Republic of Iraq
Ministry of Higher Education
and Scientific Research
University of Baghdad
College of Dentistry

# Smoking and oral health

**A project Submitted to the College of Dentistry, University of Baghdad , Department of Pediatric and Preventive Dentistry, in the Partial Fulfillment of the Requirement for the B.D.S. Degree of Research in Dental Science/Preventive Dentistry**

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B.D.S,M.Sc.

**2022**

# "Declaration"

*This is to certify that the organization and preparation of this project " Smoking and Oral Health " has been made by the student Mustafa Ziyad under my supervision in the College of Dentistry, University of Baghdad in partial fulfillment of the requirement for the degree of research in preventive dentistry.*

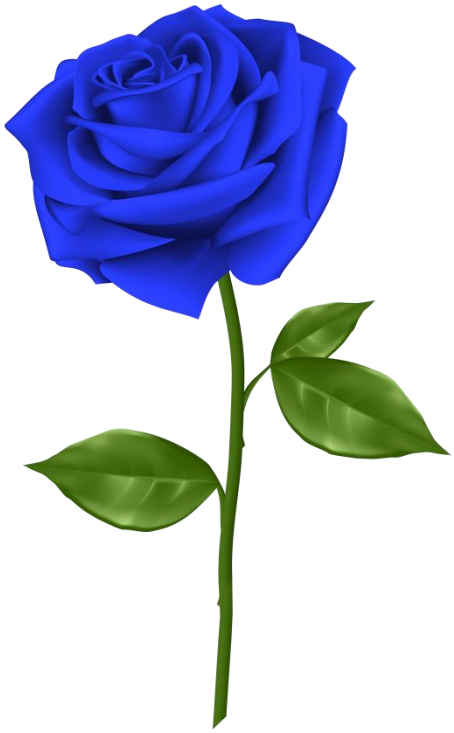
**Signature**

**Dr. Muna Abdulla Saleem**

**2022**

# "DEDICATION"

*This research is lovingly dedicated to my parents and friends who have been my constant source of inspiration. They have given us the drive and discipline to tackle an task with enthusiast and determination. without their love and support this project would not have been made possible.*



***Mustafa***

# "Acknowledgement"

**We thank Allah for giving us the strength and patience to achieve this work, which I wish it, wilI be useful and objective.**

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## Introduction

Smoking as the inhalation of the smoke of burned tobacco that may occur occasionally or habitually as a consequence of a physical addiction to some chemicals substances, primarily nicotine. Therefore, smoking must be defined as a chemical toxicosis which is able to cause detrimental effects either of acute or chronic type on different structures of the body (Anderson, 2006).

The analysis of studies showed undoubtedly that several constituents of cigarette smoking play a strong role in the development and progression of human body damage, mainly, cardiovascular damage, primarily atherosclerotic lesions and other systemic diseases (Bagaitkar et al., 2010).

In addition, tobacco products can lead to oral diseases, like gum disease by affecting the attachment of bone and soft tissue of the teeth. More specifically, it appears that smoking interferes with the normal function of gum tissue cells. This interference makes smokers more susceptible to infections, such as periodontal disease, and also seems to impair blood flow to the gums, which may affect wound healing (Buduneli et al.,2011).

The perils of smoking extend beyond active smokers to include those who are in their environment (i.e., passive smokers): second-hand smoke contains at least 50 known carcinogens and other harmful chemicals. By simply quitting smoking, however, smokers can, over time, reduce their risk levels (Avsar, 2013).

From these observations there is evidence that a large number of socio- economic and epidemiologic implications arise in smokers and that requires the necessity of specific structures which may help to face up the problem. Quitting is the only way to decrease the risk of these and other tobacco-related health problems. The addictive quality of nicotine, which is found in cigarettes, cigars and chewing tobacco, can make this especially difficult.

# REVIEW OF LITERATURE

## 1.1 Definition

Smoking is a practice in which a substance is burned and the resulting smoke breathed in to be tasted and absorbed into the bloodstream. Most commonly the substance is the dried leaves of the tobacco plant which have been rolled into a small square of rice paper to create a small, round cylinder called a "cigarette" (as in figure 1)(Reitsma, et al., 2015).

