Effects of Smoking on Cardiovascular System

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Abstract

Background: Smoking is a major risk factor for heart disease and death, and is recognized as one of the leading causes of death in the world. Cigarette smoke contains more than 4000 chemicals, including nicotine and carbon monoxide (CO), which can have harmful effects on heart function. These basic components of cigarette smoke cause oxidative stress, endothelial damage, and deactivation and are associated with very high serum concentrations of total cholesterol and triglycerides, as well as low levels of cardioprotective high-density lipoprotein. By causing intravascular inflammation, smoking promotes the development of atherosclerosis and heart disease. Exposure to secondhand smoke makes atherosclerosis disappear and increases the risk of MI, stroke, aortic aneurysm, and sudden death. Smoking can cause DM and accelerate the occurrence of minor DM and macrovascular complications. Smoking has causal relationships and frequent interactions with other major risk factors for CHD, including hyperlipidemia, hypertension. The effects of smoking are almost the same or almost the same as that of smoking. The purpose of this article is to provide a brief overview of the effects of smoking, and specifically the effects of nicotine and CO on cardiovascular function. Nicotine reduces independent cardiac function, increases sympathetic activity, and increases heart rate (HR).

Conclusion: By quitting smoking we have many options like behavioral changes and treatment instead of nicotine and bupropion.

Keywords*: Tobacco smoke; Nicotine; Inflammation; Free radicals*

Aim of the study

To understand the effects of smoking on cardiovascular system and some knowledge about associated disorders thus you can manage it to decrease the suffering and prevent catastrophic death for the patient.

Introduction

Smoking is a major risk factor for heart disease and death and is considered one of the most preventable deaths in the world. Globally, 25% of deaths from heart disease are due to smoking. The European Society of Cardiology recently reported that smoking causes 28% of heart deaths in men aged 35 to 69 and 13% in women of the same age. In the European Region of the World Health Organization (WHO), smoking is the second highest risk factor for life expectancy due to disability and the highest risk factor for premature death, accounting for 1.6 million deaths annually. happens. It is estimated that 15% of all deaths from tobacco use in the European Union are due to smoking each year, and in Greece, any smoking-related death in people aged 35 and over Also the number of deaths due to old age, accounting for 18.1% (1).

The WHO estimates that tobacco use kills about 6 million people each year, including more than 600,000 smokers from heart disease, lung cancer, and other diseases. If current trends continue, it is estimated that more than 8 million people will die annually by 2030. Smoking is one of the leading causes of heart disease, such as heart disease, ischemic stroke, peripheral arterial disease, and gastrointestinal aneurysms. It is associated with an increased risk of certain types of cancer and is a major cause of chronic obstructive pulmonary disease. Active or passive smoking can cause heart disease through a series of interdependent processes such as increased oxidative stress, hemodynamic changes, and autonomic flexibility. .., Endothelial dysfunction, thrombosis, inflammation, hyperlipidemia, or other side effects. Occasional smoking, less smoking, and even small exposures such as a few cigarettes a day are enough to cause serious side effects. Cigarette smoke contains more than 4,000 chemicals that have a serious effect on heart function (1).

Smoking contributes to other cardiovascular risk factors, such as sugar intolerance and low serum levels of high-density lipoprotein cholesterol (HDLc). However, studies have shown that smoking increases the risk of CVD in addition to the effects of smoking on other risk factors. In other words, if smoking doubles the risk level and at the same time another risk factor, adjustments are made to distinguish between smokers and non-smokers at the level of these other risk factors. The risk is estimated to increase fourfold. Smoking has been linked to peripheral arterial disease (PAD), aortic aneurysm, CHD, and cerebrovascular disease, but the associated risk of disease (RR) depends on the arterial bed (1).

The Pathophysiology of Cigarette Smoking and Cardiovascular Disease

Cigarette smoking (CS) remains a serious health risk, contributing significantly to cardiovascular morbidity and mortality. Cigarette smoking affects all stages of atherosclerosis, from endothelial dysfunction to acute clinical events, the latter of which is primarily thrombotic. Cigarette smoke exposure, both active and passive (environmental), increases the risk of cardiovascular events. Some recent experimental clinical trials have demonstrated a non-linear relationship to cigarette smoke exposure, so whether there is a clear direct dose-dependent link between cigarette smoke exposure and risk is questionable. The specific harmful components of cigarette smoke and the processes of CS-related cardiovascular dysfunction are unknown, however, CS enhances inflammation, thrombosis, and oxidation of LDL cholesterol. Recent experimental and clinical

studies back up the idea that cigarette smoke exposure increases oxidative stress, which could be a trigger for cardiovascular disease (2).

Cigarette smoking (CS) increases the risk of myocardial infarction (MI) and fatal coronary artery disease in both men and women, according to epidemiological studies (CAD). In comparison to nonsmokers, even low-tar cigarettes and smokeless tobacco have been found to increase the risk of cardiovascular events. Furthermore, passive smoking (ambient tobacco exposure) with a smoke exposure one-hundredth that of active CS is linked to a 30% increase in the incidence of CAD, compared to an 80% increase in active smokers. Thus, while there is strong evidence relating cigarette smoke exposure to cardiovascular disease, the particular components of cigarette smoke and the mechanisms underlying this link are still unknown. The current clinical and experimental observations on the probable pathobiology and processes involved in smoking-related cardiovascular disease are updated in this article (fig. 1) (3).

