# SMOKING AND ORAL EFFECTS

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## ABSTRACT

Though Smoking has systemic effects on body, one of the common sites for presentation of its effects is oral cavity. The presence and severity of various clinical entities associated with smoking vary with respect to site of oral cavity, quantity (number of cigarettes) and quality of smoke, cumulative effects of smoking and alcohol use, frequency and duration of smoking and, of course, genetic predisposition.

This paper describes the review of articles and various oral effects of smoking.

## KEYWORDS:

Smoking-cigarette/ cigar/pipe
Pathogenesis

## INTRODUCTION

Mouth can be a very visual reflection of the effects, smoking has on the body. Effects of smoking can range from something obvious and unsightly such as stained teeth to leukoplakia, a premalignant lesion to a true invasive squamous cell carcinoma.

Smoking is a major cause of cancer affecting the oral cavity and the oropharynx. On an average oral malignancy occurs up to four times more frequently in smokers than in non-smokers[1]. Approximately 57% and 51% of malignancies of the oropharynx in men and women respectively can be directly attributed to smoking[2]. Though the site predilection of malignancy caused by smoking is controversial among different authors, but according to **Mashberg** and **Meyers** the 'gutter area' of oral cavity (where pooling of saliva mixed with carcinogens occurs) i.e. floor of the mouth, ventral and/or lateral surface of tongue, and soft palate are the commonest sites of malignancy[3].

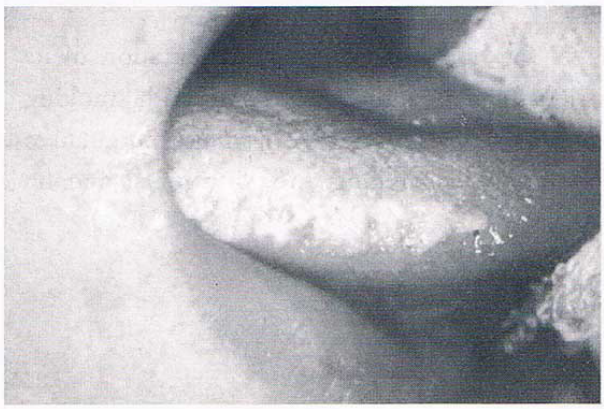
Though smoking and alcohol both are carcinogenic, but use of tobacco (smoke or smokeless) and alcohol together has also been found to markedly increase the risk of oral malignancy. The combined effects of smoking and alcohol are linked between 75% and 90% of all cases of oral malignancy[4].A male smoker of 40 or more cigarettes per day over 20 years is four times more likely to develop oral cancer. A non smoker who drinks four alcoholic drinks per day is nine times more likely to develop oral malignancy. But a person with heavy smoking and heavy drinking habit is 37 times more prone to get oral cancer compared to those who don't smoke or drink at all[5].Although the exact mechanism how tobacco and alcohol together are so damaging is not known, but it is thought that, alcohol increases the permeability of the cells for the carcinogens of smoke and alcohol itself[6].

## ORAL EFFECTS OF SMOKING

### I. Premalignant and malignant lesions

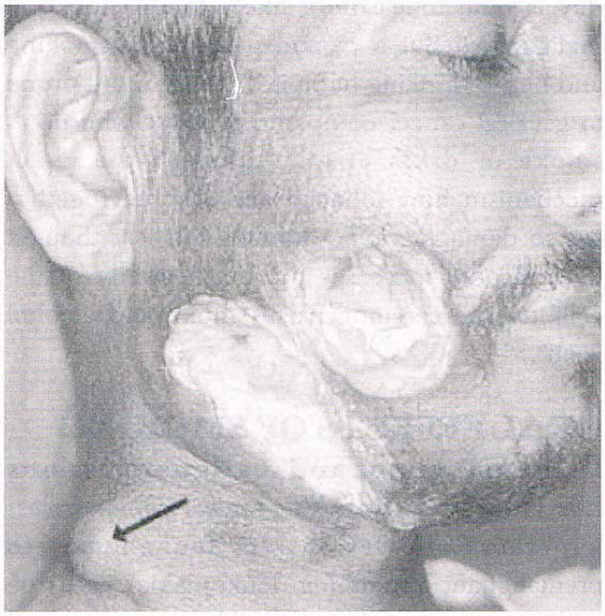
Effects of Chronic smoking ranges from harmless hyperkeratotic lesions(eg. leukedema) to premalignant lesions (eg. leukoplakia) and true malignancy of oral tissues. The presence and severity of these conditions depends on several factors including the form in which tobacco is used (cigarettes, pipes, cigars)and how long the tobacco has been in use. If cancer is not already present and smokers quit, the risk of developing