Figure 1 (cigarette)

Smoking is primarily practiced as a route of administration for recreational drug use because the combustion of the dried plant leaves vaporizes and delivers active substances into the lungs where they are rapidly absorbed into the bloodstream and reach bodily tissue. In the case of cigarette smoking these substances are contained in a mixture of aerosol particles and gasses and include the pharmacologically active alkaloid nicotine(Berridge, 2007).

Smoking generally has negative health effects, because smoke inhalation inherently poses challenges to various physiologic processes such as respiration. Diseases related to tobacco smoking have been shown to kill approximatcly half of long-

term smokers when compared to average mortality rates faced by non-smokers. Smoking caused over five million deaths a year from 1990 to 2015 (Reitsma, et al., 2015) .

Smoking is one of the most common forms of recreational drug use. Tobacco smoking is the most popular form, being practiced by over one billion people globally, of whom the majority are in the developing countries (Berridge, 2007; WHO, 2009).

Less common drugs for smoking include cannabis and opium Some of the substances are classified as hard narcotics, like heroin, but the use of these is very limited as they are usually not commercially available (WHO, 2009).

In the 20th century, smoking came to be viewed in a decidedly negative light, especially in Western countries. This is due to smoking tobacco being among the leading causes of many diseases such as lung cancer, heart attack, erectile dysfunction, and birth defects. The health hazards of smoking have caused many countries to institute high taxes on tobacco products, run ads to discourage use, limit ads that promote use, and provide help with quitting for those who do smoke (Anderson, 2006)

## 1.1.2 History

### Early uses

Smoking can be dated to as early as 5000 BCE, and has been recorded in many different cultures across the world. Early smoking evolved in association with religious ceremonies; as offerings to deities, in cleansing rituals or to allow shamans

and priests to alter their minds for purposes of divination or spiritual enlightenment (Soni, 2012) .

After the European exploration and conquest of the Americas, the practice of smoking tobacco quickly spread to the rest of the world. In regions like India and Sub-Saharan Africa, it merged with existing practices of smoking. In Europe, it introduced a new type of social activity and a form of drug intake which previously had been unknown (Winn, 2001; Soni, 2012).

Many ancient civilizations, such as the Babylonians, Indians and Chinese, burnt incense as a part of religious rituals, as did the Israelites and the later Catholic and Orthodox Christian churches. The smoking of tobacco,as well as various hallucinogenic drugs, was used to achieve trances and to come into contact with the spirit world (Berridge, 2007)

Substances such as cannabis, clarified butter (ghee), fish offal, dried snake skins and various pastes molded around incense sticks dates back at least 2000 years and Fumigation (dhupa) and fire offerings (homa) are prescribed in the Ayurveda for medical purposes, and have been practiced for at least 3,000 years while smoking, dhumrapana (literally "drinking smoke"), has been practiced for at least 2,000 years.

## 1.2 Substances and equipment

The most popular type of substance that is smoked is tobacco and There are many different tobacco cultivars which are made into a wide variety of mixtures and brands. Tobacco is often sold flavored, often with various fruit aromas, something

which is especially popular for use with water pipes, such as hookahs. The second most common substance that is smoked is cannabis, made from the flowers leaves of Cannabis or sativa or Cannabis indica.

Since cannabis is illegal or only tolerated in most jurisdictions, there is no industrial mass-production of cigarettes, meaning that the most common form of smoking is with hand-rolled cigarettes, or with pipes. Water pipes are also fairly common, and when used for cannabis are called bongs (as in figure 2) (Ramon, et al., 2002).

Figure 2 (Bong)

One of the substances that have major side effect on oral and general health is opium which is bitter brownish addictive narcotic drug that consists of the dried latex obtained from immature seed capsules of the opium poppy (as in figure 3), and that is smoked illicitly as an intoxicant with harmful effects (Ray, 2006).