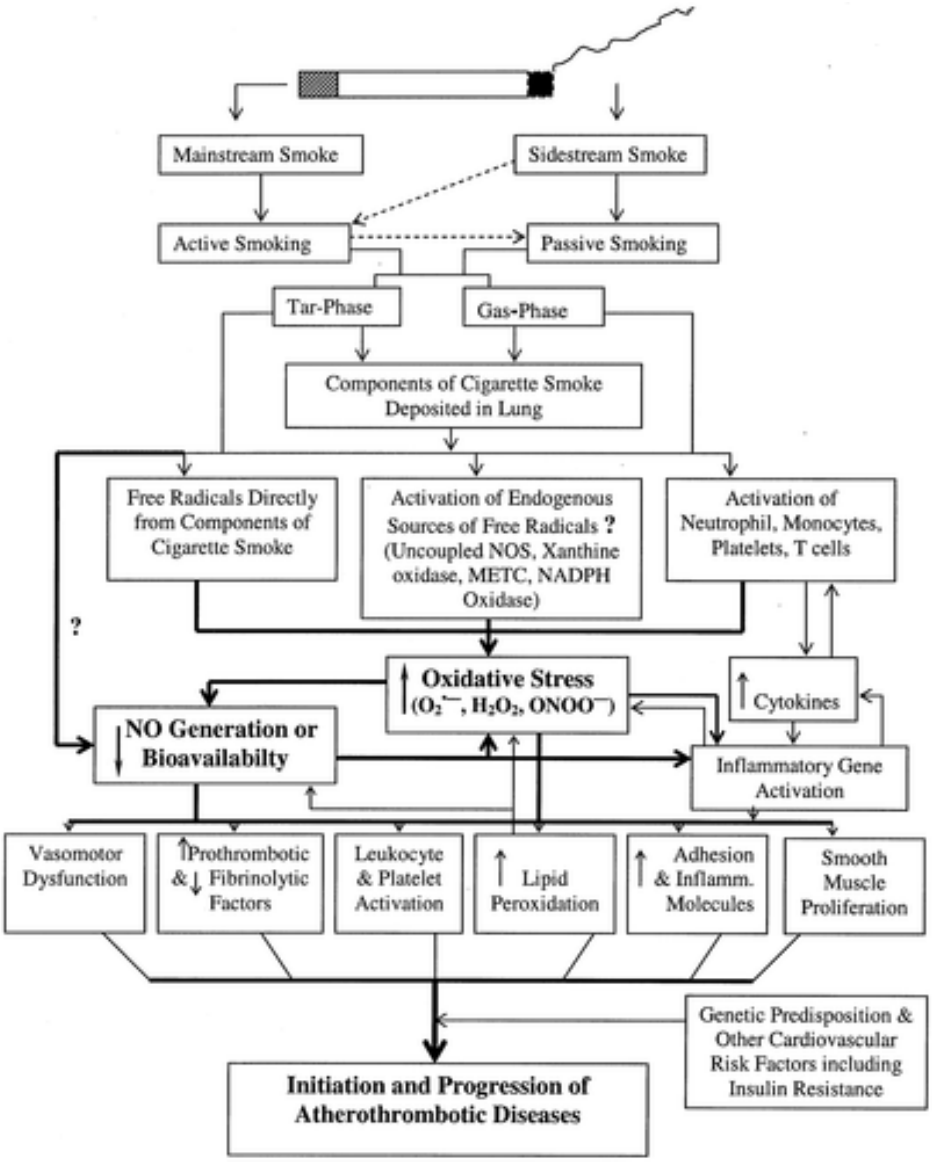

Figure 1 Cigarette smoking-induced cardiovascular dysfunction: potential paths and causes. The flow diagram's bold boxes and arrows reflect the most likely central mechanisms in the complicated pathophysiology of the cigarette-smoking-mediated atherothrombotic illness. METC = mitochondrial electron transport chain; NADPH = nicotinamide adenine dinucleotide phosphate reduced form; NOS = nitric oxide synthase; ONOO= peroxynitrite; O2= superoxide; H2O2= hydrogen peroxide (3)

Cardiovascular effects

1-Sudden Death

Most sudden deaths are due to CVD. In a detailed analysis combining data from the Framingham Heart Study and Albany Study, which examined sudden cardiac death in men aged 45-64 years, smoking was a statistically significant risk factor. In a data study of the 1986 National Mortality Follow-up Survey of People without a History of CHD, smoking was the only variable risk factor for sudden cardiac death, increasing the risk of sudden cardiac death in people with heart disease. It was a related factor. .. Known as CHD. In patients with left ventricular dysfunction after MI, there is an association between smoking cessation and reduced mortality from cardiac arrhythmias. Finally, smokers who survived out-of-hospital cardiac arrest had a lower risk of recurrent cardiac arrest in smokers than in nonsmokers (4).

