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SPECIAL ARTICLE

TOBACCO AND HEALTH: WHAT CAN THE MEDICAL PROFESSION DO?

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Introduction

Tobacco consumption, that leads to three million deaths every year of which about one million occur in developing countries is a social evil(1). Tobacco smoke is a complex mixture of as many as 4700 individual constituents including carcinogens, irritants, ciliotoxic substances and poisonous gases. There is no dearth of information about the health hazards to smokers. it has been implicated as a major risk factor in a variety of chronic diseases including cardiac, cerebrovascular, malignant, respiratory and other diseases (2), Most of the smoking related lung diseases (e.g. chronic obstructive lung disease and lung carcinoma) are dose dependent. it is often the cumulative dose of smoking which determines the risk. Parameters like pack-year (PY) have been used to express exposure to tobacco smoke. One PY implies one packet of cigarettes (or 20 gms. tobacco) smoked each day over a course of one year (3). In India the number of cigarettes and bidis per packet is quite variable. A standard packet of cigarette and bidis per packet is quite variable. A standard packet of cigarette contains 10 numbers and a packet or bundle of bidi contains 15-24 bidis. The weight of a bidi varies widely in different brands. More importantly, the Indian smokers often describe their smoking habit by the total number of cigarettes/bidis smoked per day rather than the number of packets (4). A smoking index (SI) to quantify smoking exposure is therefore developed(5-7). Smoking index is defined as the product of average number of cigarettes (or bidis) smoked per day and the total duration of smoking in years.

SI = Number of cigarettes/bidis smoked per day x total duration in years.

Both the degree (number per day) and the duration of smoking are given equal weightage. For example, a subjectsmoking 10 cigarettes (or bidis) per day for one year, and another smoking one cigarette (or bidi) per day for ten years will have SI of 10. Both these individuals are considered to be at equal risk.

Tobacco is also consumed by various other methods and these include hookahs (water pipe), Chutta (cheroot smoked with the lighted end in the mouth), chilums (clay pipes) and snuff, as well as chewing. As a result, tobacco related cancers affect the mouth, pharynx, larynx and oesophagus in addition to lung (8,9) Different brands of cigarette and bidi smoke show little difference in total particulate matter (TPM) and mean nicotine levels (8). Defining cumulative exposure in terms of TPM rather than by tobacco consumption will be fallacious since the chemical constituents may differ widely with the method of analysis from different laboratories and brand of cigarette/bidi. Smoking habit can be classified as mild (SI<100), moderate (SI 101-300) and heavy (SI>300) using the smoking index (3). It had been observed that the prevalence of lung cancerand chronic obstructive lung disease is more in heavy smokers compared to mild smokers (6,10).

Pulmonary physiological abnormalities such as bronchial hyperreactivity and abnormal spirometric measurements are also severe in subjects with high smoking index (11).

Experimental, clinical and epidemiological studies have clearly established a relationship between cigarette smoking and a wide range of diseases (12). Not only is smoking directly responsible for the cancer of the lung and other site, but is also responsible for increased morbidity and mortality from other non-neoplastic respiratory diseases such as chronic bronchitis emphysema syndrome, cor pulmonale and respiratory infections. Tobacco smoke contains a large number of tumourigenic agents (carcinogens) that include benzo(a) pyrine, aza-arenes, N-nitrosamines, aromatic amines, aldehydes and inorganic compounds. As a result, tobacco is classified as a carcinogen (13). Various studies had clearly demonstrated that nicotine is an addictive drug and nicotine controls smoker's behaviour in such a way that reducing or suppressing tobacco consumption leads to a withdrawal syndrome characterised by irritability, difficulty in concentrating. cognitive impairments and weight gain (14). U.S. Surgeon General's Report on Smoking and Health has therefore, rightly concluded that cigarette smoking is the largest avoidable cause of death and disability in United States (15). Nearly a third of all cancers that occur in India are attributable to tobacco use (16). In India, until recently cancer of the oral cavity was the most common

