Review of Literature

**REVIEW OF LITERATURE**

*Chiodo et al.* (1986) revealed in the study that oral examination often includes looking for leukoplakia and erythroplastic lesions, which can progress to cancer.

*Zheng et al.* (1993) reported that poor dentition emerged as a strong risk factor for oral cancer: the odds ratio (OR) for those who had lost 15 – 32 teeth compared to those who had lost none was 5.3 for men and 7.3 for women and the trend was significant (*P* <0.01) in both genders. Those who reported that they did not brush their teeth also had an elevated risk (OR =6.9 for men, 2.5 for women). Compared to those who had no oral mucosal lesions on examination (OR=1.0), persons with leukoplakia and lichen planus also showed an elevated risk of oral cancer among men and women. Denture wearing *per se* did not increase oral cancer risk (OR=1.0 for men, 1.3 for women) although wearing metal dentures augmented risk (OR=5.5 for men). These findings indicate that oral hygiene and several oral conditions are risk factors for oral cancer independently of the known risks associated with smoking and drinking.

*Choi and Kahyo* (1991) revealed in the study that the risk of cancer of the oral cavity, pharynx and larynx rose for current smokers and declined for ex-smokers. In males the odds ratios (ORs) for these sites rose with duration of smoking and number of cigarettes smoked per day. The relationship is strongest for laryngeal cancer. The risk for all sites was elevated linearly as amount and frequency of alcohol intake increased. Heavy drinkers, i.e. males who drank 90 g ethanol daily had an approximately 15-fold risk of cancer of the oral cavity, an 11-fold risk of pharyngeal cancer and an 11-fold risk of laryngeal cancer compared with non-drinkers. Alcohol drinking was a much stronger risk factor for cancer of the oral cavity than cancer of the pharynx and larynx. Alcohol was a much weaker risk factor for laryngeal cancer than cigarette smoking. Cancer of the oral cavity, pharynx and larynx also showed an interaction between smoking and alcohol, suggesting a synergistic effect.

*Maier et al.* (1992) reported in a case-control studies on the relative risk of head and neck cancer in association with tobacco and alcohol consumption. Of the tumour patients, 4.5% had never smoked, in contrast to 29.5% of the control group. The average tobacco and alcohol consumption of the patients was approximately twice as high as in the control subjects. The highest alcohol and tobacco consumption was
observed in patients suffering from oropharyngeal cancer. Tobacco and alcohol increased the risk of head and neck cancer in a dose-dependent fashion and acted as independent risk factors. In heavy smokers (greater than 60 pack-years) a relative risk of 23.4 (alcohol adjusted) was calculated. Combined alcohol and tobacco consumption showed a synergistic effect. The risk ratio increased more in a multiplicative than in an additive manner. Oral and laryngeal cancer were associated with the highest tobacco-associated risk values. The highest ethanol-associated risk values were associated with oropharyngeal and laryngeal cancer.

Benhamou (1993) reported that tobacco smoking is the major cause of lung cancer. Cigarette smokers have a risk of lung cancer 10 to 15 times greater than nonsmokers. Tobacco and alcohol are the main risk factors for cancers of oral cavity, larynx, pharynx and oesophagus (cancers of the upper respiratory and upper digestive tract). For these cancers, the risk associated with tobacco was about 2 to 4 among people who drink little or no alcohol. Risks of lung cancer and of cancers of the upper respiratory and upper digestive tract increase with an increasing number of cigarettes smoked per day and duration of smoking. Among males, lung cancer mortality increased regularly over time and today, lung cancer is the leading cause of death and illness from cancer.

Nakamizo et al. (1993) reported that the incidence of second primary cancers was significantly (p < 0.05) higher in patients having a lesion at the tongue (n = 502), mesopharynx (n = 188), hypopharynx (n = 224) and larynx (n = 621) and in the oral cavity (n = 203) than in the general population. With regard to organ association in multiple primary cancers, cancers in the oral cavity, esophagus and at the pharynx were found to occur as second primary cancers of the tongue, oral cavity, meso-hypopharynx and larynx at significantly high rates in males. Male laryngeal cancer was related to the lung. The incidence of second primary cancer was very high in persons who had been heavy smokers and/or heavy drinkers before the occurrence of their primary cancer in the head and neck region. In these people, second primary cancers were found to occur at high rates as cancers related to tobacco smoking and drinking. Otherwise, in cases with head and neck cancers less related to tobacco smoking and drinking, the incidence of second primary cancers was low. From the above results, tobacco smoking and drinking may be closely related to a high
incidence of second primary cancer following the first primary cancer in the head and neck region.

**Winn (1995)** observed in the study a protective effect of a diet high in fruit intake, reflected in a 20-80% reduction in oral cancer risk. A high intake of foods considered to be dietary staples in particular cultural groups indicating a generally impoverished diet, has been linked to excess risk. Indigenous dietary practices were found to increase risks include a high intake of chili powder and wood stove cooking. Supplementation with various vitamins has been protective in a few studies. The most consistent dietary findings across multiple cultural settings are a protective effect of high fruit consumption and the carcinogenic effect of high alcohol intake.

**Winn (1995)** observed in the study that chemoprevention trials generally have found that chemopreventive agents reduce the size of oral leukoplakia lesions or the frequency of second primary oral cancers.