2-Stroke

Smokers have a higher risk of stroke due to cerebral palsy, greater mortality, and a clear association with volume response than non-smokers. In addition, following a 20-year prospective mortality study controlled for by other risk factors associated with cardiovascular risk, smoking increases the risk of stroke and increases mortality as the number of smokers increases. In a meta-analysis of data from 32 studies, the data indicated an increased risk of stroke in former smokers younger than 75 years than non-smokers in the same age group. Together for all ages. During the group's 26-year follow-up study on the Framingham Heart Study, smoking was a major risk factor for stroke. However, the risk of smokers who quit smoking for two years was lower than that of smokers who did not smoke throughout their lives after quitting smoking for five years (5).

3-Aortic Aneurysm

Arteriovenous arterial occlusion disease is a different form of vascular degeneration than atherosclerotic and other arterial occlusive diseases. The formation of an aneurysm within the vascular tree has a regional distribution, and the pathological process varies depending on the localization. Abdominal infrarenal aortic aneurysm (AAA) is the most common manifestation of aneurysm disease, and smoking is the most dangerous factor. Smoking is a much greater risk for AAA than atherosclerosis. Smoking not only plays a role in the etiology of AAA but also increases the rate of development and the risk of fractures of established AAA. The mechanical

relationship between AAA and smoke is established through the use of advanced animals based on smoke or smoke components. The processes involve long-term changes in the vascular smooth muscle cell and inflammatory cell function. This review will examine the clinical, epidemiological, and technical evidence considering tobacco as a cause of aneurysms, with an emphasis on AAA (6).

Arteriovenous arterial occlusion disease is clinically, historically, and functionally distinct from arteriosclerosis and other arterial occlusive diseases. Although atherosclerotic changes can occur in most aneurysms, this is not the cause. Aneurysms are more common in some arteries than others, and their etiology varies by location, eg. Although little evidence suggests that smoking plays a role in some manifestations of aneurysm disease, the role of smoking in the development or progression of other aneurysms has not been considered (7).

4-Hypertension

Smoking is a major risk factor for cardiovascular disease, and smoking cessation is one of the most effective ways to prevent many cardiovascular diseases. Endothelial dysfunction, arteriosclerosis, inflammation, lipid conversion, antithrombotic and thrombosis-promoting changes are the major factors associated with smoking and the acceleration of atherosclerotic processes leading to cardiovascular events. Smoking has a hypertensive effect, especially by stimulating the sympathetic nervous system (Fig. 2) (8).

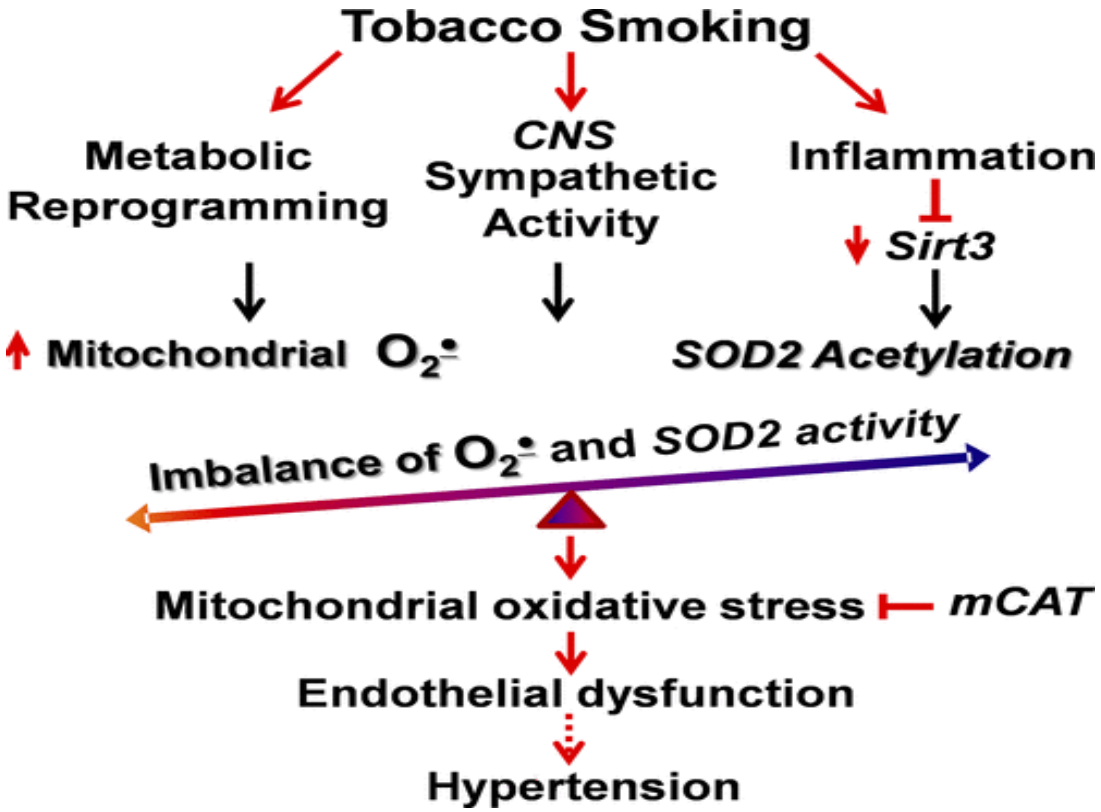

Figure 2 (Hypertension Pathogenesis) (8)

5-lipid metabolism

The effects of smoking include changes in lipid metabolism with increased lipolysis, insulin resistance, and tissue lipotoxicity. Smoking is prothrombotic and atherogenic (Fig. 3) (9).