oral cancer halves after three to five years and continue to decline[1] .oral cancer is more common in men than women, however the rate that women are developing oral malignancy is increasing. This may be due to the increasing number of women who smoke and the greater numbers of women smokers in older age group[6].



**Smoking and malignancy: pathogenesis**

The chemical carcinogens (more than 300) of smoke are in fact pro-carcinogens, most of which after metabolism in the body get converted into carcinogens. The main carcinogen of smoke is aromatic hydrocarbon benzo(a)pyrene and tobacco specific nitrosamines. The amount of benzopyrene is found to be 20-40 ngm/cigeratte.[3]



The most accepted explanations (models) for pathogenesis of oral cancer are:

1. Multistage carcinogenic model,
2. Molecular epidemiology model of biomarkers,

**1. Multistage carcinogenic model[7], (Harris, 1991)** the multistage model of carcinogenesis represents a sequence of four events leading from normal cells to clinical cancer: initiation, promotion, conversion, and progression, during which multiple genetic events are postulated to take place.

**a) Initiation**

Tumor initiation is the first stage of the multistage carcinogenic process. Initiation refers to the direct effects and irreversible change in the cell DNA induced by initiators. The genetic changes includes adduct formation (mainly O-6-methylguanine), mutations and altered gene expression leading to the activations of proto – oncogenes and/or inactivation of tumor suppressor gene. Proto oncogene, on activation, behaves as an oncogene that dysregulate cell growth and promoting neoplastic growth. Tumor suppressor gene on deactivation results in continuous signal or an abnormal signal for cell proliferation and possible growth of a neoplasm. Both alleles of a tumor suppressor gene must sustain mutations to inactivate the function of this gene.

**b) Promotion**

Tumor promotion occurs when initiated cells are selectively expanded into visible benign tumors. Promotion is the second stage of the multistage carcinogenic process, does not require a genetic event, and may be reversible. The selective expansion of initiated cells is caused by altered growth and resistance to cytotoxicity of these cells, secondary to activations of proto – oncogenes and/or inactivation of tumor suppressor gene.

**c) Conversion**

Conversion is the third stage of the multistage process. In this stage, benign tumors are transformed into malignant cells through genetic event(s). The initiated cells undergo one or more additional genetic events (adduct formation, mutations and altered gene expression), thus transforming into maliganant cells. The rate of cell proliferation is a significant factor in determining the conversion of a benign tumor cell to malignant one, because it not only

converts adducts to mutations, but also decreases the amount of time available for DNA repair processes to remove DNA adducts; subsequently, the unrepaired promutagenic DNA adducts lead to replication errors, possibly resulting in mutations and ultimately malignant change.
Before cell replication turns reversible events into irreversible events, there is a window of opportunity where covalent binding of carcinogen adducts to DNA can be repaired. This DNA repair is important in the carcinogenic process, for it can prevent the initiation of carcinogenesis. Significance – cessation of smoking reduces the incidence of oral cancer.

### d) Progression

Progression is the fourth stage of the multistage carcinogenic process. In this stage, the malignant tumor further grows into clinically detectable tumors, where there is continued clonal proliferation of the genotypically or phenotypically lettered cells. This stage also includes metastasis to other organs.

**2. Molecular epidemiology model of biomarkers,** which affords a framework for investigating the sequence of changes from normal to malignant cells and identifying the roles of environmental and genetic factors.

## II. Other Effects on Oral Cavity.

### Effects on Periodontium:

Smokers are 2.5-6.0 times more prone to have periodontal disease as compared to non smokers[8].