**Bundgaard et al. (1995)** reported that among indicators of dental care, the use of a toothbrush, gum bleeding and number of missing teeth were associated with oral cancer risk after adjustment for smoking, drinking and chewing habits.

**Tilashalski et al. (1995)** estimated in the study that, if all US smokers switched to ST use, 12 000 cases of oral cancer would occur each year, representing only 5% of all tobacco smoking related lung cancers and 50% of the oral cancers now attributed to cigarette smoking.

**Ziegler et al. (1996)** reported that increased vegetable and fruit intake is associated with reduced risk in men and women in various countries in smokers, ex-smokers, and never-smokers and for all histologic types of lung cancer. Low levels of blood beta-carotene are predictive of increased lung cancer incidence. Diets high in fat, saturated fat, and cholesterol may increase the risk of lung cancer and that the effect is not mediated through vegetable and fruit intake. Beta-carotene can function as an antioxidant other micronutrients with this potential specifically vitamins E and C and selenium also have been proposed to reduce lung cancer risk.

**Wu and Du (1996)** suggested that coal burning at home is a significant risk factor for the development of lung cancer in non-smoking females. Coal smoke contains many potential carcinogens like radon and thoron.
Parkin et al. (1997) reported that cancer of the oral cavity and pharynx is the first and third commonest cancer in Indian men and women.

Stefani, et al. (1997) reported that diets high in fat, saturated fat and cholesterol may increases the risk of lung cancer and the effect is not mediated through vegetable and fruit intake.

Gogos et al. (1998) concluded that omega-3 poly unsaturated fatty acid (PUFA) had significant immuno modulating effect and seemed to prolong the survival of malnourished patients with generalized malignancy.

Knyazhev et al. (1998) reported the principal causes of morbidity include cardiovascular diseases and cancer, the development of which is associated to nutrition. The nutritional state of most Russians is inadequate due to low intake of nutrients especially vitamins, major and trace elements essential proteins and improper ratios among individual nutrients.

Levi et al. (1998) reported that serving per day of fruit or vegetables was associated with an about 50% reduction in oral cancer risk. The most favourable diet for oral cancer risk is therefore given by infrequent consumption of red and processed meat and eggs and, most of all, frequent vegetable and fruit intake. Diet diversity was inversely related to oral and pharyngeal cancer: ORs were 0.35 for the highest tertile of total diversity, 0.24 for vegetable and 0.34 for fruit diversity. In terms of attributable risk, high meat intake accounted for 49% of oral and pharyngeal cancers in this population, low vegetable intake for 65% and low fruit intake for 54%.

Smith (1998) suggested in the study that the dietary factors to be related to cancer are fruits, vegetables and grains appear to be protective, whereas fat and alcohol appear to increase the risk for developing a number of cancer.

Nyberg, et al. (1998) concluded that increased vegetable and fruit intake is associated with reduced risk in men and women; in various countries; in smokers, ex-smokers and never smokers; and for all histologic types of cancer.

American Cancer Society (1999) concluded four basic nutrition guidelines: choose most of the foods you eat from plant sources, limit your intake of high fat foods,
particularly from animal sources, be physically active, achieve and maintain a healthy weight, limit consumption of alcoholic beverage, if you drink at all.

Rapiti et al. (1999) concluded that environmental tobacco smoke exposure during childhood is strongly associated with the risk of later development of lung cancer (OR 3.9, 95% CI=1.9-8.2).

Gammon et al. (1999) reported that alcohol consumption has however been linked to higher serum estrogen levels: women with consistently high estradiol level have a significant higher average alcohol intake (92.8g/wk) than those with consistently lower estradiol levels (alcohol intake 31.6g/wk).

Giovannucci (1999) found in the study that higher intake of tomatoes or higher blood levels of lycopene correlated with protection from cancer of the breast, cervix, mouth and colon.

Junxan (1999) reported that selenium found in garlic and onion causes tumor cells to commit suicide. In addition, selenium seems to inhibit the formation of blood vessels in cancerous tissue.

Nebraska and Yan (1999) concluded that high selenium soyabean can help to prevent metastasis in cancer patients and the development of cancer in the general population.

Peter and Newburger (1999) reported that selenium is a potent antioxidant, which can block cells DNA damage that may lead to cancer. Selenium works as a cofactor to enzymes that are important in breaking down the free radicals that can damage cells.

American Cancer Society (2000) recommended that women maintain a healthy weight and limit intake of high fat foods, particularly those from animal source, as part of a healthy lifestyle.

American Cancer Society (2000) reported that an estimated 1,220,100 new cases of non-cutaneous cancer were diagnosed in the year 2000. in addition, an estimated 1.3 million cases of squamous cell and basal cell carcinoma of the skin was diagnosed.

Hindustan Times (2000) reported that diet is now being considered as a major weapon against cancer. Although we Indian are much better equipped to take care of
our diet vis-à-vis cancers by virtue of being conventionally vegetarian and having abundance of fresh vegetable and fruits, bringing with it a learning towards junk food, is unfortunate and needs timely intervention.

Jacobs et al. (2000) reported that whole grain intake protects against various cancers.