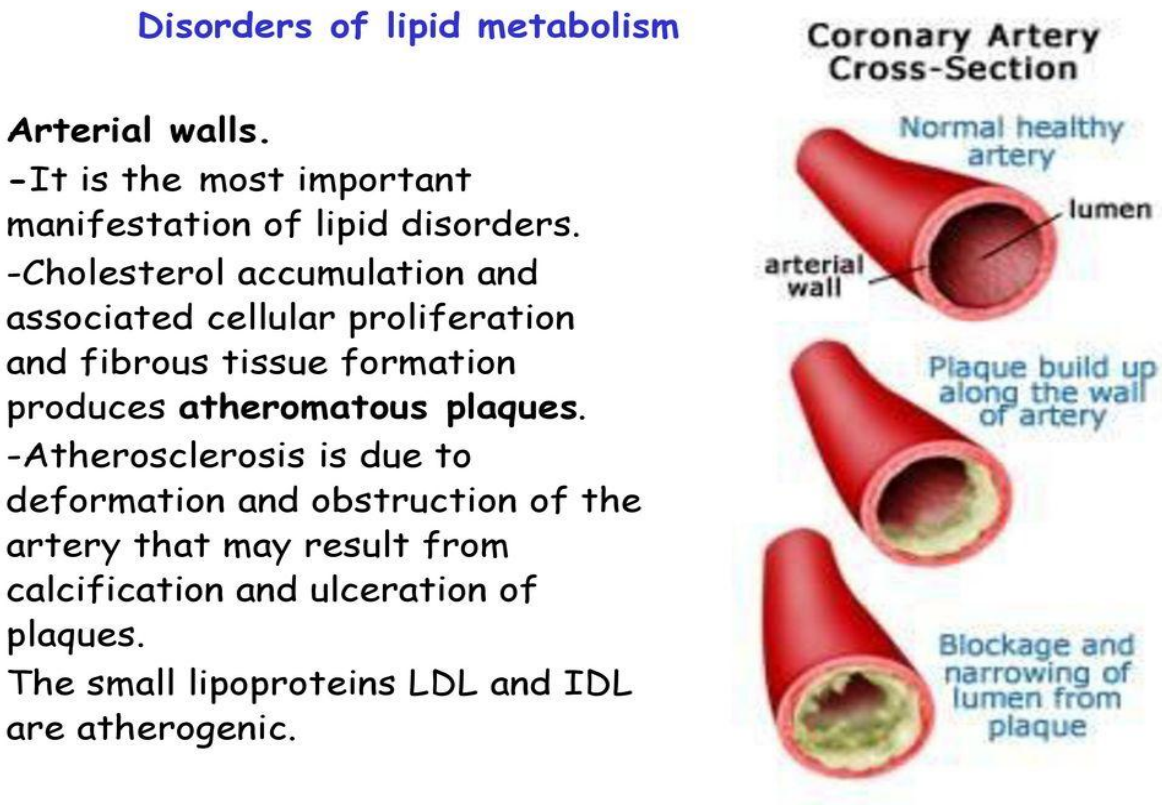

Figure 3 (Disruption of Lipid Metabolism in Smokers) (9)

6- Hemodynamic Effects

6.1-Blood Pressure and Heart Rate

Nicotine stimulates the release of epinephrine and norepinephrine from the adrenal medulla and endothelial nerve endings, resulting in increased heart rate and maximum attenuation by stimulation of myocardial β1 receptors. Peripheral vascular resistance increases with α-receptor-mediated vasoconstriction that raises blood pressure. It also promotes β2 and α2 receptors in the coronary arteries. Stimulation of β2 receptors promotes vasodilation, and stimulation of α2 receptors promotes vasoconstriction (10).

6.2-Coronary Blood Flow

Smoking alters the response of coronary blood flow to increased myocardial oxygen demand; that is, it decreases the flow of the coronary vasodilator blood flow reserve. Therefore, the increase in coronary blood flow based on the level of myocardial function is less than would be expected in the absence of secondhand smoke exposure. There is ample evidence that tobacco use causes coronary artery endothelial dysfunction. Smoking can be associated with narrowing of the coronary vessels. Although smoking increases blood flow to a person without coronary heart disease, it can decrease cardiovascular blood flow in heart disease (10).