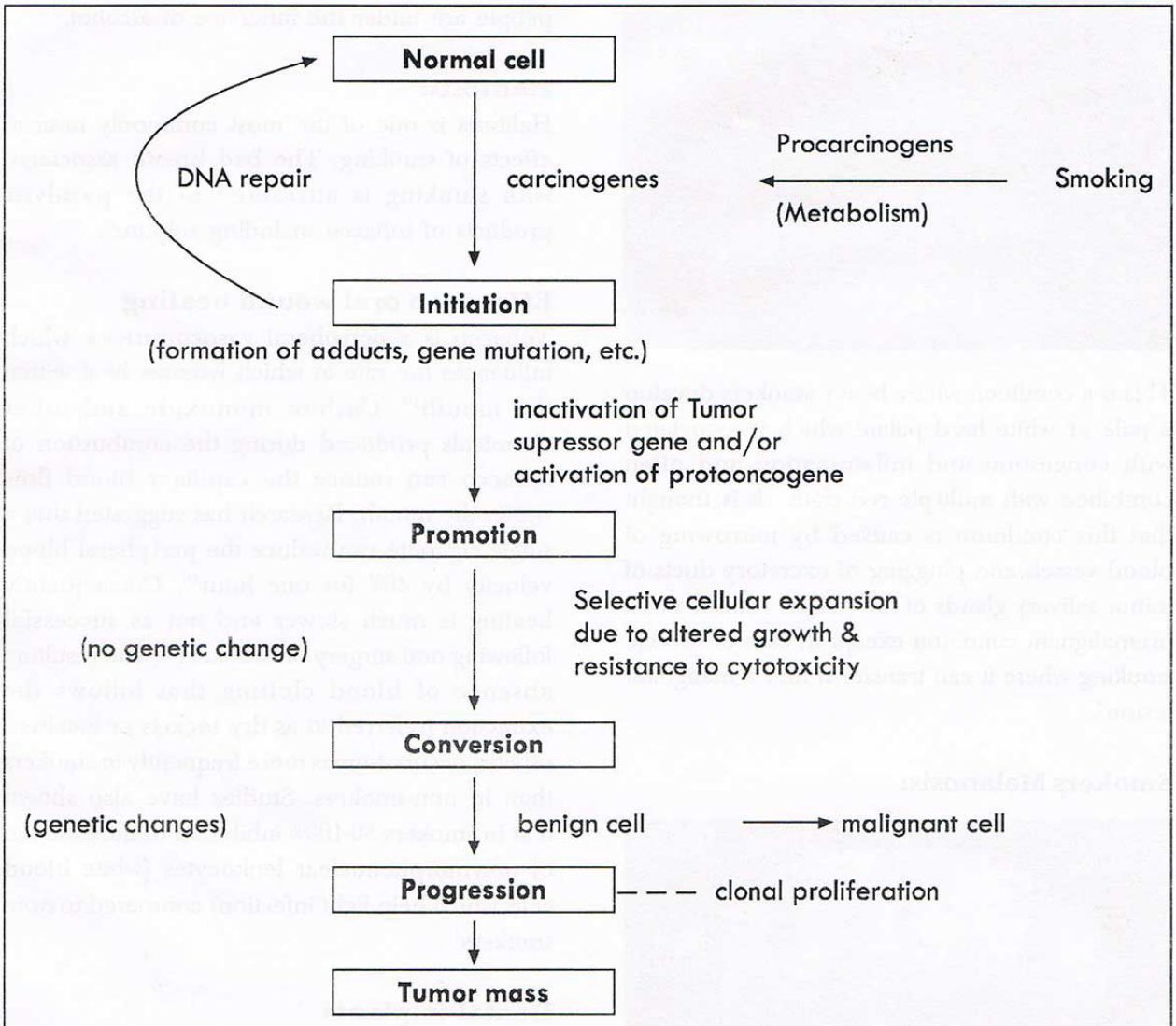
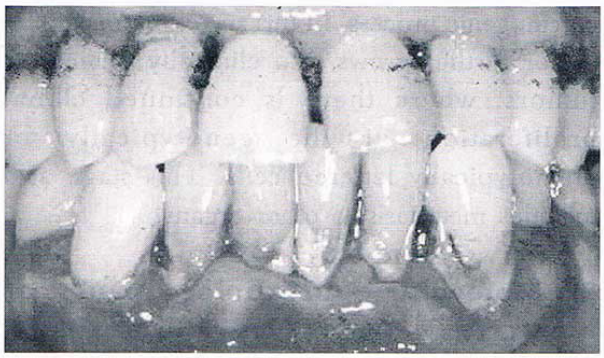
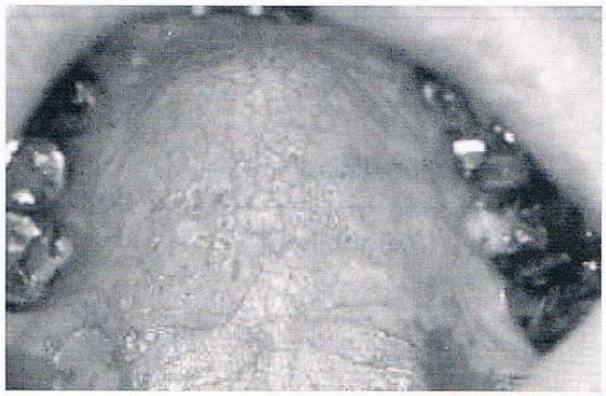