lesion because of widespread tobacco chewing habit. However, there is a gradual change over from tobacco chewing to tobacco smoking and this may increase the incidence of lung cancer in India (17). A national survey conducted by the National Sample Survey organisation in 1987/88 had revealed that average tobacco usage prevalence was 35% for men and 12% for women (18). The mortality rate due to all causes among tobacco users is 1.4 to 1.6 times higher than that in non-users and it had been estimated that approximately approximate 80,000 death could be attributed to tobacco usage (19). It had also been computed recently that a minimum of Rs.10,000/- was required for the management of each tobacco related cancer case. It does not include the cost of services provided by the Government through various health agencies and also does not include the cost of non-productivity owing to the absence of the individuals from the worksite. Thus, the management of the total number of tobacco related cancer cases will cost the exchequer a sum several times higher than the total revenue generated by tobacco (17). Annual incidence of tobacco related cancer cases (17) in India is given in the Table.

TABLE

Annual incidence of tobacco related cancer cases in India. Estimated burden in 1992.

	Male	Female	Total
Estimated populaiion (millions)	450	412	862
Crude rate/100,000	70	80	
Total new cancer cases (all forms) in 1992	315,000	329,000	644,600
New cases related to tobacco habit:-			
Mouth	36,000	27,000	63,000
Pharynx & larynx	49,000	10,600	59,800
Oesophagus	23,700	16,500	40,200
Lung	27,800	5,100	32,900
Others	15,000	7,200	22,200
Total	1,51,700	66,400	218,100
Proportion of tobacco related cancers	48%	20%$_0$	34%

(Source: Reference 17)

Effect of smoking on Pulmonary Anatomy and Physiology

Structural Changes: There is increasedmucus formation due to hypertrophy and hyperplasia of mucous glands of the large airways. This results in increased sputum production. It is mainly the tar content of cigarettes which affects the phlegm (20). In the small airways, there is inflammation, goblet cell hyperplasia and intraluminal mucous formation which result in airways closure. This may be the forerunner of chronic obstructive lung disease. Experimental and radioisotopic studies have demonstrated that tobacco smoke damages the tight junction of airway epithelium and facilitates epithelial permeability (21,22) and this abnormal epithelial permeability can be reversed after cessation of smoking (21). The enhanced absorption of inhalant toxins and other aerosols resulting from increased permeabiiity leads to exaggerated bronchial hyperresponsiveness to inhaled methacholine in both symptomatic and asymptomatic smokers (23,24).

Functional Changes: The earliest functional changes that may occur due to tobacco smoking result from small airways disease. Peripheral airways are the site of increased resistance in obstructive lung disease and peripheral airways obstruction is associated with a chronic inflammatory process in the wall and lumen of the airways(25). Various tests that include frequency dependenceof compliance, residual volume, oxygen saturation on exercise, closing volume; and tests based on the forced expiratory manoeuvre are available for screening of small airways obstruction (26). However, tests based on forced expiratory manoeuvre such as mean flow rates at 25-75% vital capacity ($FMF_{25-75\%}$), instantaneous flow rates at 50% ($V_E$max 50%) and 75% ($V_E$max 75%), flow rates with and without helium breathing, volume of isoflow and mean transit time appear to be the best of these tests Even though $FMF_{25-75\%}$ is a simple test, it has high coefficient of variation which is true for $V_E$max 50% and $V_E$max 75%. Small airway dysfunction can occur even in asymptomatic smokers. In advanced cases, expiratory flow rates are reduced and the forced expiratory volume in one second (FEV_1) can be used as a predictor of development of obstructive airway disease. A relatively bw FEV_1 compared to the predictedvalue by middle age and a faster rate of its fall with age indicate that he/she may be **a** strong candidate to develop severe functional impairment. The fall in FEV_1 can be significantly arrested by discontinuation of smoking. Even if smoking is stopped at the age of 40 years, the rate of FEV, fall in ex-smokers becomes similar to that in non-smokers (27).