7-Thrombogenic Effects

Smoke-induced thrombosis appears to be an important factor in the development of serious cardiovascular events. Epidemiological evidence suggests that smoking increases the risk of dangerous myocardial infarction and sudden death in addition to the risk of angina. Researchers believe that the risk of major myocardial infarction and sudden death is related to thrombosis and that angina pectoris is mainly due to hemodynamic factors. Successful rehabilitation in MI patients after thrombol therapy is more likely in smokers than nonsmokers (10).

8-Arteriosclerosis

The formation of platelets lining the artery wall where there is dynamic blood flow or endothelial damage may be the prodromal stage of atheromatous plaque formation. Nicotine is thought to increase blood viscosity and platelet aggregation as it inhibits the production of prostacyclin which can reduce platelet aggregation. Platelet adhesion increases the production of thrombi, separates the nerves of the coronary arteries, accelerates the process of atheromatous plaque formation, and is associated with an increased risk of cardiac ischemia. In addition, nicotine affects the metabolism of prostaglandins, impairing the defense of vessels against platelet aggregation (Fig. 4) (11).

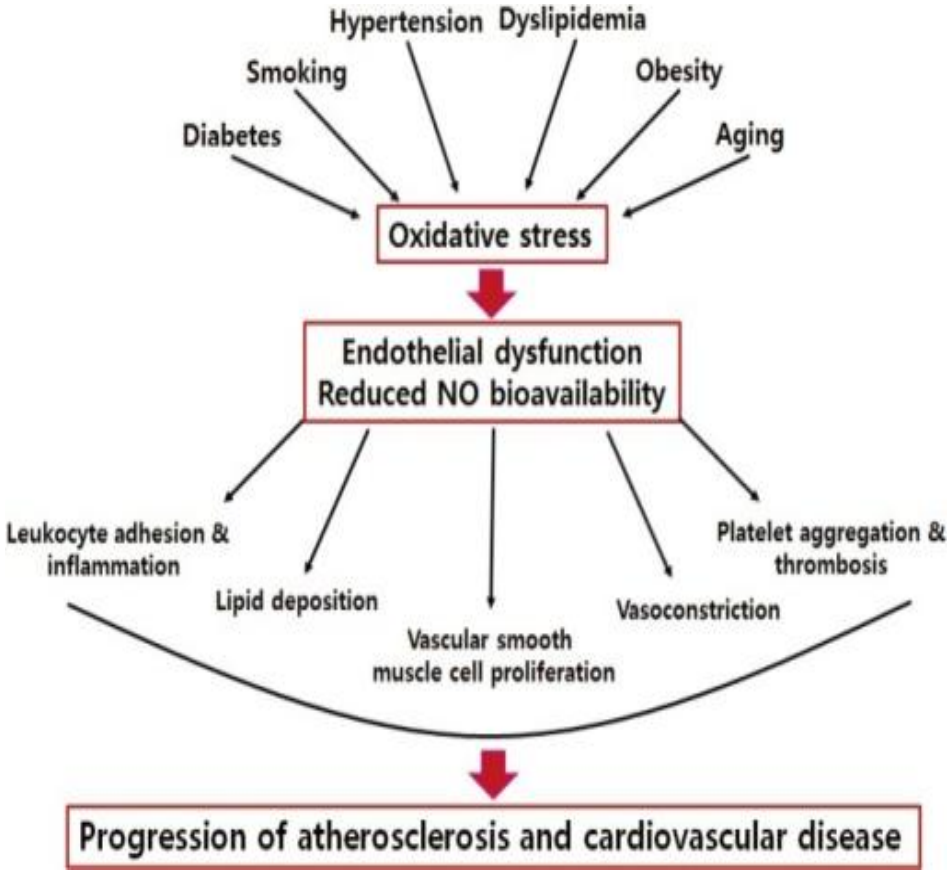

Figure 4 (Atherosclerosis in Smokers) (11)

Atherosclerosis is a degenerative process that affects the coronary arteries, cerebral arteries, iliac and femoral arteries, and the aorta, resulting in coronary heart disease (CHD), stroke, and peripheral arterial disease (PAD). It starts in the intima of the big elastic and muscular arteries during childhood, with lipid deposits in macrophages and smooth muscle cells, primarily cholesterol and its esters. During early childhood, the lesions, known as fatty streaks, generate only minor intimal thickening and no blood flow problems, but they rapidly become more severe during puberty. A core of lipid and necrotic debris is covered by a cap of smooth muscle and fibrous tissue in young adults, and a core of lipid and necrotic debris is covered by a cap of smooth muscle and fibrous tissue. These modifications result in raised lesions known as fibrous plaques, which protrude into the lumen and disrupt blood flow (11).

9-Endothelial Affection :

In response to exposure to smoke, endothelial cells are known to release inflammatory and proatherogenic cytokines. All of these processes lead to endothelial dysfunction. The direct physiological effects of smoke compounds and the production of active forms of oxygen (ROS) lead to endothelial cell loss due to apoptosis or necrosis (Fig. 5) (12).

