"Oral And Systemic Effects of Smokeless Tobacco Usage: A Review And Update."

Authors:

1. Dr.Krishnanand P S $_{MDS}$ Professor & Head, Dept of Oral Pathology & Microbiology, Dayananda Sagar College of Dental Sciences, Bengaluru, Karnataka
2. Dr. Vidya MA $_{MDS}$ Assistant Professor, Dept of Oral Pathology & Microbiology, Dayananda Sagar College of Dental Sciences, Bengaluru, Karnataka
3. Dr. Kumar G S $_{MDS}$ Retd. Principal, KSR Institute of Dental Sciences, Tiruchengode, Tamil Nadu

Introduction

Smokeless tobacco known also as chewing or spit tobacco, is usually placed in the mouth inside the cheek or between the cheek and gums. It is known to contain very many carcinogenic agents, of which the most deleterious are the group of tobacco-specific nitrosamines (TSNAs), while some others of interest are formaldehyde, acetaldehyde, arsenic, benzopyrene, nickel, and cadmium. India is the second largest producer of tobacco globally, next only to China. Tobacco occupies a predominant position in the Indian economy owing to its significant contributions in the agricultural and industrial sectors. **[1]**The Indian populace consumes tobacco in both the smoked & smokeless forms since ancient days for both therapeutic and non-therapeutic purposes. However in the recent years, therehas been a disturbing increase in the use of smokeless tobacco products in the Indian Subcontinent in the form of paan, gutkha, etc.This is a matter of great concern since the use of smokeless tobaccoproducts are associated with development of several lesions specially pertaining to the oral cavity.**[2]**It has long been a misconceived public notion that smokeless tobacco is somehow a safer form of tobacco when compared to the traditional smoked form of tobacco. However, Nicotine is found in smokeless tobacco too, like in all other tobacco products. Although it is absorbed at a considerably slower rate, 3 to 4 times more the quantity is absorbed from smokeless tobacco than from a cigarette & is also known to remain longer in the bloodstream which is partly a cause for tobacco addiction. Numerousepidemiological studies have shown a positive as well as a significant association of smokeless tobacco with a risk of developing oral cancer at one end of the spectrum to developing something as commonplace as even dental

caries at the other end with both being associated with chronic use of smokeless tobacco products. **[3]**

Smokeless tobacco is usually placed in the oral or nasal cavities against the mucosal sites that permit the absorption of nicotine into the human body.**[4]**It exists in two major forms:

i) Chewing tobacco and

ii) Snuff

Chewing tobaccois air-cured tobacco that is shredded into flakes and treated with sweet flavoring solutions. It consists of sweetened, coarsely ground tobacco leaves. It is used in the form of a 'chew' or a 'quid' that is chewed or held in the cheek respectively. There are 3 main forms of chewing tobacco: Plug tobacco, Loose-leaf tobacco &Twist or roll tobacco.

Snuff could either be dry or moist. Moist snuff which is more commonly used is taken orally. Dry snuff is inhaled through the nose.**[5]**Moist snuff consists of dark tobacco, finely cut, air cured and fermented. This is available in containers or as sachets**.**Dry snuff is fine cured, pulverized powder form of tobacco.**[6]**This article aims to compile relevant data on the oral as well as systemic changes caused due to smokeless tobacco usage done through the oral route.

Prolonged usage of smokeless tobacco products can have several adverse effects on health, with both oral and systemic manifestations.Oral effects could be classified as:

a. Effects on Dental hard tissues
b. Effects on periodontal apparatus
c. Non neoplastic Oral Mucosal lesions
d. Oral Mucosal changes with premalignant & malignant risk
e. Oral lesions showing malignant changes

The Systemic manifestations will be discussed under:

i. Non-oral cancers,
ii. Cardiovascular diseases,
iii. Respiratory changes,

iv. Nicotine addiction and

v. Other diseases.