Chronic Bronchitis and Emphysema: It is now well establishedthat chronic bronchitis and emphysema are smoking related diseases (28-31). As the pattern of smoking in women approaches that in men (in the West) the incidence of the disease in the females rises and may equal the male incidence in the near future.

Tobacco smoking is the most important risk factor associated with chronic bronchitis and emphysema(28). Tobacco smoke attracts alveolar macrophages (AM) and they cluster around the terminal bronchioles. Alveolar macrophages activated bytobacco smoke release neutrophil chemotactic factors such as leukotriene B4 (LTB4), complement components (C5a) and neutrophil chemotactic factor. Thus circulating neutrophils from the,

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blood stream are attracted to the site where AMs have already clustered around the terminal bronchioles. Tobacco smoke causesactivation of neutrophils resulting in release of human neutrophil elastase (HNE). HNE is capable of lysing elastin, collagen, proteoglycan, fibronectin and laminin which form the interstitium of lung. In a healthy individual, HNE is inactivated by alpha-1 antitrypsin which is produced in the liver. The molecular weight of alpha-1 antitrypsin is 52000 kilodalton (KD) and this molecular size enables alpha-1 antitrypsin to migrate to epithelial lining fluid of lung from blood. The binding of alpha-1 antitrypsin with HNE results in inactivation of HNE.

An active methionine residue present in alpha-1 antitrypsin molecule is essential for neutralization of HNE. Oxidants present in smoke oxidizes methionine, thus preventing the binding and inactivatan of HNE. In addition oxidants released by activated neutrophiis are also capable of oxidizing alpha 1 - antitrypsin. Thus tobacco smoke, in. addition to increasing the HNE burden in the lower respiratory tract, causes inactivation of protein alpha-1 antitrypsin which is essential for body's defense against elastases. This results in unopposed action of HNE on lung tissue leading to destructive changes (32).

It had been observed in a study of natural history of chronic bronchitis and emphysema that airflow obstruction developed in 10% of ex-smokers, 12% moderate smokers (average upto 15 cigarettes per day) and 26% heavy smokers (average over 15 cigarettes per day) (33). Life long non-smokers do not develop airflow obstruction unless other factors contribute to airflow obstruction. This study suggested that two contrasting groupscould beidentified, viz., the "susceptibles" who in their lifetime might become disabled by obstructive disease if they smoke and the : "non-susceptibles" who would not, whether they smoke or not (33). Currently, no test is available to differentiate the susceptible minority" of smokers from the "non-susceptibles". Research should be directed to identify the "susceptible smokers" who develop airflow obstruction due to smoking.

The prevalence of chronic bronchitis is high among Indian smokers (34-39). In Bombay, cough, dyspnoea or both was observed among 39% of smokers and 13% of non-smokers (39), and studies in North India had revealed a high prevalence of chronic bronchitis and obstructive airway diseases (38,40,41) among smokers. In urban Chandigarh and rural Mullapur in North India, chronic bronchitis was diagnosed in 9.9% and 16.4% of male smokers and in only 0.9% and 4.1% of non-smokers (40).

Smoking and Airway obstruction: Regular cigarette smoking increases phlegm production and airway obstruction and in susceptible smokers airway obstruction is often irreversible (20). Even the response to treatment for asthma is poorer among smokers than non-smokers(42). A steep fall in the forced expiratory volume was shown among smokers with bronchial hyperreactivity (43).

