

Editorial Commentary

Smoking Tobacco- Different Perspectives

Tobacco smoking is the largest preventable cause of death [1-3]. Every year May 31, 2010 is celebrated as a United Nations Day- the World No Tobacco Day [4]. Therefore, it seemed imperative to pen down an editorial commentary on this important public health problem. However, "tobacco and health" is an exhaustive topic, which can not be easily covered even in a dedicated single issue. This editorial aims to spread awareness and stir intellectual debate; and will give an overview of Historical perspective, Burden of smoking, Cigarette manufacture, Cigarette smoke, Deposition and Absorption of smoke, Quantification of tobacco exposure, Effects of active and passive smoking, Smoking cessation and in the end a few success stories and alternative avenues.

Historically, nicotine molecule was produced over 60 million years probably to guard against insect behaviours. The antiquity of smoking dates to 692 AD of a Mayan priest smoking. Around the 16th century, tobacco cultivation was spread by the Spanish and Portuguese sailors. In the 17th century, pipe smoking and snuff were dominant but declined by the 19th century. The precursors of present day cigarettes were long tobacco filled reeds used by the Aztecs. Spaniards refined the technique using paper tubes. In 1870s, handmade cigarettes were replaced by mechanised ones and between the two world wars cigarette smoking gained popularity and presently, the **burden of smoking** is mammoth. There were believed to be 1.2 billion smokers in the world (in 2000) and assuming same prevalence 1.5 billion smokers exist today [5]. 50% of smokers will die of smoking if they do not quit- 4.9 million worldwide every year as in 2007 [6,7]. Quitting before middle age reduces chances of dying from tobacco [8]. Each year smoking causes 4 million deaths, burden is equally shared between developed and the developing world. India is home to one-fifth of the world tobacco users and 1/3 of all tobacco users in the developing world. Six lakhs die annually which is 15% of global tobacco deaths, still 55,000 Indian children get addicted every year and 40% men light regularly. Unfortunately, there has been little change in habits in India.

If we go into the nitty-gritty of how **cigarette is manufactured**- The tobacco plant (*Nicotiana tabacum*) is sprayed with 'casing' (sauce of sugars, humefactants and flowering agents) and then harvested. Following this, it is cured by air drying (burley) or cured by artificial heat (flue curing). The tobacco leaf is then processed in to sheets; mixed with additives to improve taste and chopped to make cigarettes. Tobacco is wrapped in a tube of paper to form a cigarette and a filter of cellulose acetate may be placed at the end (filter cigarettes). Filter cigarettes have less tar and nicotine, but may have same or higher yield of carbon monoxide. Each cigarette contains about 1 gm tobacco. Burley tobacco yields 20-40 mg tar per cigarette. A regular or a king size cigarette is respectively, 74 or 84 mm in length. "Bidi", peculiarly seen in India, has 0.5 gm dried and cured tobacco flakes, hand rolled in a rectangular piece of tendu leaf (*Diospyros melanoxylon*). The outer covering contributes to high draw resistance increasing carbon monoxide production. Compared to non-filter cigarettes, bidi contributes to high total dry particulate matter, including carcinogenic hydrocarbons and other toxic agents- carbon monoxide, ammonia, hydrogen cyanide and phenols. India contributes to 85% of world's bidi production.

Cigarette smoke has both gas and particulate phase components. Various gases include nitrogen, oxygen, carbon dioxide, carbon monoxide (4%), oxides of nitrogen, nitrosamines, hydrogen cyanide, nitriles, volatile hydrocarbons, aldehydes and acrolein. The particulate matter is an aerosol of tar and nicotine. Tar is a complex mixture of polynuclear aromatic hydrocarbons including carcinogens as non-volatile nitrosamines, aromatic amines and benzopyrene. Low tar cigarettes have < 18 mg tar, while > 26 mg is high tar. Radioactive constituents in cigarette smoke include polonium and lead, which are carcinogenic. On an average, each cigarette delivers 15 mg tar and 1 mg nicotine. In a WHO report, no difference was reported between filter and non-filter cigarettes; Indian cigarettes were reported to deliver higher tar (19-27 mg) and nicotine (1-1.4 mg) content; and bidi

contributed to highest tar (>23 mg) and nicotine (1.7-3 mg) levels.

Two terminologies may be relevant here- **Mainstream smoke**, which is produced at high temperature and drawn through the butt during puffing, is the predominant source of exposure to the smoker. It carries 2-5 billion particles/mL of diameter 0.2-1 mm i.e. in the respirable range. The **sidestream smoke**- is produced at a lower temperature during the smouldering of cigarette between puffs and is the principal source of environmental tobacco smoke. At lower combustion temperature, more distillation products (increased amount of nitrogen containing bases) and less combustion products are produced. However, the combustion zone of sidestream smoke is less oxygen deficient so a greater proportion of carbon dioxide is produced as compared to carbon monoxide.