Oral effects of Smokeless Tobacco:

A. Effect on Dental Hard Tissues:

Smokeless tobacco and dental caries:

Although much has been reported on the deleterious effects of various forms of tobacco on the soft tissues of the oral cavity and the respiratory tract, little research has been done on the caries promoting potential of tobacco on teeth. Two diverse opinions prevail, one of it states that use of tobacco increases caries while the other states that it does not. The latter opinion has often been extended to suggest that chewing tobacco will retard initiation of caries either through the mechanical cleaning action of tobacco on the teeth, through the increased salivary flow associated with the physical and chemical stimulation of salivary glands, through the frequent rinsing of the mouth when the "chew runs dry", or through a direct influence of tobacco on oral micro flora **[25]**Many studies were carried out to know the total sugar and fluoride contents in smokeless tobacco to contribute new basic information that might have helped to define the effect of tobacco on increasing or decreasing caries. Going R.E. et al reported that some tobacco products prepared for customer use were adulterated with additives. The authors said that among them, the additive that causes the greatest dental concern is the amount of sugar included to provide a pleasing taste to attract potential customers. **[25]**Hsu S.C. et al also agreed to the view held by Going R.E. et al, in that they also found sugars contained in smokeless tobacco that they related to the incidence of increased dental caries. They showed the presence of refined sugars like fructose, glucose, and sucrose in the chewing form of tobacco. They said that by maintaining a chaw of tobacco at a particular site in the oral cavity, and by replacing or freshening it with a new quid, a constant source of sugar was provided as a substrate for the metabolic activity of plaque microorganisms. They conducted Gas-liquid chromatographic studies showing all the above-mentioned sugars to be present in smokeless tobacco. **[25,26]**Lindemeyer R.G. et al assessed the in vitro effect of various forms of smokeless tobacco on growth of oral cariogenic Streptococci. He showed that

the chemical components of tobacco do not inhibit the growth of oral microflora. They also added that caries formation was a multifactorial process and many other factors besides S mutans and S. Sangius were required to determine the caries susceptibility of any individual. They quoted parameters like saliva flow, pH changes, erosion, abrasion and attrition of tooth surfaces as well as the type and quantity of bacterial populations in patients using various forms of tobacco to influence caries formation. **[27]**Oppenbacher and Weathers conducted a survey in a school population to investigate the use of smokeless tobacco products and their ill effects. The overall prevalence of ST usage was 13.3% and was associated with a 1.6 fold elevation in mean DMF scores among them. They concluded saying that ST usage was a significant risk factor as far as caries incidence is concerned in individuals. **[28]**Falker W.A. Jr et al studied the effect of smokeless tobacco extracts on the growth of oral Streptococci, used aqueous tobacco extracts to supplement a basic salt solution and a microbial medium. They performed chromatography, which revealed sucrose on the extract and these extracts helped the growth of S.mutans, S.salivarius & S.sanguis. They concluded that the extracts of ST would serve as a growth substrate for these three species of oral Streptococci, which were frequently associated with dental caries. **[29]**Hirsch J.M. et al in conducted a survey on 2145 patients aged 14-19 yrs to evaluate the tobacco habits among teenagers and their possible association with dental caries. They observed that all caries epidemiological data were significantly higher among patients with tobacco habits compared to non-users. However, they also maintained that, dietary and other oral habits were to be further elucidated before any definite conclusions could be made regarding the effect of tobacco per se on development of caries. **[30]**Tomar S.L. et al conducted a study to examine the relationship between chewing tobacco use and dental caries. Their findings suggested the use of chewing tobacco might have been a risk factor for developing root surface caries and to a lesser extent coronal caries. They explained the association between ST and caries to be due to the high levels of fermentable sugars in them, which stimulated growth of cariogenic bacteria. They also found that extracts of ST were related to an increase in the in vitro growth of lactobacillus casei, a bacterium implicated in root surface caries. Their study also found significantly higher levels of collagenase on the side of the mouth where ST was placed. The authors speculated that increased collagenase activity

might interact with specific bacteria to enhance the development of root caries due to the organic nature of cementum. One more possible contributory mechanism in development of root surface caries, according to the authors, was gingival recession, which exposes the root surface to cariogenic bacteria. Gingival recession was reported among people who used either snuff or chewed tobacco. **[31]**In a case control study carried out in Karnataka among smokeless tobacco chewers , the oral micro biota, dental caries and periodontal status were evaluated. Caries experience among chewers was significantly less at 61.9% as compared to non-chewers with 90.5%. 26.2% of chewers and 19.1%. nonchewers were found to have deep pockets measuring > 5.5 mm on a CPI probe . Counts of Lactobacillus species were significantly lower among chewers than among non-chewers.**[32]**

Smokeless Tobacco and Attrition & Cervical Abrasion

Attrition, the physiologic wearing of teeth associated with mastication that affects primarily occlusal or incisal surfaces, has been associated with smokeless tobacco use. Robertson P B et al also recorded loss of tooth substance on all buccal sites resulting in smooth, hard depressions adjacent to the cemento-enamel junction. It was observed that loss of tooth structure generally occurred on the root surface in areas of recession. **[22]**.