Exposure to Environmental Tobacco Smoke

Studies in Western countries have shown that exposure to environmental tobacco smoke (ETS), or passive smoking, also known as secondary or involuntary smoking, is associated with impaired lung function, increased rates of respiratory illness and chronic airway diseases (44-46). It is estimated in 1986 apart from 35000-40000 annual deaths due to ischemic heart disease (47), ETS has been associated with variety of lung diseases including lung cancer in 3000 patients (48). In 1990, Environmental Protection Agency published a similar report (49). In asthmatic children, exposure to ETS leads to increased severity of the disease (50). In US, the National Research Council estimated an increased risk from lung cancer of 34% for non-smoking spousesof smokerscompared with non-smoking spouses of non-smokers (48). It had also been observed from Japan that standardized mortality ratios for lung cancer in non-smoking Japanese women were increased in, direct proportion to the amount smoked by their husbands (51). These studies suggest that smoking is a public health hazard with the potential to injure innocent non-smokers exposed to environmental tobacco smokers.

ETS have adverse effects on the treatment of asthmatic patients (52). The worsening of airway obstruction is due to the reflex stimulation of parasympathetic pathways by the smoke particles, Asthmatics, charactarized by increased airway responsiveness to various stimuli react abnormally to particulate matters (53). Even in nonasthmatic patients, cigarette and bidi smokiig are known to increase bronchial responsiveness (54-55). Exposure to ETS may also increase airway responsiveness (56).

In adults, respiratory symptoms, chronic bronchitis and asthma attacks are more common in nonsmokers exposed passively to tobacco smoke as compared to those who are not exposed (57). There is an increased bronchial reactivity to inhaled bronchoconstrictor aerosols in wives of smokers presumably due to increased epithelial permeability. This would imply increased absorption of most tobacco combustion byproducts inhaled by these subjects in the company of smokers. Peak expiratory flow rate was significantly lower in passive women smokers and the reduction was found to be in

proportion to the amount of smoking (58). Environmental tobacco smoke along with other air pollutants may be responsible for the high prevalenceof chronic respiratory disease and cor-pulmonale in non-smoking subjects especially women in ourcountry (59).

Parental smoking exacerbates respiratory diseases in children. The occurrence of cough and cold and the risk of hospitalization for respiratory illness had been shown to be higher among children when one or both parents were smokers (60,61). Maternal smoking has been shown to be a leading cause of paediatric deaths resulting from low birth weight, short gestation, respiratory distress syndrome and sudden infant death syndrome (48). There is twice the risk of pneumonia and bronchitis amongst children of smokers during first year of life (43). Heavy smoking during pregnancy may have a direct effect on the offspring's subsequent lung function and a relationship was suggested between early childhood bronchitis or pneumonia and impairment of lung function in later childhood with maternal smoking habit (62). Furthermore, asthmatic children of mothers who smoked had lower expiratory flow rates and more emergency room visits than those with non-smoking mothers (49,63). These observations emphasize the adverse effects of exposure to ETS in children.

Effect on Exercise Tolerance

Smoking reduces the exercise tolerance even in asymptomatic individuals. Even a single cigarette or bidi smoking has been shown to increase airway resistance (64). Exercise tolerance is further reduced in bronchitic patients with continued smoking due to high camoxyhemoglobin level (65,66).

Other Effects

lncidence of respiratory infections especially pneumonia and influenza is enhanced in smokers (67). Post operative respiratory complications are also more in smokers (68,69).

It had been established that there was an interaction between occupational carcinogens such as asbestos and radon and smoking (70). In comparison with non-smokers in general population, the risk of lung cancer deaths in asbestos workers who smoked was increased by 90 - fold (71). A multiplicative interaction between smoking and radon also been seen in uranium workers (72). Since radon is a ubiquitous indoorair pollutant (73). smokers are at particularly high risk for developing cancers from indoor radon (72). Smoking has also synergistic effect with certain occupational exposures, like coal mine dust, grain or cotton dust, in producing chronic bronchitis and airways obstruction. Airways obstruction is found to be significantly more in smoking Workers in silica industry (74-76).Therefore, smoking is a powerful risk factor which increases the risk not only of lung cancer but also airway obstruction associated with occupational exposure.