A few other recreational drugs are smoked by smaller minorities. Most of these substances are controlled, and some are considerably more intoxicating than either tobacco or cannabis. These include crack cocaine, heroin, methamphetamine. Even the most primitive form of smoking requires tools of some sort to perform. This has resulted in a staggering variety of smoking tools and paraphernalia from all over the world. Whether tobacco, cannabis, opium or herbs, some form of receptacle is required along with a source of fire to light the mixture (Monteiro, 2013).

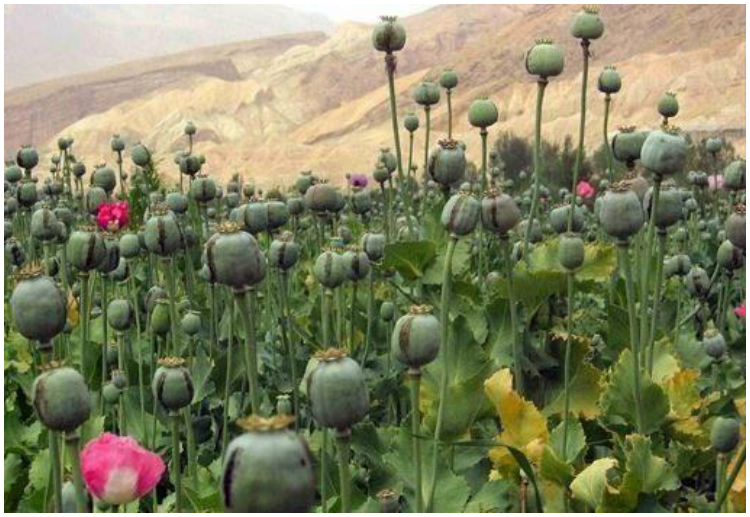

Figure 3 (Opium poppy)

Globally, the deleterious and hazardous effects of cigarette smoking and other tobacco usages affect public health. Smokeless tobacco (ST) includes products that are consumed orally. They are of two major forms: snuff and chewed form. Snuff may be available in the form of moist or dry bolus, the former being taken as oral snuff and the latter usually inhaled through the nose. While chewing, the smokeless tobacco (as in figure 4) variety is usually placed in the buccal vestibule of the oral cavity, (Walsh, 2000).

Figure 4 (Smokeless tobacco)

In India, many tobacco consumption patterns are used because many tobacco products are commercially available. After the introduction of gutka, the marketing and commercial production of smokeless tobacco (ST) has been up scaled.In India, cigarette smoking is the most common pattern of tobacco consumption (Soni, 2012; Simon, 2013; Kumar, 2015)

There are more of 93 known harmful and potentially harmful chemicals in cigarettes which include : Nicotine, Cadmium Lead Acrolein Acetaldehyde , Benzene, Ammonia, Carbon Monoxide (Hecht SS,2012)

## 1.3 Smoking and oral health

### 1.3.1 Smoking and periodontal disease

The role of tobacco smoking as a causative factor in the development of the periodontal disease has long been debated and recently a large number of papers have been published in the literature regarding this symbiosis. Smoking also gives an encouraging environment for microbes in the mouth such as Porphyromonas gingivalis, Prevotella intermedia, and Aggregatibacter actinomycetemcomitans because the by-products of smoking prevent the mechanisms that limit the

growth of harmful bacteria in the oral cavity (Bagaitkar et al., 2011). Thus, smoking promotes early stages of periodontal disease .

Smoking in the form of cigars and pipes have similar deleterious effects that cigarettes do on oral health. Much of the literature has also showed that smokers affected with periodontitis respond less favorably to periodontal treatment be it nonsurgical and surgical (Calsina, 2002; Buduneli et al., 2011).

Periodontal disease is among the main contributors to the global burden of oral disease. However, somewhat limited attention has been given to periodontal disease in many countries by providers of oral health care and public health administrators. Since last decade dentists and dental researches have become more alert over the serious role of smoking on incidence and severity of periodontal disease, (as in figure 5), thus naming smoking as a risk factor for periodontal disease (Yanagisawa et al,. 2009).

