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Review

Cigarette smoking—Effect of metabolic health risk: A review

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ABSTRACT

Aims: Cigarette smoking is a serious health problem and most important avoidable causes of death in worldwide. The aim of this report is to briefly review describe cigarette smoke composition and carcinogenesis. A second part will discuss the association between tobacco use and high risk of health disorders, existing evidence regarding smoking and cardiovascular risk and other health disorders.

Methods and Methods: The evidence so far shows that smoking dose-dependently increases the risk of impaired glucose tolerance, the incidence of type 2 diabetes mellitus, pulmonary diseases, smoking and cancers and abdominal-type obesity.

Results: Tobacco products contain more than 50 established or identified carcinogens and these may increase risk of cancer by causing mutations that disrupt cell cycle regulation, or through their effect on the immune or endocrine systems. Certain factors such as genes, diet and environmental exposures may alter susceptibility to cancer in tobacco user.

Conclusions: Today at least 20% of all cancers are estimated to be attributable to smoking, but this figure is expected to increase because of the uptake of tobacco use in low-income countries.

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1. Introduction

Cigarette smoking is a serious health problem and most important avoidable causes of death in world [1]. Smoking has

been strongly implicated as a risk factor for chronic obstructive pulmonary disease, cancer and atherosclerosis, etc. [2–4]. The World Health Organization predicts that tobacco deaths in India may exceed 1.5 million annually by 2020 [4]. In recent years, large household surveys have shown that in middle age, more than one-third of men and a few percent of women smoke tobacco and that there are about 120 million smokers in India [5,4]. The leading causes of death from smoking are cardiovascular diseases (1.69 million deaths), chronic obstructive pulmonary disease (0.97 million deaths) and lung cancer (0.85 million deaths) [6]. The aims of this report are to briefly review the existing evidence for

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smoking as a main cardiovascular (CV) risk factor; smoking increases the risk for diabetes, and analyze more extensively the links between tobacco use and the risk of type 2 diabetes mellitus (T2DM), chronic obstructive pulmonary disease, tobacco and cancer, insulin resistance and abdominal type obesity, and to address any changes in weight after smoking cessation. This review describes global patterns of tobacco use, composition of cigarette smoke and involved in carcinogenesis. A second part will discuss the association between tobacco use and risk of specific health disorders. Tobacco use has traditionally been a practice of high-income countries, but it has cancer and health disorders in tobacco users. Today at least 15% of all major health problems are estimated to be attributable to smoking.

2. Composition of cigarette smoke

Most tobacco products are made from the species *Nicotiana tabacum* [7]. Cigarette smoke is a complex mixture of chemicals containing more than 4000 different constituents [8,9]. In the last 30–40 years, a large body of knowledge has accumulated identifying the exact chemical composition of cigarette smoke both qualitatively and quantitatively [10,11]. Some of the compounds identified include different pyridine alkaloids such as nicotine, ammonia, acrolein, phenols, acetaldehyde, N-nitrosamine; polycyclic aromatic hydrocarbons such as benzopyrene; combustion gases such as carbon monoxide, nitrogen oxides, hydrogen cyanide; trace metals, α -emitter radioactive elements such as polonium, radium, and thorium [11,12] (Table 1).

3. Free radicals and carcinogens of cigarette smoke

Two major phases were identified in cigarette smoke: a tar phase and a gas phase; both phases are rich in oxygen-centered, carbon-centered and nitrogen-centered free radicals as well as non-radical oxidants. From the analysis of each phase, it was estimated that a single cigarette puff contains approximately, 10^{14} free radicals in the tar phase, and 10^{15} radicals in the gas phase

[10]. These include various compounds, which are capable of causing an increase in the generation of various reactive oxygen species (ROS) like superoxide ($O_2^{\bullet-}$) hydrogen peroxide (H_2O_2), hydroxyl (OH^{\bullet}) and peroxy (ROO^{\bullet}) radicals. These reactive oxygen species in turn are capable of initiating and promoting oxidative damage in the form of lipid peroxidation [13].