*Fig:-Schematic representation of Multistage model of pathogenesis of Oral Cancer by smoking.*



Though the exact mechanism of periodontitis associated with smoking is unknown but it is thought that smoking decreases the immune system, transiently reduces the vascular supply to the gingiva and favors the formation of plaque and calculus.

**Smokers Palate:**



This is a condition where heavy smokers develop a pale or white hard palate which is associated with congestion and inflammation and often combined with multiple red dots. It is thought that this condition is caused by narrowing of blood vessels and plugging of excretory ducts of minor salivary glands of the palate[4]. This is not a premalignant condition except in case of reverse smoking where it can transform into a malignant lesion[3].

**Smokers Melanosis:**

This is a condition where heavy smokers have an increased pigmentation especially on the buccal mucosa and gingiva. It occurs in five to ten percent of smokers. Although it is not associated with a risk of oral disease, it can be unsightly. However stopping smoking can reverse this condition, although it usually takes more than a year after stopping for the colour to return to normal[4].

**Effects on Tooth and Restoration:**

Tobacco stain can penetrate into enamel, tooth colored restorative materials and dentures creating ugly brown to yellow discoloration.

**Smokers Lip:**

This is created by burns caused by smoking unfiltered cigarettes but is generally rare unless people are under the influence of alcohol.

**Halitosis:**

Halitosis is one of the most commonly noticed effects of smoking. The bad breath associated with smoking is attributed to the pyrolytic products of tobacco including sulphur[9].

**Effects on oral wound healing**

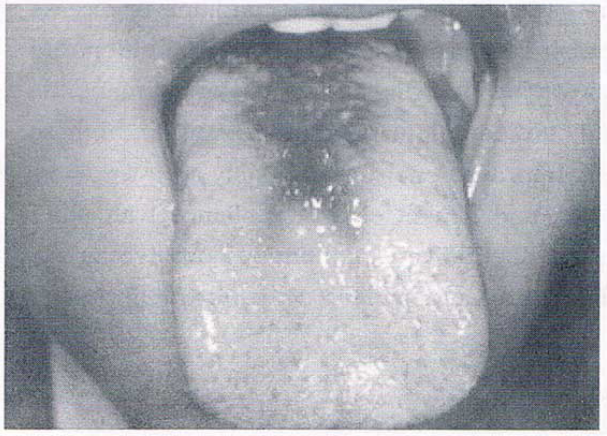
Tobacco is a peripheral vasoconstrictor which influences the rate at which wounds heal within the mouth[10]. Carbon monoxide and other chemicals produced during the combustion of tobacco can reduce the capillary blood flow within the mouth. Research has suggested that a single cigarette can reduce the peripheral blood velocity by 40% for one hour[10]. Consequently healing is much slower and not as successful following oral surgery on smokers[14]. The resulting absence of blood clotting that follows the extraction (referred to as dry sockets or localised osteitis) occurs 4-times more frequently in smokers than in non-smokers. Studies have also shown that in smokers 50-100% inhibition of the function of polymorphonuclear leukocytes (white blood cells which help fight infection) compared to non-smokers[11].