B. Smokeless tobacco and its effect on periodontium:

Unlike cigarette smokers who experience widespread periodontal destruction, the oral effects of smokeless tobacco are localized to the site of placement. The primary periodontal alteration in smokeless tobacco users is localized gingival recession. In general, it occurs in 25-30% of the Smokeless Tobacco users **[21]**Robertson P.B. et al who studied the periodontal effects associated with smokeless tobacco in detail found that the major effects of smokeless tobacco were gingival recession and associated loss of attachment. This loss of periodontal tissues was localized to areas adjacent to mucosal lesions, areas that in turn correspond to the placement of the smokeless tobacco quid. It was also found that use of smokeless tobacco was not necessarily associated with other forms of periodontal disease. Poor oral hygiene and presence of gingivitis were also not related to occurrence of mucosal lesions in smokeless tobacco users. **[22]**With regard to attachment loss, Robertson et al observed about 90% of the

sites with attachment loss exceeding 3mm and attributed it to be a function of recession.However, severe forms of early onset or rapidly progressive periodontal disease were not observed in any of the subjects. **[23]**Nociti et al. investigated the effect of nicotine administration on periodontal breakdown resulting from ligature‐induced periodontitis in rats. Daily administration of nicotine enhanced, in a dose‐dependent manner, the effects of local factors in producing periodontal breakdown. Furthermore, the nicotine seemed to have a direct deleterious effect on the periodontal tissues.[25]

Smokeless Tobacco and Gingival Recession

Smokeless tobacco has also been associated with localized gingival recession21 and could lead to mucogingival defects and caries. Recession can be corrected surgically, and numerous studies22 describe methods to address the loss in gingival tissue height that arises from frequent and repeated tobacco use in the same intraoral location. Cessation of tobacco use will not reverse gingival recession. Soft tissue pathoses, dentin hypersensitivity, surgery, and caries originating from tobacco use can be avoided by not using smokeless tobacco.

Laboratory based research assessing the relationship between smokeless tobacco and gingival recession has led to an understandingthatperiodontal breakdown and gingival recessionis triggered by exacerbated inflammatory responsesto smokeless tobacco productsat the site of placement. Mechanical trauma induced by the abrasive nature of the smokeless tobacco products which are held in close proximity to the thin gingival tissues is also considered a contributory factor to recession.**[24]**

Malagi et al in their study carried out to evaluate and compare the effects of smokeless tobacco on oral hygiene status, gingival health, and periodontal status concluded that smokeless tobacco may indeed negatively influence the same.

C. Non Neoplastic oral mucosal lesions:

There are a few smokeless tobacco associated lesions, which over the years have been proven to have very little or no risk of turning malignant at al. Some of these lesions are Betel Chewer's Mucosa (BCM), tobacco pouch keratosis, leukoedema, etc.

BCM, first described and defined by Mehta et al in 1971 is clinically characterized by a brownish-red discoloration of the oral mucosa covered with an irregular epithelial surface having a predisposition to desquamate or peel off. (Mehta FS, Hamner JE. Tobacco related oral mucosal lesions and conditions in India. A guide for dental students, dentists and physicians. Mumbai: Published by basic dental research unit, Tata institute of fundamental research;1993) The buccal mucosa is most frequently affected. The etiology is thought to be more due to local trauma caused by quid placement and the act of chewing and also possibly due to the chemical nature of the mixture that is chewed. (Trivedy CR, Craig G, Warnakulasuriya S. The oral health consequence of chewing areca nut. Addict Biol 2002;7:115-25). Discontinuing the habit almost always leads to resolution of this so called lesion.

Tobacco pouch keratosis, also called as Snuff Dipper's Lesion is clinically characterized by a thin grayish white translucent plaque that is soft on palpation. Stretching of the mucosa often reveals a distinct "pouch" caused by flaccidity in chronically stretched tissues in the area of tobacco placement. Habit cessation however results in resolution of this lesion and reverting back of the affected mucosal areas to normal within 2 weeks in 98% of the lesions.

