tissue regeneration therapy at deep infrabony defects, smokers demonstrated less than half the

attachment gain that was observed in nonsmokers (2.1 mm versus 5.2 mm). In a second study, 73 smokers also showed less attachment gain than nonsmokers (1.2 mm versus 3.2 mm), more gingival recession and less bone infill of the defect. Similarly, after the use of bone grafts for the treatment of infrabony defects, smokers showed less reduction in probing depths as compared with nonsmokers.

In patients who had undergone implant therapy, smoking increases the risk of implant failure. Overall, the risk for implant failure in smokers appears to be approximately double the risk for failure in nonsmokers and the risks appear to be higher in maxillary implants and when implants are placed in poor-quality bone. Smoking has also been shown to be a risk factor for peri-implantitis. With a majority of studies showing a significant increase in peri-implant bone loss as compared with nonsmokers. Collectively, these data indicate that implant failure is more common among smokers than nonsmokers. Given the current evidence that all patients who are considering implant therapy should be informed about the benefits of smoking cessation and the risks of smoking for the development of peri-implantitis and implant failure.

Maintenance therapy:

The detrimental effect of smoking on treatment outcomes appears to be long lasting and independent of the frequency of maintenance therapy. After four modalities of therapy (scaling, scaling and root planning, modified Widman flap surgery, and osseous surgery), maintenance therapy was performed by a hygienist every 3 months for 7 years. Smokers consistently had deeper pockets than nonsmokers and less gain in attachment when evaluated each year for the 7-year period. Even with more intensive maintenance therapy given every month for 6 months after flap surgery.

*Smokers had deeper and more residual pockets than nonsmokers, although no significant differences in plaque or bleeding on probing scores were found.These data suggest that the effects of smoking on the host response and the healing characteristics of the periodontal

tissues may have a long-term effecton pocket resolution in smokers, possibly requiring more intensivemanagement during the maintenance phase.

*Smokers also tend to experience more periodontal breakdown than nonsmokers after therapy.

*Tobacco smoking was positively associated with tooth loss even when regular recall maintenance care was performed (overall, smokers had a risk of losing their teeth that was up to 380% higher than that of nonsmokers).

*Similarly, smoking has a detrimental effect on peri-implant tissue status, even when patients are under strict peri-implant preventive maintenance care.

*It is clear from these studies that **(1)** Smokers may present with periodontal disease at an early age, **(2)** They may be difficult to treat effectively with the conventional therapeutic strategies; **(3)** They may continue to have progressive or recurrent periodontitis; and **(4)** They may be at an increased risk of tooth loss or peri-implant bone loss, even when adequate maintenance control is established.

- ✓ **For these reasons, smoking cessation counseling must be a cornerstone of periodontal therapy in smokers.**

❖ Effects of smoking cessation on periodontal treatment outcomes:

Smoking cessation positively influenced periodontal treatment outcomes. When patients received nonsurgical therapy as treatment for their periodontitis, in addition to smoking cessation counseling for a period of 12 months, those individuals who successfully quit smoking for the entire 12 months of the study had the best response to periodontal treatment. The benefit of smoking cessation on the periodontium is likely to be mediated through various pathways, such as a shift toward a less pathogenic microbiome, the recovery of the gingival microcirculation and improvements in certain aspects of the immune– inflammatory responses.

- **In conclusion**: smoking is a major risk factor for periodontitis and smoking cessation should be an integral part of periodontal therapy among patients who smoke. Smoking cessation should be considered a priority for the management of periodontitis in smoker.