JAMC, Vol. 2, No. 1, 1989

REVIEW ARTICLE

HEALTH EFFECTS OF TOBACOO

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Tobacco in various forms has been used for centuries. American Indians were apparently the first to use tobacco in various forms: they smoked, chewed and sniffed.

Tobacco plant is a native of tropical America. The original ancestor is not known because tobacco is a very old plant and has been under cultivation for centuries. The plant spread all over North America before the arrival of whiteman. When Columbus first landed in 1492, some of the natives brought him a gift of dried leaves.[1] Throughout the West Indies, Columbus found that the tobacco trade between Indian tribes had been prevalent for hundred of years. It was found that the Indians either inhale the smoke of the leaves when set alight in pipes, cigarettes, cigars or chewed the leaf itself. Smoking and other forms of tobacco use had become a part of everyday life, and tobacco had real economic significance. In addition it was used in the form of poultices and pastes for treating burns, sores, cancers, sciatica, diseases of the liver, spleen and womb, chills, convulsions, worms, colic, warts, corns and bites by rabid dogs. This habit was adopted by white sailors and through them reached Europe from where it spread to Africa, Australia and Asia.

The practice of smoking was not very common in Europe until 1586 but from there on-ward its popularity increased inspite of opposition by Clergymen and rulers.

Linnaeus in 1753, named the genus of Tobacco Plant as "Nicotiana tobaccum" after the French statesman Nicot. In 1828, the most important known ingredient of tobacco was isolated and called Nicotine.[2] The eighteenth century was the century of smokeless tobacco (snuff and chewing tobacco). Its decline started about in 1850, when the sale of cigars and pipes began to take the lead and during the late 1800s the chewing tobacco decreased greatly, when chewing and spitting were excoriated by leaders in medicine and science such as Koch, Pasteur and Lister who associated the habit with germs and transmission of communicable diseases.[3]

Since the early 1970s, there has been a great resurgence in the use of all forms of smokeless tobacco in the United States. The sales of smokeless tobacco has increased about 11% each year since 1974 with an estimated 22 million users in the United States.[4]

In Pakistan, the annual production of tobacco amounts to 70-80 million kg of which 85% is consumed within the country in various forms

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(smoking, chewing, nasswar and hooka). Over 80% of the country's smokers are males aged 15 and above whereas smoking among females in relatively uncommon.[5]

Health Effects

Evidence from the biomedical literature indicates that the use of tobacco is associated with a variety of serious effects on different organs and systems of the human body including oral cavity, oesophagus, gastrointestinal tract, pancrease, respiratory and cardiovascular systems, genitourinary tract, nervous system and haemodynamics. The evidence of the effect is increasing day by day.

Oral Cavity

Cancer of the tongue and use of tobacco in some form were associated as early as 1921. The North Carolina study showed that women who used snuff for more than 50 years or longer were 48 times more likely to have cancer of the gum and buccal mucosa than were non-users. The authors attributed 87% cancers to the use of snuff.[6] Jayant and his co-workers found that persons who chewed tobacco were six times more likely to develop cancer than non-users.[7]

Experiments, to see the effect of smokeless tobacco on oral mucosa in rabbits exposed for twenty-six weeks, have shown to produce hyperkeratosis, ulceration and inflammatory changes in the gums and other parts of the buccal mucosa.[8] Application of snuff for 9 to 22 months to surgically created test canal in the lip induced one squamous cell carcinoma in 42 rats as compared to none in the controls. Short term treatment of mouse labial mucosa with not snuff and herpes simplex Type-I virus produced more epithelial dysplasia than either treated alone. Chewing tobacco extract has also been found to enhance the growth of two types of streptococci which are responsible for the development of caries.[9] Some investigations have suggested that the use of smokeless tobacco may be associated with a lower incidence of dental caries due to increased salivation and fluoride content of the product.[10]

The increase in risk of oral cancer in Central Asia, Republic of Soviet Union has also been attributed to the use of nass (snuff). In a study it was found that frequency of the oral lesions such as atrophy and hypertrophy of the mucosal membrane and leukoplakia increased with the length of use of nass reaching 20 per cent among those who use it for more than 15 years.[11] The frequency was greatest in people with simultaneous addiction to nass and cigarette smoking and the pathological changes (including cancer) usually occured in sites which come in contact wit nass.[11] Wynder and his associates also suggest development of oral cancer due to prolonged use of tobacco.[12]