Leukoedema is a grayish-white lesion of the oral mucosa. It was once thought to be a forerunner to the formation of leukoplakia, however it has been proven over the years that careful clinical examination differentiates it fromany form of precancer. (Martin JL. Leukoedema: A Review Of The Literature J Natl Med Assoc. 1992;84:938-940). Although of unknown etiology, local irritation has been suggested as one of the main reasons for leukedema to develop. When associated with tobacco chewing, repeated accumulation of tobacco results in a chrocic irritation to the affected mucosal site which then shows leukedematous changes. However, careful clinical differential diagnosis helps distinguish this lesions from most other precancerous ones

Smokeless tobacco and oral leukoplakia:

The prevalence of leukoplakia and mucosal disease in regular Smokeless Tobacco users ranges from 8-43% **[7]**. In 2005 WHO defined it as "a white plaque of questionable risk having excluded other known diseases or disorders that carry no increased risk of cancer". **[8]**Of the various oral effects of smokeless tobacco, Oral Leukoplakia is of most concern because of its potentially malignant nature.

A number of studies have been done which strongly implicate smokeless tobacco as an important causative factor in the development of oral leukoplakia.

Zaridze D.G et al in 1986conducted a study on a population with high incidence of oral cancer. He found out that a high proportion of men among his study group used 'Nass', a mix of tobacco, lime, ash and cotton oil. He showed through his study that nass use increases the frequency of micronucleated cells in the exfoliated sublingual cells. Also, that nass had a clastogenic and genotoxic effect. He concluded that nass use did induce oral leukoplakia.**[9]**

Wolfe MD and Carlos JP (1987) in their study on native Navajo Indians observed that duration (in years) and frequency (in days/ week) of smokeless tobacco use were highly significant factors associated with leukoplakia. **[10]**

In 1991, Commissionat Y studied the consequences of chewing tobacco in France and noted that reactional keratosis and the risk of malignant transformation were some of the risks of tobacco chewing. **[11]**

Tomar SL et al 1997 **,** with help of data from the 1986-87 National Survey of oral health in US school children examined the cross sectional relationship between use of tobacco and alcohol and presence of white or whitish oral soft tissue lesions. He found the lesions to be strongly associated with duration, monthly frequency and daily minutes of snuff use and tobacco chewing. He also observed that use of either of these forms of tobacco exhibited a dose-response relationship with occurrence of lesions. **[12]**

Zhang X et al in 2001 described a 'Shammah' induced oral leukoplakia like lesion in a 44 years old Algerian patient. 'Shammah' is a chewing tobacco form consisting of powdered tobacco leaves with carbonate of lime and other substances. It has been associated with oral cancer in Saudi Arabia. He observed that Shammah seemed to cause changes, which he thought might transform into oral cancer. He also was of the view that such lesions should be considered precancerous, which was concurred by Bethke G. and Reichart PA in 2004. **[13]**

Thomas G. et al in 2003 conducted a case control study on a group of 115 patients with oral premalignant lesions in Kerala, India. He did this as a study to ascertain the risk factors of premalignant lesions. His patient group included patients with oral leukoplakia, OSMF, erythroplakia and a combination of these. He observed a dose-response relationship between the frequency and duration of tobacco chewing with risk of multiple oral premalignant lesions. He then concluded that tobacco chewing was the most important risk factor for multiple oral premalignant lesions and that it may be a major source of field cancerization on the oral epithelium in the Indian population. **[14]**

Shiu MN et al and Avon SL in 2004, separately conducted studies on betel quid with tobacco chewing habit and concurred that chewing betel quid placed the subject at a higher risk of developing oral leukoplakia and also oral cancer eventually.**[15]**

Smokeless Tobacco & Oral Submucous Fibrosis (OSMF)

OSMF, a potentially malignant disease, first described in the 1950's, owes much of its development to the habit of chewing areca nut. And since areca nut is almost always an ingredient in any of the smokeless tobacco mixtures, it assumes significance all the more. It is a debilitating disease that is characterized by many features like burning sensation of the mouth, reduced mouth opening, blanched and taut fibrosis of the mucosa that at times extends all the way into the upper third of the esophagus at times. This is a multifactorial disease with areca nut playing a role of leaching out certain chemicals along with being abrasive to the mucosal surface, leading to trauma & injury of the local soft tissues. In this situation, tobacco with its carcinogens find an easy portal of entry into the tissues thereby increasing the risk of malignant

transformation potential, first explained by Paymaster in 1956 & later confirmed by various other studies too.