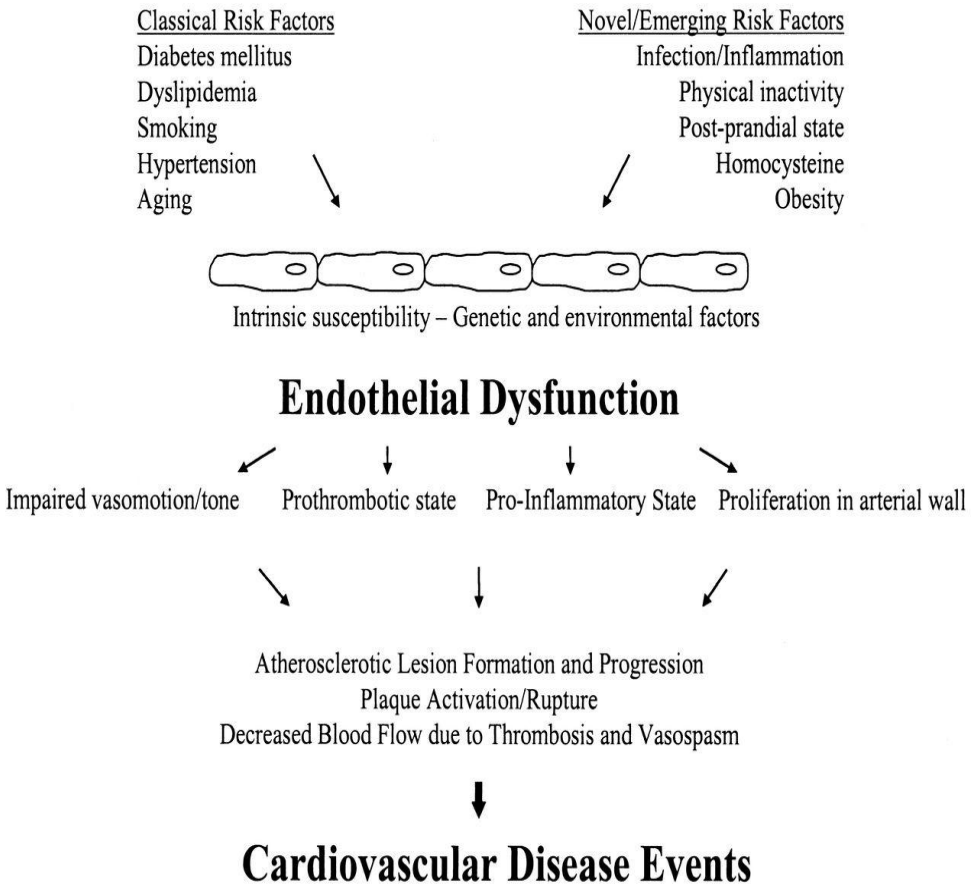

Figure 5 (Endothelial Damage in Smokers) (12)

10-Inflammation

Studies suggest that inflammation affects atherosclerosis as high leukocyte counts and high CRP and fibrinogen levels are potent predictors of future cardiovascular events. However, the mechanism by which tobacco smoking promotes inflammation has not been fully elucidated (Fig. 6) (13).

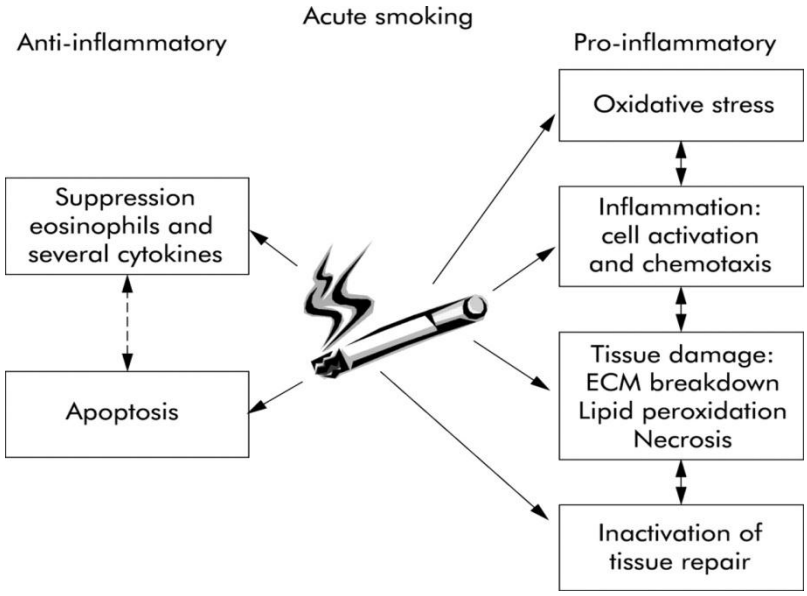

Figure 6 (inflammation pathogenesis in smokers) (13)

11-Smoking and Diabetes

Numerous studies have shown that smoking negatively affects glucose and lipid metabolism in people with or without diabetes. The investigators reported that smoking in diabetic patients was associated with decreased metabolic control and an increased risk of microvascular and macrovascular complications and death (Fig. 7) (14).