Medical Profession's Role

Even though the ill effects of tobacco smoke to human race is scientifically well established, the eradication of this pandemic, tabacosis, from the world was not possible because of the commercial and political power of tobacco industry. In India one million marginal farmers and five million farm workers are involved in tobacco cultivation and processing. Tobacco cultivation does not require intensive irrigation and the leaf growing costs are the lowest in India. As a result tobacco cultivation is popular in Karnataka and Andhra Pradesh. In addition to depriving the farmers of their livelihood, the income generated to the coffers of Government by the tobacco industry stands in the way of our efforts to eradicate this preventable malady. India is the world's third largest grower of tobacco and the earning per hectare is from Rs. 15,000 to 30,000 compared to Rs. 6,000 from non-tobacco crop. Cigarettes contribute to 90% of tobacco revenue and no doubt, it is good for farmer, good for revenue through exports, but is bad for nation's health. The impact of health hazards of tobacco as discussed in newspapers seems to have hardly any long lasting effect. Do we know our right to breathe air that is free of tobacco smoke?

Medicine is not a Science but a learned profession charged with the obligation to apply them for the benefit of mankind. Medicine is defined as a human activity undertaken for the benefit of others whether in the area of public health, statistical comparison, or in the care of the individual (77). It is the foremost duty of every person engaged in the profession of health care delivery system to boldly expose the bare facts without consideration of money, fame or credit.

The statutory warning on a cigaretee pack appears to have a low impact. Although every cigarette package has the statutory warning printed, the same appears at the comer of huge hoarding (in barely visible letters) and the advertisements from the tobacco industry in the media are too tempting for everybody in general, and for teenagers in particular. Therefore, such warning remains inconsequential. A survey in USA involving chronic smokers had revealed that 70% were unable to identify the specific theme of even one of the four surgeon general's warnings on cigarette packages, despite adequate opportunity for exposure to such a warning (78). A one-pack-per day (10 cigarettes per day) smoker is theoretically exposed to the 'warning' 3650 times per year. These data originate from societies with much higher literacy rates and much easier accessibility to health information as compared to

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those found in our set-up. No wonder then that the real state of affairs in developing countries is expected to be still worse and more saddening, where such warnings are unlikely to have any impact on the health behaviour of those using these products, as consumers, continue to remain to a large extent not warned at all.

Instead of giving such warning, it is better to increase the tax on cigarettes. Increasing the price of cigarettes may decrease tobacco consumption (78). This would help in two ways: to decrease smoking habit and to generate revenue for potential use in smoking prevention and health promotion efforts such as health education, stop-smoking campaigns, research into tobacco related diseases and prevention of tabacosis. These efforts will certainly pay back benefits in the form of improved health and reduce medical care expenditure for the individual, the family, the society and ultimately the nation at large. In order to initiate a preventive programme, at least the hospitals should be made a smoke-free environment.

What is the most important health problem today? Is it human immunodeficiency virus (HIV) disease or tobacco-related disease? Even though tobacco abuse is the leading preventable cause of death of mankind, political leaders and scientific community have failed in their duties to provide adequate attention to this alarming problem compared to those devoted to HIV disease.