Smokeless tobacco and Oral cancer:

Unlike tobacco smoke, smokeless tobacco is not a combustion product and hence the latter contains less complex carcinogens compared to the former. The carcinogens known to be present in the types of smokeless tobacco used for snuff dipping are nitrosamines, aldehydes, polonium-210 and polynuclear aromatic hydrocarbons**[16]**NNN and NNK are the only carcinogens in smokeless tobacco that have been shown to induce oral cavity tumors in lab animals**[17]**. The presence of NNN and NNK in saliva of snuff dippers has been confirmed **[18]**. Metabolic studies with human tissues have demonstrated that buccal mucosa can activate NNN and NNK to intermediates that can bind to DNA **[19]**. Taken together these data provide strong support for role of NNN and NNK as causative factors in oral cancer induction by smokeless tobacco **[19]**

The most common forms of cancer associated with the use of smokeless tobacco are verrucous carcinoma and squamous cell carcinoma. They usually tend to occur in sites where the quid is held as compared to oral cancers developing in response to other factors and most commonly occur on the tongue, tonsils retro molar area and floor of the mouth.

In a study by McGuirt and Wraythe records of 128 patients were evaluated.78% of these patients had used smokeless tobacco for 40years or more .Among them, a five-year survival rate of 37% and three-year survival rate was 47% was noted.40% presented with leukoplakia, erythroplakia or both at the time of clinical examination. This study concluded suggesting that there exists a strong association between use of smokeless tobacco and oral cancer.**[5]**

Newell Johnson in 2001stated that any form of tobacco acts potentially as a risk factor for oral cancer, while snuff lies on the lower end of the scale. He added that alcohol synergizes with tobacco and is responsible for all upper aero digestive tract squamous cell carcinomas. **[20]**

Systemic effects:

Smokeless tobacco products are meant to be generally expectorated, but some of the tobacco is swallowed by the user**[33]**. As suggested in an in vitro porcine mucosal model, NNN can be absorbed through the oral mucosa. The formation of hemoglobin adducts to NNN suggests that carcinogens from smokeless tobacco reach the circulation and are available to body tissues **[34]**. Blood levels of Nicotine & Cotinine in smokeless tobacco users compared with cigarette smokers shows that there is considerable gastrointestinal absorption of nicotine from smokeless tobacco **[33]**

Ames Salmonella test showed significant increase in the carcinogenic activity in urine during the use of chewing tobacco as well as smoking cigarettes. **[33]**

These studies can support the hypothesis that smokeless tobacco may increase the risk of cancer in other anatomical sites which includes larynx, esophagus, prostate, bladder and colon.

Laryngeal and Esophageal cancer:A report was submitted in 1986 by the Advisory committee to the Surgeon General described the effects of smokeless tobacco. This report included a study on nasal sinus cancer, five studies on laryngeal and esophageal cancer and three on stomach cancers. Although many of these studies showed an increase in the risk associated with use of smokeless tobacco products, these studies, collectively for each cancer type, were not definitive due to lack of control of confounding factors, small sample sizes, inconsistencies regarding dose-response relationships or findings for snuff versus chewing tobacco **[34]**

Prostate cancer: Winn DM, reviewed studies on smokeless tobacco and prostate cancer and concluded that they did show a positive association between using smokeless tobacco and having or dying from prostate cancer. He also singled out one study in which he said that a dose-response relationship was evident in it, which examined the extent of smokeless tobacco use. **[34]**

Colorectal cancer:A cohort study with a 26 year follow up and almost 3,00,000 study subjects examined the role of various forms of tobacco and colorectal cancer. Relative risk for colon and rectal cancers are 1.2 & 1.4 for users of smokeless tobacco compared to non-users. Relative risk was higher for people who were not the heaviest users, indicating no dose-response. The

authors concluded that there was little evidence of a role of smokeless tobacco in colorectal cancer etiology.**[35]** An earlier study by Williams and Horm in too indicated little, if any relationship between smokeless tobacco and colorectal cancer. **[36]**

Bladder cancer: The advisory committee to the surgeon general reviewed eight published studies of smokeless tobacco and bladder cancer (1986). They concluded that there was no consistent evidence that the habit affects the risk of this cancer. In addition, two other studies conducted by Mommsen and Aagaard in 1983 andSlattery et al in 1988 conducted also supported the finding of the committee. **[37, 38]**

Pancreas: Two studies conducted by Heuch et al and Williams and Horm found an association between smokeless tobacco and pancreas cancer, but the number of pancreatic cancer patients was very small to arrive at any definitive conclusion. **[39,36]**

Other cancers: Smokeless tobacco use was associated with cancer of the Ampulla of Vater in a case control study of biliary tract cancer, but all cases smoked as well.**[40]** No evidence of smokeless tobacco risk was apparent in a single study of acute leukemia from a Swedish case control study **[41]**. Cervical cancer was linked to smokeless tobacco use by women in a study based on cancer patients from Williams and Horm **[36]**. A hospital based case control study of renal cell carcinoma conducted between 1977 and 1993 by Muscal et al strongly associated chewing tobacco among males with the risk of developing renal cancer.