Kerry (2000) revealed in the study that exercise has a protective effect on a broad range of equality of life parameters after patients are diagnosed as having cancer. The general exercise prescription is moderate intensity exercise, 3 to 5 days per week 20-30 minutes per session. Conditions that warrant prescription modification include fatigue periods during treatment, acute or chronic physical impairments that may have resulted from surgery or adjuvant therapy, and the presence of bone cancer. Physicians who prescribe exercise improve motivation in their patients who have cancer.

Zhong et al. (2000) studied and reported that Environmental tobacco smoke is a known lung carcinogen. Environmental tobacco exposure carries a relative risk of development of lung cancer of 1.48 (1.13-1.92) in males and 1.2 (1.12-1.29) in female.

Marchand et al. (2000) reported that consumption of flavonoid-rich onion and apples contain large amount of one flavonoid called quercetin has been associated with protection against cancer.

Moore, et al. (2000) reported that the annual incidence of oral cancer in men in India is estimated to be 10 per 1,000,000.

Rahman, et al. (2000) concluded that bidi smoking is also considered to cause about 2-3 times greater nicotine and tar inhalation than do conventional cigarette, due to the poor combustibility of the bidi wrapper and greater puff frequency needed to keep the bidi alight and have various cancerous and chronic conditions in comparison with other cigarettes.

Morse et al. (2000) concluded in the study an inverse association of fruit intake and the development of oral cancer, particularly in those who use tobacco. Fiber, in the form of vegetable intake, has similarly been shown to be associated with a decreased
risk of oral cancer. It is estimated that intake of fruits and vegetables may lower the risk of development of oral cancer by 30% to 50%.

**Kumar (2000)** reported that in India in each year tobacco use results in about 160,000 cases of cancer, 4.5 million heart disease 3.9 million chronic obstructive lung diseases.

**Franceschi et al. (2000)** reported that cancer of the oral cavity and pharynx is the first and third commonest cancer in Indian men and women, respectively. Whereas in most areas at high risk for cancer of the oral cavity other than India (e.g., central and Eastern Europe, South America), the ratios between male and female incidence rates range between 3 and 10, in India the male-to-female ratio is approximately 1 (e.g., Madras) or lower than 0.5 (Bangalore).

**Kumar (2000)** reported that in India each year tobacco use results in about 160,000 cases of cancer, 4.5 million heart disease 3.9 million chronic obstructive lung diseases.

**Boucher (2001)** reported that the majority of ST studies have been carried out in India where both the habits and the negative outcomes (mainly oral cancers) are most prevalent, and strong dose dependent associations have been found. Tobacco here is generally used with other substances such as areca nut and lime, which may themselves be associated with oral disease.

**Mack (2001)** reported that very high incidence rates in Indian women reflect the persistent importance in India of paan chewing, a habit that is equally common in the genders. Paan generally includes calcium hydroxide, areca nut (from the *Areca catechu* tree) and betel leaf (from the *Piper betle* vine). Tobacco and/or various spices are commonly added.

**Gupta et al. (2001)** reported that among risk factors for lung cancer, cumulative exposure of > 45 years to indoor air pollution in women from use of coal or wood for cooking or heating showed an OR of 1.43 (95% CI=0.3-6.30)45. Residence in urban areas, however, did not entail an increased risk for developing lung cancer.

**Mork et al. (2001)** concluded that the primary risk factors for oral cancer in men and women are tobacco (including smokeless tobacco) and alcohol use. Infection with
human papillomavirus 16 has been associated with an excess risk of developing squamous cell carcinoma of the oropharynx.

Elizabeth (2001) reported that the goal of cancer screening are to reduce morbidity and mortality from the disease through the application of a screening test or examination that can detect the disease at an earlier stage in an asymptomatic patient population. The effectiveness of a screening test can be proved by its ability to decrease the disease related morbidity in a screened population as compared to unscreened population.

Khuri et al. (2001) reported that lung cancer is the leading cause of cancer deaths in developed countries and is also rising at alarming rates in developing countries. Deaths due to lung cancer are more than those due to colorectal, breast and prostate cancers put together. Incidence and mortality from lung cancer in females is rising while it is declining in males in developed countries. This is the single most devastating cause of cancer-related deaths2 with approximately 1.5 million cases world-wide and more than 1.3 million cancer-related deaths in 2001.

Srilakshmi (2001) suggested in the study that a dietary guideline a judicious inclusion of fruits, vegetables, whole grains in our diet rather than pills containing nutrients is suggested. In addition participation in regular physical activity may reduce a person’s risk for developing cancer. She further reported that some foods may cause cancer in some of the following ways- by being direct carcinogen, carcinogen may be produced by cooking, micro organism may be produce carcinogens in stored foods, food stuff may act as substrates for the formation of carcinogen in the body, food stuff may alter the bacterial flora of the bowel, thereby producing carcinogen.

Srilakshmi (2001) suggested that in many families there is a strong hereditary tendency to cancer. This probably results from the fact that most cancer require not one mutation but two or more mutations before cancer occurs. In those families that are particularly predisposed to cancer the genes are already mutated in the inherited genome.

Johnson (2001) reported in the study that in both genders, cancer of the mouth and pharynx ranks sixth overall in the world. It is also the third most common site among males in developing countries. In industrialized countries, men are affected two to
three times as often as women, largely due to higher use of alcohol and tobacco. Ethnicity strongly influences prevalence due to social and cultural practices, as well as socioeconomic differences. In terms of etiology, the effects of tobacco use, heavy alcohol consumption, and poor diet together explain over 90 percent of cases of head and neck cancer. All forms of tobacco represent risk factors for oral cancer. Alcohol synergizes with tobacco as a risk factor for all upper aerodigestive tract SCC: this is super-multiplicative for the mouth, additive for the larynx, and between additive and multiplicative for the esophagus. The increase in oral cancer in the Western world has been related to rising alcohol use.