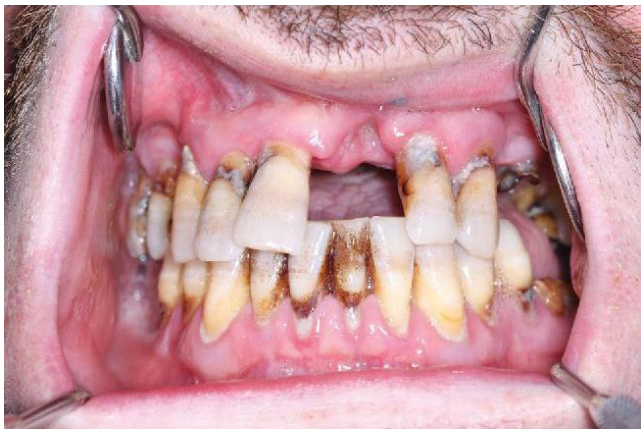

Figure 5 (Periodontal disease)

The relationship between smoking and periodontal health was investigated as early as the middle of last century. Smoking is

an independent risk factor for the initiation, extent and severity of periodontal disease. Additionally, smoking can lower the chances for successful treatment (Bagaitkar et al., 2011).

Periodontal diseases due to smoking show more predilections for males than females. There are more than 4000 chemicals in a cigarette that slow down the healing process during periodontal treatment which includes formaldehyde, carbon monoxide, ammonia, and arsenic. Thus, smoking can encourage early stages of periodontal lesions. Smoking also decreases the periodontal response to treatment and causes refractory disease (Buduneli et al., 2011).

Periodontal treatment tends to be less likely to be successful in smokers than in non-smokers. Studies evaluating the efficacy of periodontal disease control and specific periodontal procedures including regenerative procedures, soft tissue grafting procedures and implant procedures have consistently demonstrated a negative effect from smoking on success rates because the smoking impair the blood flow to the site of injury and cause delay in wound healing (Bagaitkar et al., 2010).

During periodontitis, cigarette smoking may differentially affect neutrophil function, generally preventing elimination of periodontal pathogens, but, in heavy smokers also stimulated reactive oxygen species release and oxidative stress mediated tissue damage,( Matthews, 2011).

Tymkiw et al., 2011, compared the expression of 22 chemokines and cytokines in gingival crevicular fluid from smokers and non-smokers, with periodontitis and periodontally healthy control subjects, and concluded that periodontitis subjects had significantly elevated cytokine and chemokine

profiles. Smokers exhibited a decrease in several pro-inflammatory cytokines and chemokines and certain regulators of T-cells and natural killer cells.

## Smoking and dental biofilm

It has been reported that smokers have a poor level of oral hygiene when compared to nonsmokers, the tooth brushing effectiveness of smokers is much less, and calcium concentration in the dental plaque (as in figure 6) of smokers has been found to be significantly higher than innonsmokers, which suggests a direct influence on the rate of calculus formation and deterioration of oral hygiene (Tymkiw et al., 2011).