**Dental implants**

Tobacco can be damaging to both the initial and long-term success of dental implants. Indeed, in

one study smoking was the most significant factor predisposing implant failure - rates were 4.8% in non-smokers and 11.3% in smokers[12]. The higher failure rates associated with smoking is supposed to be due to the reduced bone density associated with smoking and vasoconstrective effect of smoking[13].

**Leukedema, Median-rhomboid glossitis, hairy tongue etc.**:



Though these conditions have a number of etiologies, smoking is one of the significant contributing factor.

**Effects on Taste and Smell:**

The smell and taste functions of smokers can be acutely affected by the gases and chemicals within tobacco and the ancillary particulate matter associated with smoking. The greater the amount smoked, the greater the impact, and only once smoking is stopped do these functions begin to improve again. A reduced ability to taste and smell may lead to potentially problematic changes in diet such as an increased use of salt[14].

**Chronic Sinus Infections:**

People who are especially sensitive to tobacco smoke can develop swelling in their nasal membrane and sinus cavities. Sinusitis occurs more regularly among smokers than non-smokers[9].

## CONCLUSION:

It is now clear that smoking has a number of defined effects on oral cavity. The association of smoking with development of pre-malignant lesions/conditions and true invasive carcinoma is one of the most important topic of concern because quitting smoking not only prevent/ minimizes the development, but also increases the chances of resolution of such lesions (during initial stages).

So educating people about oral effects of smoking, self identification of such lesions, motivating them to quit such habits, early diagnosis and treatment not only reduce the incidence of such effects/ lesions but also help to improve the quality of life of an individual.

## REFERENCE


1. US Department of Health and Human Services the Health Benefits and of Smoking Cessation. A report of the Surgeon General., Rockville, Meryland: US Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 1990.
2. Australian Institute of Health and Welfare (AIHW) and Australasian Association of Cancer Registries (ACCR) 1999. Cancer in Australian 1996. Incidence on mortality data for 1996 and selected data for 1997 and 1998. AIHW cat. No. CAN 7. Canberra: AIHW (Cancer Series), 1999.
3. Shah JP, Johnson NW, Batsakis JG. "Mechanism of Tobacco Carcinogenesis" in Shah JP, Johnson NW, Batsakis JG editor, Oral Cancer. 1st Ed: 47-48, 2003.
4. Watt R and Robinson M Helping Smokers to Stop: A guide for the dental team, London: Health Education Authority 1999,.
5. Blot WJ, Alcohol and Cancer, Cancer Research (Supp.) 1992, 52:2119s-2123s.
6. American Cancer Society, Oral Cavity and Oropharyngeal Cancer Resource Centre, URL:http://www.cancer.org Accessed October 1999.
7. Sophia S. Samet JM, Wang BS; Tobacco Smoking and Cancer: The Promise of Molecular Epidemiology, Salud Publica Mex, Vol. 39 (No. 4): 331-345, 1997.
8. Blot WJ, Mc Laughlin JK, Winn DM et al Smoking and Drinking In Relation To Oral and Pharyngeal Cancer, Cancer Research 1998,48:3282-3287.
9. Christen AG, Klein JA. Tobacco and Your Oral Health. Quintessence Book, Illinois; 1997
10. Preber H. Smoking and periodontal disease. Karolinska Institute, Stockholm; 1986
11. Palmer, RM. Tobacco smoking and oral health. Health Education Authority; Occasional Paper No.6; Nov 1987; p.17
12. Allard R, Johnson N, Sardella A et al. Tobacco and Oral Diseases – Report of EU Working Group, 1999. Journal of Irish Dental Association; 1999; 46(1): 12-23
13. Zarb GA, Bolender CL, Carlsson GE, "The Significance of Osseointegration" in Zarb GA, Bolender CL, Carlsson GE editors, Boucher's Prosthodontic Treatment for Edentulous patients, Ed 11: 504-505, St. Louis CV Mosby 1997.
14. Mecklenburg RE, Greenspan D, Kleinmann DV et al. Tobacco Effects in the Mouth. National Cancer Institute & Institute of Dental Research Guide; NIH Publication No.96-3330; 1996.