It is also important to realise that the delivery of tar, nicotine and carbon monoxide are affected by the way a cigarette is smoked- residual butt length, butt filter nicotine and smoking behaviour (number of puffs, puff pressure profile, puff volume, depth and duration of inhalation). The tobacco rod (cigarette) is akin to a fractionation column, concentrating compounds towards the butt. The particulate matter is produced at much higher concentration towards finishing a cigarette, this is primarily the reason why it is advised to leave longer stubs.

Quantification of tobacco exposure is pertinent but difficult. A clinical quantification is by counting the pack years, wherein 20 gm tobacco (20 cigarettes) per day for 1 year equals one pack year. Measurement of nicotine and conitine (by radioimmunoassay or gas phase chromatography) can be performed. Urinary cotinine indicates smoking within last 36 hours. In non-smokers, no urinary nicotine is present and cotinine < 10 mcg%.

Exhaled carbon monoxide measurement > 8 ppm is strongly suggestive of smoking. Venous blood carboxyhaemoglobin levels can be assessed- mean level correlates with the number of cigarettes smoked while non-smokers have < 1.7%.

The **physiological effects** of smoking are mentioned in Table 1 while **adverse pharmacological effects** are outlined in Table 2 [9-11]. Smoking [12,13] is a strong adverse risk factor for the heart and blood vessels- coronary artery disease, sudden cardiac death, abdominal aneurysms, progression of hypertension to malignant phase, cerebrovascular disease, renal artery stenosis, ruptured berry aneurysms and peripheral vascular disease; apart from playing an aetiological role in chronic obstructive airway disease. Tobacco smoke harbours several organ-specific carcinogens targetting the oesophagus, lung, pancreas, kidney and bladder. Carcinoma of the oral cavity and larynx are also known to occur in those who chew tobacco. Tobacco also has adverse effects on pregnancy, increasing the risk for intra-uterine growth retardation, spontaneous abortions, foetal and neonatal death. Maternal smoking during pregnancy may also adversely affect infant's long-term growth, intellectual development and behavioural characteristics. Smoking also worsens peptic ulcer disease and nephropathy in diabetics. Interestingly cigarettes can function as vectors by becoming contaminated with workplace chemicals like polytetrafluoroethylene, formaldehyde, methylparathion, inorganic fluorides, inorganic mercury and lead. Cigarettes may facilitate their entry in to the body and also may cause chemical transformation. Cotton textile workers, coal miners and firefighters have greater degree of airway obstruction if they are smokers. Asbestos and coal gas workers have higher risk of cancer if they are smokers.

Table 1- Physiological effects of smoking (Nicotine exerts its action at cholinergic receptors)

Physiological	Typical acute effect
• EEG	Shift towards higher frequencies in relaxed individual
• Sensory receptors	Stimulated
• Cardiovascular	Tachycardia, peripheral vasoconstriction, small rise in BP
• Circulatory Hormones	Release of catecholamines, vasopressin, cortisol & growth hormone
• Respiratory	Minimal effects
• Skeletal muscle	Depressed reflexes & muscle tone, increases finger tremor
• Gut	Increased tone and motor activity of bowel
• Body weight	Decreases as BMR rises, less efficient food absorption & less appetite

Table 2- Adverse pharmacological effects of smoking

Cardiovascular effects

- ↑ Heart rate
- ↑ Blood Pressure
- ↓ Coronary blood flow
- ↓ Blood oxygen carrying capacity
- ↓ Vascular prostacyclin
- ↓ Contractility
- ↑ Peripheral vascular resistance

Haematological effects

- ↑ Leucocytosis
- ↑ Leucocyte activation
- ↑ Platelet activation
- ↑ Fibrinogen
- ↑ Haematocrit
- ↑ Carboxyhaemoglobin
- ↑ Blood viscosity

Metabolic effects

- ↑ Total cholesterol
- ↑ Triglycerides
- ↓ HDL-cholesterol

Endocrine effects

- ↑ Catecholamines
 - ↑ Anti-diuretic hormone (ADH)
 - ↑ Adrenocorticotropin hormone (ACTH)
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Passive smoking defined by exposure to tobacco combustion products from the smoking of others, also is harmful. Environmental tobacco smoke derives from the mainstream which is first filtered by cigarette and then lungs and the side-stream smoke, which contributes to 85% of the smoke in the room. Typical range of respirable particulates in smoking areas like café etc is 100-700 mcg/sq. m.; which is 25 times of non-smoking areas. Most frequent symptom experienced by non-smokers is eye irritation (69%) followed by headache, nasal irritation and cough. Passive smoking causes a small but significant reduction in pulmonary function, can precipitate an asthmatic attack, cause cough and dyspnoea in obstructive airway disease and worsen angina. Parental smoking can cause chronic cough, chronic phlegm, persistent wheeze and respiratory infections in children. Non-smokers living with

smoking spouses have increased risk for lung cancer.