Bagnardi et al. (2001) found in the study that alcohol most strongly increased the risks for cancers of the oral cavity, pharynx, esophagus, and larynx. Statistically significant increases in risk also existed for cancers of the stomach, colon, rectum, liver, female breast, and ovaries. Concurrent tobacco use, which is common among drinkers, enhances alcohol’s effects on the risk for cancers of the upper digestive and respiratory tract.

Fenley et al. (2001) concluded that incidence rates for oral cancer in India are among the highest in the world.

Gajalakshmi, et al. (2001) reported that in India the most frequently reported cancer sites in males are lung, oesophagus, stomach and larynx. In females, cancers of the cervix, breast, ovary and oesophagus are the most commonly encountered.

Jeng et al. (2001) reported that Betel-nut chewing is associated with an increased risk of squamous cell cancer of the head and neck.

Balaram et al. (2002) reported that oral cancer cases reported significantly fewer years of education than control subjects. The difference was more marked in women (OR for 0 versus 7 years of education 5.5) than men (OR 2.1). Lack of oral hygiene, no use of toothbrush is accounted for 32% of oral cancer in men and 64% in women. All together, the factors above seemed to explain 76% of oral cancer in males and 95% in females.

Abrol and Khanna (2005) studied the effect of smoking on dietary intake, nutrient status and personality of adult men and found that majority of the smokers were
predominantly affected (82%) by problems like inflation, prolonged illness and tension at home or at work spot than non-smokers (56%) stress (52%) and tension (58%) were the common problem found in smokers than in non-smokers.

Hebert, et al. (2002) revealed in the study that use of tobacco in smoking conferred a 5.19 times higher risk of oral pre-cancerous lesions of the palate than did use of chewing tobacco.

Parkin, et al. (2002) reported that cavity and pharynx cancers combined (hereafter referred to as oral cancer) were responsible for more than 400,000 new cases of cancer and 210,000 deaths worldwide. Oral cancer is primarily a disease that occurs in men and ranks as the seventh most common form of cancer worldwide when both sexes are combined.

Mackay and Eriksen (2002) reported that Cigarettes are the main type of tobacco product consumed in the world. About 5.5 trillion cigarettes were used annually in 1990 through 2000 about 1,000 cigarettes for every person on earth. More than 15 billion cigarettes are smoked per day.

Schantz and Yu (2002) reported that more than 90% of oral cancers occur in patients older than 45 years. The incidence increases steadily until age 65 years and substantial increase in the incidence of oral cancer (particularly of the tongue) among adults younger than 40 years.

Rodu and Cole (2002) identified the risk of SLT (smokeless tobacco) use for cancers of the oral cavity and adjacent sites.

Weinberg and Estefan (2002) reported that Smokeless tobacco has a stronger effect than a smoking because of the direct contact of the tobacco carcinogens with the oral epithelium as the chewing tobacco products are chewed or kept in the mouth.

Balaram et al. (2002) reported that chewing and poor oral hygiene explained 95 per cent of oral cancer of the women and among men, 35 per cent of oral cancer was attributed to a combination of smoking and alcohol and 49 per cent to tobacco chewing. Among men 35% of oral cancer is attributable to the combination of smoking and alcohol drinking and 49% to pan-tobacco chewing. Among women, chewing and poor oral hygiene explained 95% of oral cancer.
Znaor et al. (2003) concluded that oral, pharyngeal and esophageal cancers are 3 of the 5 most common cancer sites in Indian men and observed a significant dose-response relationship for duration and amount of consumption of the 3 habits (smoking, chewing and alcohol drinking) with the development of the 3 neoplasms (oral, pharyngeal and esophageal cancers). Tobacco chewing emerged as the strongest risk factor for oral cancer, with the highest odds ratio (OR) for chewing products containing tobacco of 5.05 [95% confidence interval (CI) 4.26-5.97]. The strongest risk factor for pharyngeal and esophageal cancers was tobacco smoking, with ORs of 4.00 (95% CI 3.07-5.22) and 2.83 (95% CI 2.18-3.66) in current smokers, respectively. An independent increase in risk was observed for each habit in the absence of the other 2. For example, the OR of oral cancers for alcohol drinking in never smokers and never chewers was 2.56 (95% CI 1.42-4.64) and that of esophageal cancers was 3.41 (95% CI 1.46-7.99).

Gajalakshmi et al. (2003) concluded that a quarter of the cigarette or beedi smokers in India would be killed by tobacco at the ages of 25-69 yr losing 20 yr of life expectancy.

Fabricius and Lange (2003) reported that a diet rich in fruit and vegetables reduces the incidence of lung cancer by approximately 25%. The reduction is of the same magnitude in current smokers, ex-smokers and never smokers. Supplementation with vitamins A, C and E and beta-carotene offers no protection against the development of lung cancer.

Critchley, et al. (2003) reported that tobacco is also consumed, especially in India and South East Asian countries, through chewing (for example, paan masala, gutka and mishri. Chewing tobacco is a risk factor for oral cancers.