Oesophagus

The effects of tobacco use on oesophagus are mentioned in the literature. They include oesophagitis, hyperplasia of the mucosa, keratosis, inflammation, and cancer. Reed considers tobacco smoking responsible for lowering the lower oesophageal pressure which result in oesophageal reflux and thus oesophagitis.[13] Shah and Nagi, in an experimental study, have found that hyperkeratosis, mucosal hyperplasia, ulceration and inflammatory changes occur due to the use of smokeless tobacco.[8] Cacinoma of oesophagus is produced by both forms: smoking tobacco as well as use of smokeless tobacco in the form of nass (snuff).[2]

Lungs

Several studies suggest that the use of tobacco specially cigarette smoking is important in the production of brochogenic carcinoma. Adler,[14] Tylecote,[15] Hoffman,[16] Licckint,[17] Ochner[18] Hirayama[19] and Trichonlos[20] are a few examples of the large number of workers who suggested that cigarette smoking is associated with increased frequency or bronchogenic carcinoma. Wynder et al have also found that tobacco usage is associated with cancer of the lungs and larynx.[21]

Several studies have demonstrated a strong dose response relation between the risk of mortality from chronic obstructive lung disease and the number of cigarettes smoked per day, the earliness of smoking initiation and the depth of smoke inhalation.[22]

Cardiovascular System

Voluntary and involuntary smoking is a major risk factor associated with cardiovascular diseases. This has documented in the past two decades by several U.S. surgeon general's reports and some 30,000 articles.[23]

Cigarette smoking has been strongly linked to coronary heart disease and peripheral atherosclerosis and appears to contribute to atherothrombotic cerebrovascular disease.[24]

Smoking in the presence of other risk factors for coronary heart disease like obesity, diabetes mellitus and hypertension has a synergistic effect on mortality from the disease. Smoking cessation results in a decreased risk of mortality from coronary heart disease and the degree of risk reduction is determined by the length of time after cessation, the amount smoked and the duration of smoking before cessation.

Based on data from a case control study carried out by Rosenberg et al it was concluded that the risk of myocardial infarction increased with the number of cigarettes smoked per day, both in the presence and absence of factors that predispose to an infarction.[25]

Blood levels of Nicotine achieved by cigarette smoking which are similar to those achieved by smokeless tobacco use, cause elevation of blood

pressure, heart rate, certain blood lipid levels and catecholamine levels. However, no epidemiological data are available on cardiovascular mortality in association with tobacco use.[26]

Cerebro Vascular Diseases

Several studies from various countries have shown 1.2 to 1.5 times increase mortality from smokers than non-smokers.[27] Studies also suggest improvement in cerebral perfusion after abstinence from cigarette smoking.

Gastrointestinal Diseases

Tobacco smoking is considered to be cause of duodenal ulcer by many authors. The incidence of peptic ulcer in smokers has been found to be as much as 5 fold greater than in non-smokers.[28]

Several cohort studies have reported the mortality ratio from peptic ulcer disease and duodenal ulcer among smokers of more than half pack per day to be approximately 2.5 times that of non-smokers.[29] Wormsley suggested that such studies cannot yet be interpretted in terms of the pathogenesis of duodenal ulcer and must not, therefore, be taken as providing a basis for treating patients with duodenal ulcers. However, he agreed that smoking should be discouraged in patients with duodenal ulcer.[30]

Epidemiological studies have provided inconstant data on association of smoking and cancer of the stomach.[31] Ulcerative colitis is less frequent in smokers than non-smokers. A two centre study in United States was carried out with a case control comparison of the effects of tobacco on risk of ulcerative colitis among 304,000 members of a health insurance organisation.[2] They assessed smoking history before onset of equal number of age and sex matched controls. The relative risk of ulcerative colitis was reduced to (0.6) in current smokers as compared with non-smokers with no relation to smoke.

Conclusion to Comments

Nicotine, and probably other constituents of tobacco, are clearly toxic chemicals, and threat to the public health. Strong efforts should be made for the prevention of the use of this toxic substance.

Massive educational efforts are needed to reach this goal. We must focus specially on the young, for whom early and rapid addiction to nicotine is a threat to life.

Individual physicians and the medical community at large should play a central role in this field. Physicians should serve a non-smoking examplors to their patients and the community, and work to educate their patients and the public of the serious health hazards involved.

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CORRIGENDUM

JAMC Vol. 1, No. 3, 1988, "Temperature rise in rise in patients with Acute Myocardial infarction," by Sharif-uz-Zaman and Shakeel Ahmed, on page 22, the following lines were inadvertently not printed.

... tachartythmias. This fact was studied by Cecillie and Co-workers by giving beta blockers to reduce the infarct size. They used the drug in the first few hours of infarct. It works by reducing the temperature in these patients, (other Mechanisms may be involved too) but basically it is by reducing the temperature with beta blockers in these patients.