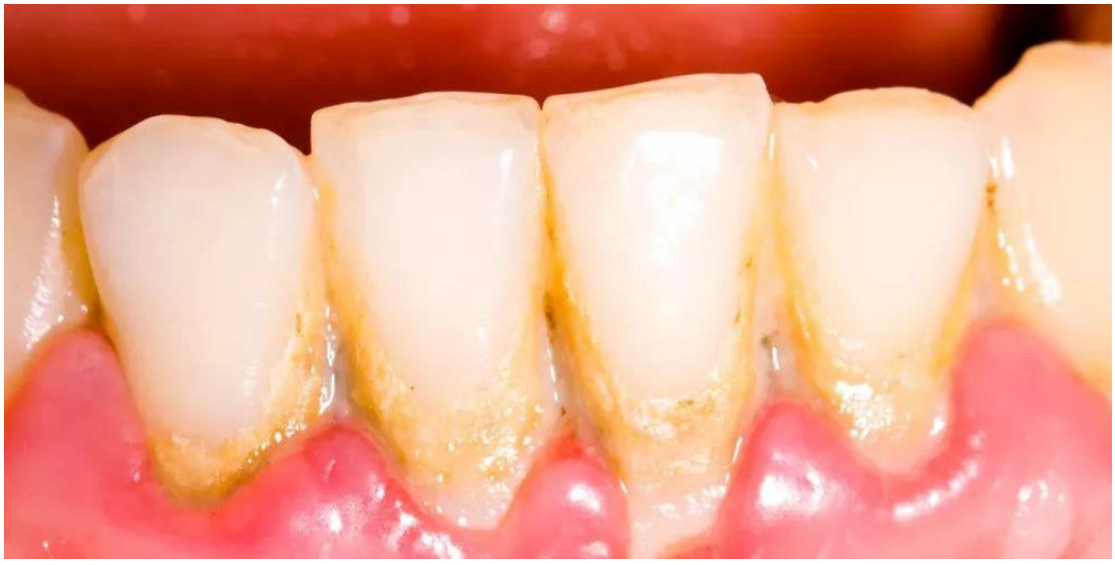

Figure 6 (Plaque deposition in smoker)

It is well known that smokers have higher plaque index scores than nonsmokers. Males have significantly more plaque than females and in both genders, smokers have almost twice the percentage of marginal line with an adherent plaque as nonsmokers (Faddy et al., 2000). researches have suggested that plaque is more adherent to the tooth and less freely

removed from the teeth of smokers due to the deposition of tars from smoke (as in figure, 7)(Faddy et al., 2000; Kinane, 2000).

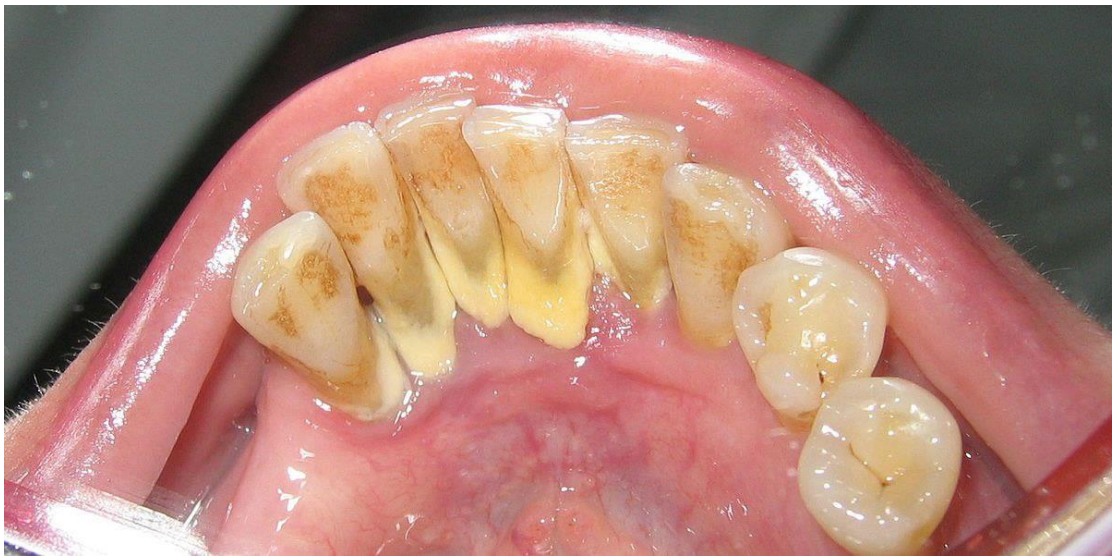

Figure 7 (Dental tar)

For many years sciences did not know how smoking affect periodontium and why people with chronic periodontitis have reduced clinical inflammation. we know that tobacco smoke induces alterations to the 3-OH fatty acids present in lipid A in a manner consistent with a microflora of reduced inflammatory potential. In investigation smookers had significant reductions in the 3-OH fatty acids associated with the consensus (high potency) enteric LPS/ structure were noted in smokers compared with non- smokers with chronic periodontitis (de Olivera, 2012).