A carcinogen is defined as an agent that causes a series of genetic alterations to occur, leading to the formation of cancerous growth. Tobacco smoke has long been recognized as a chemical carcinogen. Tobacco smoke contains some deadly carcinogenic chemicals. Some of these cancer-causing chemicals, such as the tobacco-specific nitrosamines, N-Nitrosomorpholine N'-Nitrosornicotine (NNN), 4-(Methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), N'-Nitrosoanatabine (NAT) and N'-Nitrosoanabasine (NAB), are formed from natural components of the tobacco plants [7,14,15].

4. Smoking and cardiovascular risk

Cigarette smoking is a major cause of atherosclerotic disease and is considered one of the major risk factors for coronary heart disease (CHD), along with hypertension and lipid disorders. This finding is most significant because CHD is the most common cause of death in the United States and in most of the industrialized countries [16,17]. Although atherosclerosis begins in childhood and progresses during adolescence and young adulthood [18,19], whether smoking was associated with pre-clinical atherosclerosis in persons under age 35 was not known. In 1985, investigators organized a multicenter cooperative study, Pathobiological Determinants of Atherosclerosis in Youth (PDAY), to determine the relation of CHD risk factors to atherosclerosis in young people. We previously reported a strong association of smoking with atherosclerosis in the abdominal aorta, and a weaker and less consistent association of smoking with atherosclerosis of the coronary arteries [20,21,19].

The two major mechanisms involved in coronary heart disease are atherosclerosis, which is a pathologic process that results in stenosis of the arteries, and thrombosis. Atherosclerosis involves endothelial injury, intimal smooth muscle cell proliferation, proliferation of macrophages with lipid accumulation, and formation of foam cells and development of plaques and plaque calcification and rupture. Thrombosis, which causes the acute occlusion of the arteries usually at the site of a ruptured atherosclerotic plaque is the final common precipitant of most acute coronary and other vascular events. Progression of the atherosclerotic lesions involves many metabolic and physiologic processes, most of which are augmented by cigarette smoking.

Smoking causes endothelial injury, which is considered the antecedent to atherosclerosis. It has been demonstrated that nicotine has a desquamating effect on the endothelium, probably by increased shear stress from increased blood viscosity and the rise in heart rate, cardiac output, blood pressure, and vasoconstriction induced by smoking. In addition to this endothelial damage by mechanical factors, chemical injury to the endothelium, is caused by polycyclic aromatic hydrocarbons in cigarette smoke. Tobacco smoke increases smooth muscle cell proliferation by inducing platelet adherence to the injured endothelium, with the resulting release of platelet-derived growth factor (PDGF). The major hemodynamic effects of smoking are produced by the release of catecholamines by the sympathetic nervous system, which is activated by nicotine. These effects include increased heart rate and blood pressure, cardiac output, and vasoconstriction along with the resulting increased myocardial oxygen demand. These changes contribute to endothelial injury. In addition, carbon monoxide from cigarette smoke reduces the oxygen-carrying capacity of the blood; smoking may also induce

Table 1
Selected cigarette smoke composition.

Subjects	Effects
<i>Particulate phase</i>	
"Tar" ^a	Carcinogen
Polynuclear aromatic hydrocarbons	Carcinogens
Nicotine	Neuroendocrine stimulant and depressant; addicting drug
Phenol	Co-carcinogen and irritant
Cresol	Co-carcinogen and irritant
β -Naphthylamine	Carcinogen
N-Nitrosornicotine	Carcinogen
Benzo[a]pyrene	Carcinogen
Trace metals (e.g., nickel, arsenic, polonium 210)	Carcinogen
Indole	Tumor accelerator
Carbazole	Tumor accelerator
Catechol	Co carcinogen
<i>Gas phase</i>	
Carbon monoxide	Impairs oxygen transport and utilization
Hydrocyanic acid	Ciliotoxin and irritant
Acetaldehyde	Ciliotoxin and irritant
Acrolein	Ciliotoxin and irritant
Ammonia	Ciliotoxin and irritant
Formaldehyde	Ciliotoxin and irritant
Oxides of nitrogen	Ciliotoxin and irritant
Nitrosamines	Carcinogen
Hydrazine	Carcinogen
Vinyl chloride	Carcinogen

^a The aggregate of particulate matter in cigarette smoke after subtracting nicotine and moisture International Agency for Research on Cancer (IARC) [7].

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