Goldstein, et al. (2003) reported that by 2020 tobacco consumption has been projected to account for 13% of all deaths in India.

Rajkumar, et al. (2003) estimated the influence of paan, body mass index, diet, infections and sexual practices on oral cancer. BMI was inversely associated, and paan chewers with low BMI had a very high risk of developing oral cancer. Frequent consumption of fish, eggs, a variety of raw and cooked vegetables and fruits was associated with a decreased risk of oral cancer.
Lee et al. (2003) reported that Betel quid chewing was strongly associated with oral diseases like oral leukoplakia (OL) and oral submucous fibrosis (OSF). The attributable fraction of OL being 73.2% and of OSF 85.4%. While the heterogeneity in risk for areca nut chewing across the two diseases was not apparent, betel quid chewing patients with OSF experienced a higher risk at each exposure level of chewing duration, quantity and cumulative measure than those who had OL. Cigarette smoking had a significant contribution to the risk of OL. For the two oral premalignant diseases combined, 86.5% was attributable to chewing and smoking. Results suggested that betel quid chewing was a major cause for both OL and OSF.

Sinha et al. (2003) concluded that diets low in vegetables and fruits and high in alcohol increase the risk of oral cancers.

Znaor et al. (2003) reported that the smokeless tobacco to be the higher risk factor than smoking tobacco (OR 5.05). In the smokeless tobacco consumption, the risk was strongly determined by gutkha followed by tobacco flakes consumption. It is probably due to the combined effect of the ingredients (tobacco, areca nut, catechu, cardamom, lime and number of fine natural perfuming materials) present in gutkha.

Nair et al. (2004) conducted a case control study in Mumbai and identified that betel leaf chewing as major risk factor for oral cancer in India.

International Agency for Research on Cancer (2004) reported that Lung cancer is the dominant malignancy caused by smoking. The total number of cases is about 1.2 million annually, with 90% attributed to smoking. The strongest determinant of lung cancer in smokers is duration of smoking and risk also increases with the number of cigarettes smoked. Secondhand smoke causes lung cancer in nonsmokers, although the risk is far less than that of a smoker. In spouses of smokers, the excess risk is about 20% in women and 30% in men. Workplace exposures to secondhand smoke increase lung cancer risk in nonsmokers by 12% to 19%.

International Agency for Research on Cancer (2004) reported that cigar and/or pipe smoking cause cancers of the oral cavity, oropharynx, hypopharynx, larynx, and esophagus, with the risk being similar to that of cigarette smoking. Cigar and/or pipe smoking are causally associated with lung cancer and possibly with cancers of the pancreas, stomach, and urinary bladder.
Varghese (2004) reported that 80-90% of all cancers are due to environmental factors of which, lifestyle related factors are the most important and preventable. The major risk factors for cancer are tobacco, alcohol consumption, infections, dietary habits and behavioral factors. Tobacco consumption either by way of chewing or smoking accounts for 50% of all cancers in men. Dietary practices, reproductive and sexual practices account for 20-30% of cancers. Appropriate changes in lifestyle will reduce the mortality and morbidity caused to cancer.

Davey Smith, et al. (2004) reported that all forms of tobacco carry serious health consequences, most importantly oral and pharyngeal cancers and other malignancies of the upper aerodigestive tract.

Nichter and Sickle (2004) studied that among the college students those having a tobacco using household member were three times more likely to use tobacco compared to those who did not have any tobacco user in the household. Friendship was the most common reason cited for smoking.

Altieria et al. (2004) reported that alcohol use is a risk factor associated with oral and pharyngeal cancers. Consumption of all types of alcoholic beverages increases a person’s risk for oral and pharyngeal cancer. Those who consume 3-4 drinks a day are at a 2.1 times higher risk for oral and pharyngeal cancer, 5-7 drinks 5 times higher, 8-11 drinks 12.2 times higher, and 12 or more drinks a day 21.1 times higher risk. There is a significant trend across the levels of drinking (p<0.0001) among those who consume beer or spirits, and no wine, the increase in risk is markedly lower, with the highest risk being for those who consume three or more beers a day, with a 2.3 times higher risk. Wine has the most significant single effect on oral pharyngeal cancer risk, with those who consume 3-4 drinks a day are at a 2.2 times higher risk, 5-7 drinks 7.1 times higher, 8-11 drinks 11.8 times higher, and 12 or more drinks a day 16.8 times higher risk. There is again a significant trend across the levels of drinking (p<0.0001).

Pradeepkumar et al. (2005) reported that Boys having friends who were current tobacco users were 2.9 times more likely to use tobacco compared to those whose friends were not using tobacco.

Sachdev (2005) reported that smoking cause cancer or “Voice Box” in throat and the voice has to be going on.
Rosenquist et al. (2005) studied a possible relationship between oral cancer, oral hygiene, dental status, oral mucosal lesions and some lifestyle factors. Average and poor oral hygiene and inadequate dental status are independent risk factors for oral and oropharyngeal squamous cell carcinoma (OOSCC) irrespective of tobacco and alcohol consumption.

Godtfredsen et al. (2005) revealed in the study that reducing the risk of lung cancer through smoking reduction, rather than smoking cessation. The authors emphasize the fact that while quitting smoking should always be encouraged as the best possible choice for smokers, reducing the number of cigarettes smoked does appear to lower lung cancer risk.