Thus, smoking is associated with specific structural alterations to the lipid-A-derived 3-OH fatty acid profile in saliva that are consistent with an oral micro flora of reduced inflame matory These findings provide much-needed potential. mechanistic insight into the established clinical conundrum of increased

infection with periodontal pathogens but reduced clinical inflammation in smokers (Sreedevi, 2012).

## Smoking and gingivitis

Cigarette smoking also causes a lowering of the oxidation-reduction potential (Eh), which causes an increase in anaerobic plaque bacteria. According to previous studies, Eh values in gingiva drops significantly after smoking one cigarette (Friedman, 2008). Furthermore, tobacco smoke contains phenols and cyanides which can account for antibacterial and toxic properties. Smokers harbor significantly higher levels of these and are at significantly greater risk of infection with Bacteroides forsythus than nonsmokers (Sham, 2003)

P. gingivalis is also more likely to sub gingival infect smokers than nonsmokers. However, the relative risk for infection due to this bacterium is significantly higher. It has been found that three species of Gram-negative bacteria, Branhamella catarrhalis, Neisseria perflava, and Neisseria sicca are more susceptible to cigarette smoke than three species of Gram-positive bacteria Streptococcus mitis, Streptococcus salivarius, and Streptococcus sanguinis (Calsina et al., 2002).

The vasoconstriction of peripheral blood vessels which is caused by smoking can also affect the periodontal tissue as smokers have less overt signs of gingivitis than nonsmokers and clinical signs of gingival inflammation such as redness, bleeding, and exudation are not as apparent in smokers (Lie, 2002).

Smoking increases the number and depth of periodontal pockets and attachment loss of periodontal ligaments. Loss of tissue strength which caused by harmful compounds in tobacco

can increase gingival recession (as in figure 8) and changes in the oral mucosa(Pihlstrom 2005).

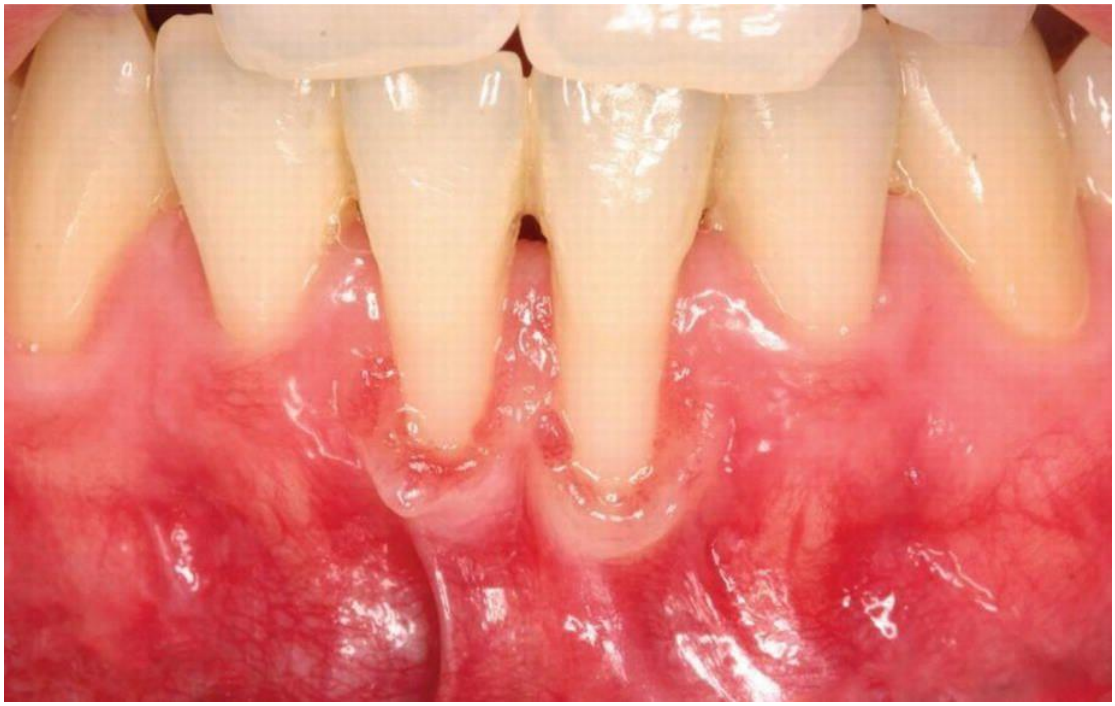

Figure 8 (Gingival recession)