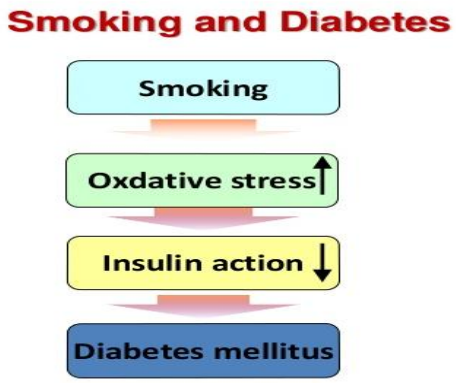

Figure 7 (How Smoking causes Diabetes) (14)

Complications:

1-Microvascular Complications

The microvascular disorders of diabetes (retinopathy, nephropathy, neuropathy) are associated with metabolic control in type 1 and type 2 diseases. The mechanisms of development of

microvascular diseases are not fully understood, although several pathogenic mechanisms have been proposed. Hyperglycemia plays a key role as a trigger for downstream events such as the conversion of glucose to sorbitol by aldose reductase; non-enzymatic glycosylation of proteins and receptors in affected tissues; increased exposure to oxidative stress; and activation of protein kinase C and mitogen-activated protein kinases (Fig. 7, 8) (15).

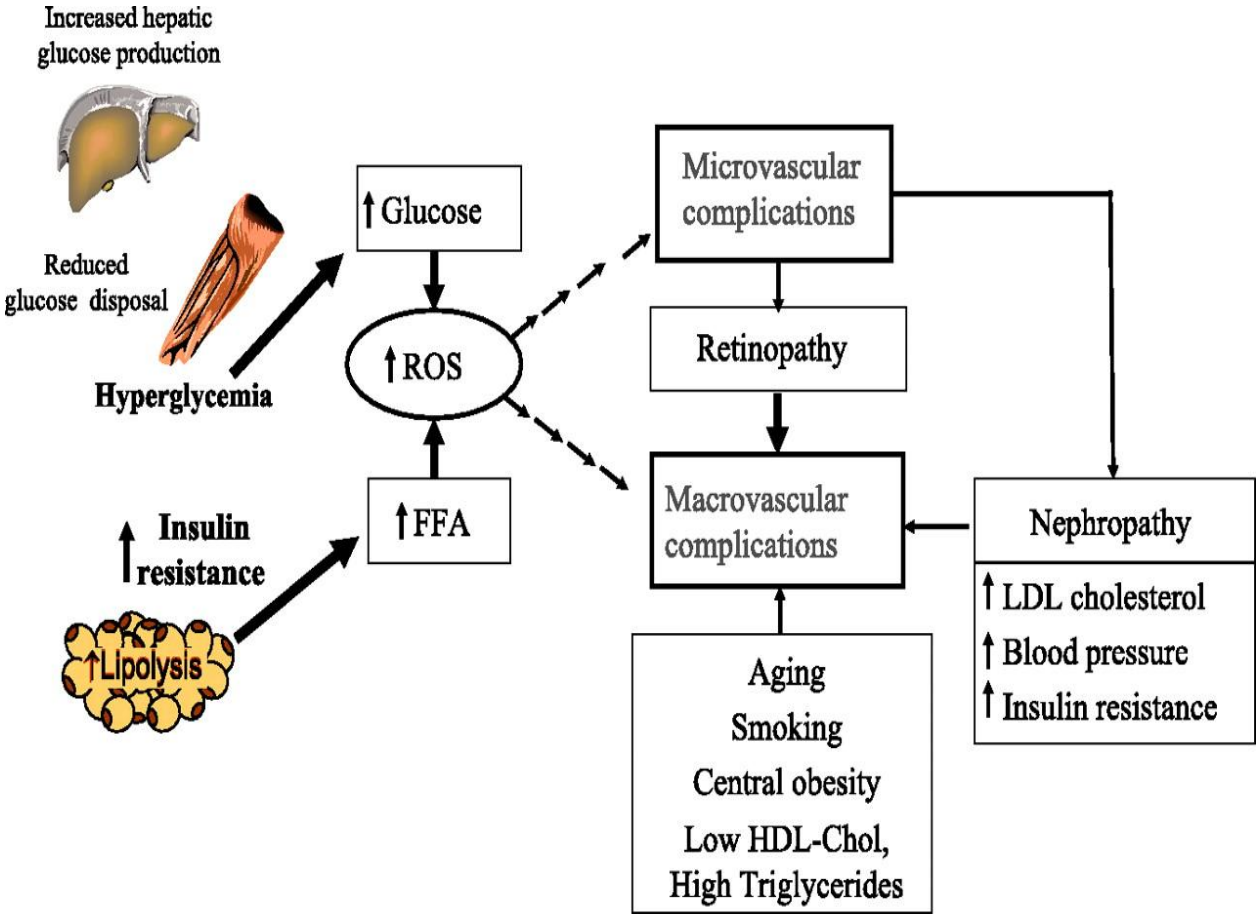

Figure 8 (Microvascular Complications of Smoking) (15)

1.1-Nephropathy:

Smoking and hyperglycemia increase oxidative stress and lipid accumulation, which regulates TGF-β, accumulates AGEs, reduces nitric oxide production, leads to inflammation of the lower glomerular membrane and mesangial proliferation, and further progression of mesangial glomerulosclerosis and interstitial fibrosis, as well as the effects of fibrosis (fig. 9) (15).