Schabath et al. (2005) reported the effect of diet rich phytoestrogens has on lung cancer risk reduction. Phytoestrogens are plant-based chemicals that are similar in nature to the female hormone estrogen. They have numerous health benefits, and act as antioxidants and tumor suppressors. These plant chemicals are found in grains and a variety of fruits and vegetables.

Pradeepkumar (2005) reported that 46 per cent people knew that smoking causes lung cancer while 32 per cent reported that smoking causes or may exacerbate asthma. Only 10 per cent was aware of the relationship between tobacco use and cardiovascular diseases.

Regional Cancer Centre Trivandrum (2005) reported that the age adjusted incidence rate of cancers of oral cavity increased from 11.8 per 100,000 males in the year 2000 to 14.1 in 2002 in urban areas. Figures for lung cancer were 7.6 and 8.1 respectively.

Nelson et al. (2006) reported that the rate of smokeless tobacco use was 9.0% in rural (non MSA) area, while it was 3.3% in urban (MSA) area. Among women, the prevalence of smokeless tobacco use was only 0.3%, compared with a prevalence of 4.5% in males. Males ages 18-44 were most likely to use smokeless tobacco, over 5% of the population.

Maria et al. (2006) concluded that each portion of fruit consumed per day significantly reduced the risk of oral cancer by 49% (OR: 0.51; 95% CI: 0.40, 0.65). For vegetable consumption, the meta-analysis showed a significant reduction in the
overall risk of oral cancer of 50% (OR: 0.50; 95% CI: 0.38, 0.65). The multivariate meta-regression showed that the lower risk of oral cancer associated with fruit consumption was significantly influenced by the type of fruit consumed and by the time interval of dietary recall.

Subapriya et al. (2006) found in the study that an increased risk of oral cancer for bidi smokers compared to never smokers (OR (95%CI) 4.1 (2.4-6.9), whereas no significant pattern of risk was found for cigarette smokers. It may be due to the higher content of nicotine in bidi. The nicotine concentration in bidi is 21.2 mg g\(^{-1}\) compared to commercial filtered cigarette (16.3 mg g\(^{-1}\)) and unfiltered cigarette (13.5 mg g\(^{-1}\)).

American Cancer Society (2006) suggested in the study the nutritional guidelines for cancer patients including foods that help prevent cancer. Most all fruits and vegetables and important sources of vitamins and other chemo-preventative elements, and almost all are naturally low in fat and high in fiber. Fruits and vegetables are also rich in a newly explored class of compounds known as photochemical thought to have important, healthful properties.

American Association for Cancer Research (2007) reported that Being physically active not only helps prevent lung cancer in the first place, but it appears to improve survival and quality of life for those already diagnosed.

Thomas et al. (2007) concluded the information on smoking and betel quid chewing. Current smoking was associated with an increased risk of oral cancer with an adjusted odds ratio (OR) for daily smokers of 2.63 (95% confidence intervals (95% CI) 1.32, 5.22) and amongst heaviest smokers of 4.63 (95% CI 2.07, 10.36) compared to never-smokers. Betel chewing was associated with increased risk of oral cancer with an adjusted OR for current chewers of 2.03 (95% CI 1.01, 4.09) and in the heaviest chewers of 2.47 (95% CI 1.13, 5.40) compared to non-chewers. The OR in those who both smoked tobacco and chewed betel quid was 4.85 (95% CI 1.10, 22.25), relative to those who neither smoked nor chewed. In studies that reported results for non-smokers the combined OR was 2.14 (95% CI 1.06, 4.32) in betel quid chewers and in studies that adjusted for smoking the combined OR was 3.50 (95% CI 2.16, 5.65) in betel quid chewers. Smoking is a risk but it appears to be the relatively weak risk factor for oral cancer. Daily smokers have about 3 fold increased risk compared to never-smokers.
U.S. Cancer Statistics Working Group (2007) reported that the age-adjusted death rate from oral-pharyngeal cancers was 4.0 per 100,000 (5,312 cases) for males and 1.5 per 100,000 (2,514 deaths) for females.

Hashibe et al. (2007) reported that among never drinkers, cigarette smoking was associated with an increased risk of head and neck cancer (OR for ever versus never smoking = 2.13, 95% CI = 1.52 to 2.98), and there were clear dose-response relationships for the frequency, duration, and number of pack-years of cigarette smoking. Among never users of tobacco, alcohol consumption was associated with an increased risk of head and neck cancer only when alcohol was consumed at high frequency (OR for three or more drinks per day versus never drinking = 2.04, 95% CI = 1.29 to 3.21). The association with high-frequency alcohol intake was limited to cancers of the oropharynx/hypopharynx and larynx.

Hashibe et al. (2007) concluded that in industrialized areas such as Europe and the United States it has been well-established that tobacco use and alcohol consumption account for roughly 75% of all cases of oral and pharyngeal cancers. Tobacco and alcohol use commonly occur together, which makes it difficult to attribute risk to either alcohol or tobacco alone. Among smokers who have never used alcohol, the risk of oral and pharyngeal cancer is 2.13 times higher than persons who have never used alcohol or smoked.