Cardiovascular effects of smokeless tobacco:

Smokeless tobacco contains nicotine. The levels of nicotine in smoking tobacco and in smokeless tobacco are generally similar, but overall exposure may be greater in smokeless tobacco users due to prolonged absorption of the nicotine. The cardiovascular effects associated with nicotine from smokeless tobacco include increase in heart rate and blood pressure **[34]**

Nicotine is also known to increase risk of atherosclerosis and acute cardiovascular events through numerous potential channels. Atherosclerosis could be promoted through its effect on lipid metabolism, platelet hyper-reactivity and endothelial injury due to heart rate and increase

in cardiac output. Myocardial infarction could occur as a result of nicotine-mediated thrombosis, excessive oxygen demand and/or coronary spasm**[34]** Nicotine is also known to aggravate underlying hypertension by increasing cardiac output and constricting blood vessels .**42**

Several studies have examined the prevalence of cardiovascular risk factors among smokeless tobacco users. In a study conducted by Siegel et al it was found that smokeless tobacco users were similar to non-users in total cholesterol, high-density lipoproteins, pulse rate and diastolic and systolic blood pressure. **[43]**, In contrast two other studies, Bolinder et al &Tucker found that smokeless tobacco users had elevated cardiovascular risk factors. **[44,45]**

Swedish investigators in a study noted that smokeless tobacco users were 40% more at risk of death from cardiovascular disease than non-users were.[**34**]

Sodium in smokeless tobacco is part of an alkaline buffer that facilitates the buccal absorption of nicotine. It was noted that an average of 26 & 41mEq of sodium, was absorbed during daily use of oral snuff and chewing tobacco respectively. This level of sodium consumption, the authors stated, could contribute to hypertension in salt sensitive individuals and could aggravate congestive heart failure or other edematous states in individuals with such medical conditions**.[42]**

Smokeless tobacco: an addicting drug

In 1986, a report from the consensus conference at the National Institutes of Health, USA independently concluded that smokeless tobacco was addicting and shared many characteristics in common with drugs such as cocaine, heroin, and alcohol.

The evidence included epidemiological data showing that, patterns of smokeless tobacco use bore key similarities to patterns of use of other addictive drugs. Smokeless tobacco provided users with doses of nicotine known to be addicting, and that clinical signs of dependence and withdrawal for smokeless tobacco were similar to those for cigarettes.

Some of the main evidence comes from clinical and laboratory studies on the effects of nicotine and nicotine delivery by smokeless tobacco products. Smokeless tobacco follows an abuse pattern similar to that of addictions to other drugs of abuse (USDHHS, 1986). Use of smokeless tobacco leads to the development of tolerance, causing the user to use stronger products to achieve the same effects as addiction progresses. A withdrawal syndrome associated with smokeless tobacco use has been identified that is qualitatively similar to that from cigarettes **[46]**. This suggests that individuals may continue to use smokeless tobacco products despite known health risks in part to alleviate withdrawal symptoms. Finally, smokeless tobacco products can provide nicotine in doses that are known to be psychoactive in humans.

Other diseases

The relationship of smokeless tobacco to inflammatory bowel disease has been studied in one epidemiological investigation in Sweden. This study was undertaken because of two other studies that linked cigarette smoking to these digestive disorders. Sixty-three people with Crohn's disease and eighty-two people with Ulcerative Colitis were chosen for the study. Smokeless tobacco in combination with cigarette smoking was associated with a risk of about three fold for Ulcerative Colitis and Crohn's disease **[34]**, but smokeless tobacco alone showed no risk for developing these diseases**.**

Conclusion :

The various forms of smokeless tobacco products are said to contain about 3000 chemical constituents and prolonged usage of these products increases the risk of oral, esophageal, lung and pancreatic cancers and even stroke. Schools, colleges, hospitals, NGO'sand policy makers must come together in an effort to promote anti-tobacco education through well organized outreach programmes. These programmes can help educate the general public regarding the health hazards stemming from tobacco use and also the signs and symptoms of tobacco-related cancer.There is definitely a huge responsibility on the part of dental professionals to detect lesions associated with the use of smokeless tobacco products and inform their patients about its potential harmful sequale. When dental professionals help their patients in discontinuing the

tobacco habit, they eliminate a substantial causative/contributing factor for a number of adverse oral and systemic conditions, including cancer.

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