### 1.3.2 Smoking and dental caries

Dental caries is a multi-microbial disease caused by various associations and is not infectious. It is considered a diet- and pH-dependent process due to the acid demineralization of the tooth enamel by sugar-fermenting microorganisms (Simon-Soro, 2013). The data on dental caries prevalence in tobacco smokers and chewers were not completely known. The literature discussed both the increased and decreased prevalence of dental caries in tobacco users. (Sathyanarayanan, 2002; Prokhorov, 2006).

Smoking and its relation to dental caries is a subject of controversy. Early reports in literature indicated that dental caries were reduced in individuals having a smoking habit,

which suggests that smoking actually reduces dental caries (Sathyanarayanan, 2002; Simon, 2013).

Schmidt, in 1951, reported that an increase in tobacco smoking led to a decrease in dental caries rate supporting the belief of previous study The concentration of thiocyanate, a constituent of tobacco smoke, has possible caries-inhibiting effect. It was found to be higher in smoker's saliva, thus depicted that smoking had inhibitory effect on smoking (Johnson, 2000).

On the other hand, few studies also stated that increased susceptibility to dental caries might be due to possible lower pH levels, a decreased buffering capacity of smoker's saliva, and the higher number of lactobacilli and Streptococcus mutans (Johnson, 2000; Reibel, 2003).

Results from few studies reported that salivary flow rates between smokers and nonsmokers showed no significant differences. To date, significant correlation between smoking frequency and dental caries was found (Rudzíński, 2010; Campus, 2011).

In 2007, Dietrich studied tobacco use and the incidence of tooth loss among US male healthcare professionals. They found that the data on the dose-dependent effect of smoking and smoking cessation on tooth loss were scarce. They hypothesized that smoking had both dose- and time-dependent effects on tooth loss incidence.

### 1.3.3 Smoking and saliva

Saliva is a complex and important body fluid which is very essential for oral health. Saliva is required for protecting the oral mucosa, teeth remineralization, digestion, taste sensation, pH balance and phonation. It includes a variety of electrolytes, peptides, glycoproteins, and lipids which have antimicrobial, antioxidant, tissue repair, and buffering properties. Saliva is the first biological fluid that is exposed to cigarette smoke, which contains numerous toxic compositions responsible for structural and functional changes in saliva (Kelbauskas, 2005; Sreedevi, 2012).

Smoking causes a short-term increase in salivary secretion, the long-term effects of tobacco use are unclear. Intense smokeless tobacco use has been shown to result in degenerative changes of more than 40% of minor salivary glands located in the site of chronic tobacco placement (Kelbauskas, 2005).

### 1.3.4 Smoking and halitosis

Bad breath, also known as halitosis, is a symptom in which a noticeably unpleasant breath odor is present (Kapoor, 2016).

A special reason that smoking tobacco causes halitosis. In fact, there are several reasons why people who smoke are far more likely to suffer from a particular type oral odor called, what else, smoker's breath. The most immediate way that cigarettes cause bad breath is by leaving smoke particles in the throat and lungs. This effect is typical of nearly any tobacco product that involves inhaling smoke or rolling it around in the mouth. The smell of a freshly smoked cigarette can linger in the

lungs for hours, hence the stale scent associated with smoker's breath (Harvey- Woodworth, 2013).

However, that's just the beginning. The chemicals in tobacco smoke can remain in the mouth, leading to a host of secondary causes of bad breath. Multiple investigations have been conducted over the years to determine just what it is in smoke that leaves the mouth smelling so awful. Chemical reviews found that tobacco smoke contains more than 60 aromatic hydrocarbons, most of which are carcinogenic in addition to conveying a fragrance, (Kapoor, 2016).