Camphausen et al. (2008) reported that Alcohol and tobacco use are the most common risk factors for head and neck cancer in the United States. Alcohol and tobacco are likely synergistic in causing cancer of the head and neck.

Ridge et al. (2008) reported that there were 22,900 cases of oral cavity cancer, 12,250 cases of laryngeal cancer, and 12,410 cases of pharyngeal cancer in the United States.

Ries et al. (2008) reported that the overall incidence rate for oral and pharyngeal cancers of males and females of all races and ethnicities is 10.1 per 100,000.

Muwonge et al. (2008) reported that tobacco chewing was the strongest risk factor associated with oral cancer. The adjusted odds ratios (ORs) for chewers were 3.1 (95% confidence interval (CI)=2.1-4.6) for men and 11.0 (95%CI=5.8-20.7) for women. Effects of chewing pan with or without tobacco on oral cancer risk were elevated for both sexes. Bidi smoking increased the risk of oral cancer in men.
Dose-response relations were observed for the frequency and duration of chewing and alcohol drinking, as well as in duration of bidi smoking.

Thomas et al. (2008) studied that current betel chewing was associated with increased risk of leukoplakia with an adjusted odds ratio for current chewers of 3.8 (95% CI 1.7, 8.4) and in the heaviest chewers of 4.1 (95% CI 1.8, 9.1) compared to non-chewers. Current smoking was associated with an increased risk of leukoplakia with an adjusted odds ratio for current smokers of 6.4 (95% CI 4.1, 9.9) and amongst heaviest smokers of 9.8 (95% CI 5.9, 16.4) compared to non-smokers.

The American Institute for Cancer Research (AICR) 2008 reported that 30 to 40% of cancers could be prevented based on a healthy diet and moderate exercise alone.

Meyer et al. (2008) investigated in the study the association between periodontal disease, tooth loss and several diseases including cancer, cardiovascular disease, and preterm birth. Periodontal disease is a chronic inflammatory condition highly prevalent in adult populations around the world, and may be preventable. Estimates of prevalence vary between races and geographic regions, with a marked increase in the occurrence of periodontal disease with advancing age. The relationship between oral health and cancer has been examined for a number of specific cancer sites. Studies reported associations between periodontal disease or tooth loss and risk of oral, upper gastrointestinal, lung, and pancreatic cancer in different populations. There is associations persisted after adjustment for major risk factors, including cigarette smoking and socioeconomic status.

National Cancer Institute (2008) reported that Oral and pharyngeal cancers include cancers of the lip, tongue, salivary gland, floor, gum, and other parts of the mouth, nasopharynx, tonsil, oropharynx, hypopharynx, and other sites in the oral cavity and pharynx.

Muwonge et al. (2008) found in the study that approximately 94% of patients with oral cancer have used tobacco products and the relative risk of developing oral cancer was 11 times that of the risk in never use tobacco. All forms of tobacco use (active and passive smoking as well as smokeless tobacco) were associated with oral cancer. However, a maximum risk was found among the smokeless tobacco users OR = 7.8 (5.4-11.4).
Jacob et al. (2009) analyzed in the study that Among nonsmokers and nondrinkers, chewing betel quid without tobacco conferred ORs of 22.2 (95%CI = 11.3, 43.7) for oral leukoplakia, 56.2 (95%CI = 21.8, 144.8) for oral submucous fibrosis, 29.0 (95%CI = 5.63, 149.5) for erythroplakia and 28.3 (95%CI = 6.88, 116.7) for multiple oral precancers, after adjustment for age, sex, education and BMI. Dose-response relationships were observed for both the frequency and duration of betel quid chewing without tobacco on the risk of oral precancers. chewing betel quid without tobacco elevates the risks of various oral precancers.

Eldridge (2009) reported that physical activity is linked with a lower risk of developing lung cancer and the benefit extends to everyone men and women, as well as those who were smokers, former smokers, or had never touched a cigarette, all benefited from exercise. For both sexes exercise reduced the risk of death from lung cancer, although the benefits seem greater in women.

Fayed (2009) suggested four standard methods of treatment for cancer: surgery, chemotherapy, radiation therapy, and immunotherapy / biologic therapy. When initially diagnosed with cancer, a cancer specialist will provide the patient with cancer treatment options based on the type of cancer, how far it has spread and other important factors like age and general health. Side effects of chemotherapy like hair loss and nausea are usually the first things and other problems in the mouth, such as mouth sores, infections, dry mouth, bleeding of the gums and lining of the mouth, general soreness and pain of the mouth.

Fayad (2009) reported that oral cancer is a type of head and neck cancer that affects the mouth. It can form in the lining of the cheeks, gums, roof of the mouth, tongue, and lips.

Pednekar et al. (2009) revealed in the study that bidi is prepared by rolling tobacco in dried leaf of tendu (Diospyros malanoxylon) or Temburi tree (Diospyros ebenum) in comparison to US cigarettes, the mainstream smoke of bidi contains a much higher concentration of several toxic agents such as hydrogen cyanide, carbon monoxide, ammonia, other volatile phenols and carcinogenic hydrocarbons such as benz(a)anthracen and benzoypyrene. Moreover in India, bidi smoking being affordable to mass of population is most common than cigarette smoking. This aspect may explain bidi being a factor for increased risk of oral cancer in India.
Valeria et al. (2010) reported that animal products pattern was positively associated with oral cancer (OR = 1.56, 95% CI: 1.13–2.15 for the highest vs. the lowest score quintile), whereas the Starch-rich pattern (OR = 0.71, 95% CI: 0.50–0.99), the Vitamins and fiber pattern (OR = 0.47, 95% CI: 0.34–0.65) and the Unsaturated fats pattern (OR = 0.63, 95% CI: 0.45–0.86) were inversely associated with it.