On a worldwide scale, tobacco consumption is increasing by 2.1% per annum. Tobacco consumption is increasing by about 3-4% per annum in developing countries while it is decreasing by 0.2% per annum in developed countries (79). Per capita cigarette consumption in developing countries has risen on an average by over 70% during the last 25 years and going by current trends WHO estimates that there will be about seven million deaths a year from tobacco in the developing, countries within the next two or three decades (79). In the developed countries, at least two million deaths a year (20% of all deaths) can be attributable to smoking during the same period (79). In the United States, there is a decline in smoking prevalence and was found to be linear across most socio-demographic groups. Smoking prevalence which was 50.2% in men and 31.9% in women in 1965 had declined to 31.7% in men and 26.8% in women in 1985. Smoking prevalence was thus found to decrease at a rate of 0.84% per year among males and 0.21% per yearamong females (4880). A World Health Organization expert panel has estimated that three million people die every year because of tobacco use (1). The panel predicts that if current smoking patterns continue, the toll will increase to 10 million deaths a year by 2025. This means that half a billion people now living - 10% of the world's population - could ultimately die of tobacco use (81,82). "Smoking represents the most extensively documented cause of disease ever investigated in the history of biomedical research" (83). The United States Office on Smoking and Health's bibliographical database contains more than 50000 citations from biomedical publications, summarized in more than 8000 pages in 21 reports from the surgeon general. The BMJ Publications Group has launched a new journal - "Tobacco Control" in March 1992 in time for the eighth world conference on tobacco and health in Buenos Aires(84). As the control of an infectious disease requires an understanding of the causative agent, the vector, the host, and the environment in which transmission occurs, the same principles apply to the control of tobacco abuse (85). Therefore, tobacco control involves much more than the effects of smoking on health. The journal will pay attention to the host, publishing epidemiological and behavioral research on tobacco use, and will analyze the environment in which tobacco is used. The impact of tobacco on the environment - for example on public health, economy, and political processes - will be considered, as well as the impact of the environment on tobacco. Programmes and policies that discourage tobacco consumption are an important part of that environment, and publishing evaluations of these interventions will be a priori for the new journal. Similarly, it will publish survey research on public beliefs, opinions, and attitudes related to tobacco (84)

Transnational tobacco companies - based predominatly in the United States and Britain - are aggressively marketing their products in the developing world (86-90) as their home markets shrink. A major goal of the journal will be to report on the activities of the developing world.

Will we be able to control this social evil? It is suggested that social evils like alcoholism, commercial sex and tobacco abuse can be controlled by community and religious participation.

Smoking is one of the important threats to the medical men since health risks from smoking are abundant. The prevention of smoking demands a political action because it is probably the largest single preventable cause of ill health in the world. A conflict of interest between health professionals and tobacco industry is evident and a coexistence is not possible between these two establishments. Why should then the people suffer unnecessarily? Who is to tell the politicians about the sufferings, tragedies, miseries and economic tosses caused through smoking? Who is to narrate the enormous magnitude of smoking related problems? Who will make them realize the gravity of the greatest epidemic of the present time arising out of abuse of tobacco? Who will reveal the medical facts that 5% of all deaths are caused by smoking? It is the duty of doctors

as well as the health profession to act immediately. The prevalence of smoking in doctors, teachers and medical students (91-98) is quite alarming, and is the same as that in the general population and they themselves need a cleansing operation.

Doctors must realize that their contribution does not end with health education and disease management. Their duty towards community is extended as opinion makers of the society and of providers of healthy atmosphere. A national coalition of concerned physicians, teachers, religious leaders and opinion makers is suggested (99) to bring about fundamental changes to curtail the tobacco cultivation and promotion and to prevent the epidemic of tobacco related cancers, obstructive respiratory diseases and cardiovascular disorders..

Many health professionals are afraid of getting involved in such a process considering that exerting political pressure is below dignity and is a distasteful act. But in such a situation, our battle will be lost by default. We must gather courage; and fortunately, we have with us some major international voluntary health organizations who are actively involved in exercising political pressure. That makes our case more strong but that gives us doctors a little jerk too, to realize our duty as doctors towards suffering humanity (2). With concerted efforts, the environment can be made tobacco-free; of course with the help of politicians and the Government. After all, politicians are also reasonable people, they are also open to arguments and pressure which are based purely on medical facts and meant solely for the community well being. They can be made to realize the danger to the community from smoke and the benefits of eradication of this social evil. The day is not far when they will stand up, admit their right to serve, realize their responsibility and perform graciously their national duty and. help the medical profession in creating a tobacco-free atmosphere for every citizen of the universe.

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