Smoking cessation is not an easy job and requires a strong will power. Self care groups, smoking-cessation clinics, behavioural methods, physician advice and counselling, hypnosis, acupuncture, mass media and community programmes and medication (nicotine replacement therapy, bupropion, bupropion, etc.) all have their respective roles to play.

When tobacco is so bad and poses such an immense health problem, and despite bans in various cities on smoking, why have we consistently failed in curbing this menace. Instead of starting the blame-game, it will be prudent to mention the **success story** of Thailand where 25% middle-aged men have stopped smoking. In Thailand, the packet warnings are very clear, there is a ban on tobacco advertising, anti-smoking media campaigns are active and high-ranking politicians have put in positive efforts. Truly, Thailand is lucky as both the government and the non-governmental organisations are active in that country. Cultivation of large stretches of tobacco can not be converted overnight to some other cultivation and this is a major problem. In Phillipines, tobacco is being turned into food supplements, antibiotic ointments, skin creams, building materials, paints, pesticides and paper so that the tobacco farming sector is supplemented. It is important to realise that it is only the leaves of the tobacco plant which are used for smoking. The seeds, stalks and roots can be used for **alternative harmless purposes**- stem is woody and good for particle boards and dissolving pulps. Tobacco was used for medicine before it was used for smoking. Folk remedies formed the basis for medicinal applications- antibacterial, antifungal creams and topical analgesics. The minty smelling oil pressed from seeds can be turned in to soap and paints. Whole seeds which are free from nicotine and high in animal protein have the potential to be used as animal feed. India, US and UK are experimenting with proteins from tobacco leaves to be used as nutritional supplement.....so may be the future has in store instead of the poison a nutritional supplement from tobacco!

References

1. Chelland Campbell S, Moffatt RJ, Stamford BA. Smoking and smoking-cessation- the relationship between cardiovascular disease

- and lipoprotein metabolism: a review. *Atherosclerosis* 2008;201:225-35.
2. Raupach T, Schafer K, Konstantinides S, Andreas S. Secondhand smoke as an acute threat for the cardiovascular system: a change in paradigm. *Eur heart J* 2006; 27: 386-92.
 3. Taylor BV, Oudit GY, Kalman PG, Liu P. Clinical and pathophysiological effects of active and passive smoking on the cardiovascular system. *Can J Cardiol* 1998; 14: 1129-39.
 4. <http://www.who.int/tobacco/wntd/2010/en> Accessed on June 19, 2010.
 5. Tobacco smoking. From Wikipedia, the free encyclopedia. http://en.wikipedia.org/wiki/Tobacco_smoking. Last updated on 14th June, 2010. Accessed on June 19, 2010.
 6. Thomson CC, Rigotti NA. Hospital- and clinic-based smoking cessation interventions for smokers with cardiovascular disease. *Prog Cardiovasc Dis* 2003; 45: 459-79.
 7. Le Houezec J, Sawe U. Smoking reduction and temporary abstinence: new approaches for smoking cessation. *J Mal Vasc* 2003; 28: 293-300.
 8. Joseph AM, Fu SS. Smoking cessation for patients with cardiovascular disease: what is the best approach? *Am J Cardiovasc Drugs* 2003; 3: 339-49.
 9. Meinertz T, Heitzer T. Primary and secondary prevention of coronary heart disease: smoking. *Z Kardiol* 2002; 91 suppl. 2: 3-11.
 10. Nikodemowicz M. The effects of smoking on cardiovascular system. *Przegl Lek* 2007; 64 suppl. 4: 42-4.
 11. Rigotti NA, Pasternak RC. Cigarette smoking and coronary heart disease: risks and management. *Cardiol Clin* 1996; 14: 51-68.
 12. Tonstad S, Andrew Johnston J. Cardiovascular risks associated with smoking: a review for clinicians. *Eur J Cardiovasc Prev Rehabil* 2006; 13: 507-14.
 13. Bartal M. Health effects of tobacco use and exposure. *Monaldi Arch Chest Dis* 2001; 56: 545-54.

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