Eldridge (2010) analyzed that Radon is the second leading cause of lung cancer after smoking, and the number one cause of lung cancer in non-smokers. Up to 15% of lung cancers worldwide are due to radon exposure.

Edefonti et al. (2010) reported five major dietary patterns named Animal products, Starch-rich, Vitamins and fiber, unsaturated fats and Retinol and niacin. The Animal products pattern was positively associated with oral cancer (OR = 1.56, 95% CI: 1.13–2.15 for the highest vs. the lowest score quintile), whereas the Starch-rich pattern (OR = 0.71, 95% CI: 0.50–0.99), the Vitamins and fiber pattern (OR = 0.47, 95% CI: 0.34–0.65) and the Unsaturated fats pattern (OR = 0.63, 95% CI: 0.45–0.86) were inversely associated with it. These findings confirm that diets rich in animal origin and animal fats are positively, and those rich in fruit and vegetables, and vegetable fats inversely related to oral and pharyngeal cancer risk.

Singh (2010) revealed in the study that in Manipur, the incidence of lung cancer, oesophageal cancer, hypopharyngeal cancer and oral cancer is very high both in males as well as females. This is owing to the habit of heavy tobacco use in the form of smoking, hookah etc. by our elders (A pastime in the past) and smoke filled housing environments. Paan eating habit with or without tobacco is a relatively newly acquired habit in Manipuri females in the last one decade or so. Predictions are that there will be higher incidence of oropharyngeal cancers in Manipuri women in coming 15-20 years (Carcinogenesis roughly takes 20-25 years from start of exposure).

Singh (2010) concluded that Cancer prevention is achievable by avoiding all the harmful carcinogenic agents like tobacco, strong chemicals, preserved, charred and infected foods, excessive ultraviolet rays etc. and by consuming wholesome food mainly based on green leafy vegetables and fruits and by following an active lifestyle. Next to prevention, early detection (Detection in stages of I and II) is the second
best approach. If detected early, with proper treatment, a cure of almost 50% of the patients can be achieved, a contradiction to the common belief that cancer is incurable. Early detection can be achieved by having regular health check-up, at least once yearly after 40 years age and screening programmes to susceptible subgroups of the populace also pays a long way in the early detection of cancer.

Madani et al. (2010) concluded in the case control study that consumption of tobacco-in any form (chewing, active and passive smoking) was significantly different between cases and controls (p<0.001). Similarly, overall drinking alcohol habit and non-vegetarian diet were also significantly different between the two groups (p<0.000 for both). Alcoholic beverages, country liquor, hard liquor and beer were significantly more common among cases (p<0.05 for all). The difference was observed in the dietary habits of the two groups. In case of both active and passive smoking, the prevalence was significantly higher in cases compared to controls (p<0.001 for both). In the active smoking sub categories viz., filtered cigarette, non-filtered cigarette and bidi (hand-rolled locally available cigarette), the prevalence was significantly larger in cases compared to controls (p<0.001 for all). The bidi smokers had the higher risk of oral cancer. The prevalence of almost all types of smokeless tobacco was significantly higher in cases (p<0.001 for all). Of the types, chewing of gutkha was more common among the cases and had the higher risk of oral cancer. The next significant risk factor was the chewing of tobacco flakes. The use of paan-parag and betel leaf was not significant risk factor for oral cancer. Overall tobacco use was highly significant risk factor for oral cancer with OR of 11.2, (95% CI of OR, 7.2-18.0), followed by non-vegetarian and alcohol consumption, 4.0 (2.8-5.8) and 2.7 (1.8-3.9) respectively. Whereas, the vegetarian dietary habit was found to be a protective factor for oral cancer 0.2 (0.1-0.3).

Madani et al. (2010) reported in the multivariate analysis that multivariate Odds Ratios (OR) for smoking and smokeless tobacco use as an exposure through different multivariate models for adjustment of possible confounders like age, gender, tobacco types, alcohol, non-vegetarian habit, location of residency, education and occupation as appropriate (adjusted OR).It is apparent that the multivariate and unadjusted risk assessment is different for various types of tobacco use. The unadjusted risk for filtered, cigarette smoking was not significant (OR = 1.4; 0.9-2.3), but the multivariate risk ranged from 2.7-3.4 after adjusting for other appropriate risk factors.
The unadjusted risk for non-filtered cigarette smoking was 2.5 (1.0-6.7), the multivariate risk ranged from 3.3-4.3 after adjusting for other appropriate risk factors through models 1-6. The multivariate risk for bidi smoking ranged from 3.7-5.5.

American Cancer Society (2010) reported that 36,540 new cases of oral cancer are expected to be diagnosed in the United States in 2010 and an estimated 7,880 people will die of the disease. This form of cancer accounts for about 3% of cancers in men and 2% of cancers in women.

Ries et al. (2010) concluded that Oral cancer occurs more frequently in blacks than in whites.