A more recent study published focused only on which compounds can be found in the saliva after smoking one-half of a cigar. Its authors determined that ethyl pyrrole, 2,3-dimethyl pyrazine and 2-ethyl pyridine are some of the most pungent chemicals found in what is often referred to as cigar breath. They added that many such compounds are generated during the combustion of tobacco and its chemical additives (Nachnani, 2011: Ferguson, 2014).

However, tobacco causes chronic bad breath by drying out the palate. It's not too hard to see how this happens. Repeatedly inhaling hot gases parches the tongue and gums, leaving a dry, chemical-filmed environment where anaerobic oral bacteria can run amok (Kumar, 2015).

## 1.4 Passive smoking

Passive smoking, also known as involuntary smoking, second hand smoking or exposure to environmental tobacco smoke (ETS), is defined as inhalation of the cigarette smoke of another individual or the exhale of a smoker ,Passive smoking can adversely affect the health of non-smokers of all age groups (El_Batran, 2009)

### 1.4.1 Passive smoking and oral health

Environmental tobacco smoke (ETS), contains over 4000 chemical agents adversely affecting the oral health of passive smokers (Avşar, 2008). Cotinine is a nicotine biomarker with a half-life longer than that of nicotine. Measurement of cotinine level is a suitable and reliable objective and quantitative screening tool for determination of exposure to ETS as it is for active smoking (Tanaka, 2005; Avşar, 2008)

A dose-dependent correlation exists between the number of cigarettes smoked by a smoker and the plasma and saliva cotinine levels of his/her non-smoker companion (Avşar, 2008)

Passive smoking changes the normal oral and nasopharyngeal flora and may cause upper airway infection (Hajifattahi, 2010). It may decrease alveolar bone density (El Batran, 2009) or cause severe periodontitis, implant failure, gingival pigmentation in children and adults, primary and permanent tooth decay and tooth loss. It may also delay tooth development. Passive smoking is a risk factor for occurrence of or facial clefts as well (Taghavi, 2012).

The cigarette smoke products in active and passive smoking result in edema and inflammation via the activity of pro-

inflammatory agents and local vasoconstriction. Systemically, these products decrease the level of saliva IgA and serum IgG and suppress the funetion of T helper cells in host immunity responses. It appears that passive smoking, via the above-mentioned mechanisms and oxidative stress, can cause periodontal disease like severe periodontitis, decrease the alveolar bone density and lead to tooth loss, In active smokers, in comparison to passive smokers, plaque accumulation is among the main causes of periodontal disease (Sanders, 2011)

Via the same mechanism, it also causes implant failure and increases the risk of failure of dental implants in passive smokers 2-3 times bigger than the risk of those who are not exposed to passive smoking (Twito, 2014). The mechanism of action of cigarette smoke in increasing gingival pigmentation is via the activity of polyeyclic amines such as nicotine and benzoperylene that are present in cigarette smoke and enter into the blood circulation following inhalation. They indirectly stimulate the melanocytes (Hajifattahi, 2010).

# Conclusion

Smoking generally has a negative health effects, because smoke inhalation inherently poses challenges to various physiologic processes. Diseases related to tobacco smoking have been shown to kill approximately nalf of long-term smokers when compared to average mortality rates faced by non-smokers.

Passive smoking, also known as involuntary smoking, second hand smoking or exposure to environmental tobacco smoke (ETS), can adversely affect the general and oral health of non-smokers of all age groups.

Smoking is well-established risk factor for periodontal disease. It changes the human micro flora, human immune response that leads to destruction of the supporting tissues of the tooth, the vasoconstriction of peripheral blood vessels which is caused by smoking have less overt signs of gingivitis than nonsmokers and clinical signs of gingival inflammation such as redness, bleeding, and exudation are not as apparent in smokers.

Contrary to the number of studies showing dental caries in smokers, the studies demonstrated that the smokeless tobacco (ST) chewers had more caries experience when compared to tobacco smokers. In the present scenario, tobacco is a major killer, thus a major drive against tobacco should be started, and a public health, dentist, shall be a model role plaver in this and dentists have an important role in creating awareness among the public regarding the detrimental effect of smoking on oral health.

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