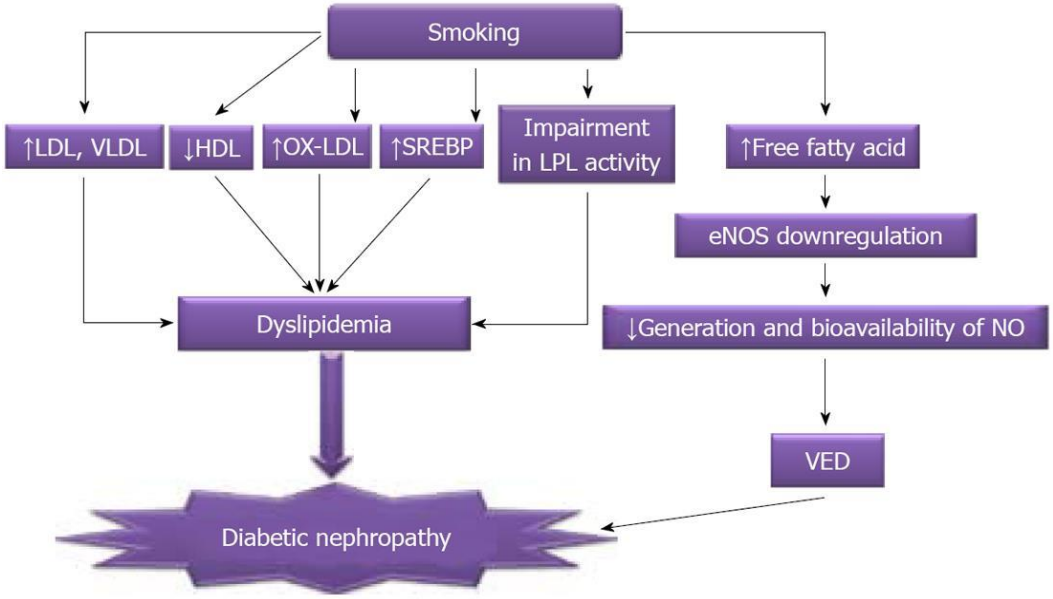

Figure 9 (How Smoking Causes Diabetic Nephropathy) (15)

1.2-Retinopathy

In general, researchers have never considered smoking to be a major risk factor for diabetic retinopathy. The results of a very large multicenter study show no strong support for such associations, except in the elderly with certain medical conditions. However, at least two studies of patients with type 1 diabetes suggest that smoking prioritizes patients with this retinopathy (Fig. 10) (16).

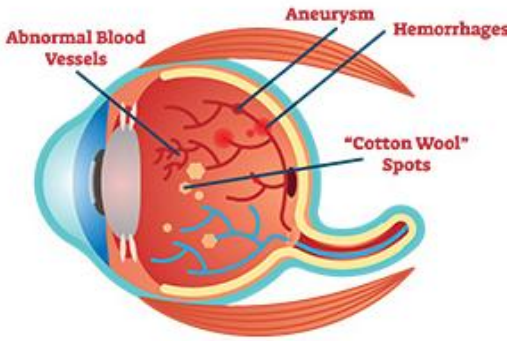

Figure 10 (Effect of Smoking on the Eye) (16)

1.3-Neuropathy

Smoking affects blood circulation and can increase the risk of peripheral neuropathy. Care should be taken in foot care, as peripheral neuropathy can begin slowly with numbness in the feet. Check your feet daily for cuts and other wounds (Fig. 11) (17).

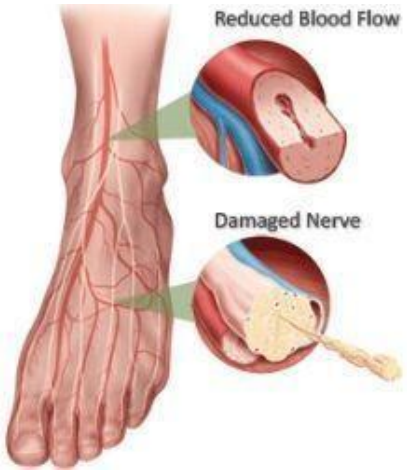

Figure 11 (Effect of Smoking on Neurons) (17)

2-Macrovascular Complications

Patients with diabetes are at increased risk of smoking-related complications as they are at increased risk of coronary artery disease and death. In a study group called the Multinational Study of Vascular Diseases of Diabetes, sponsored by the World Health Organization, in a study group in London, England, among other things, smokers with type 1 or type 2 diabetes did not significantly increase their risk of CHD. After an eighth year in the Stroke Diabetes and Complication Management Study (New England Journal of Medicine 1993), which examined the role of intensive insulin therapy and glycemic-controlled therapy in type 1 diabetes, smoking was not a major risk factor for macrovascular complications. Because the participants were small, this study was not well designed to investigate the role of tobacco use. Some studies in young participants with type 1 diabetes have reported that smoking increases the risk of CHD (Fig. 12) (18).

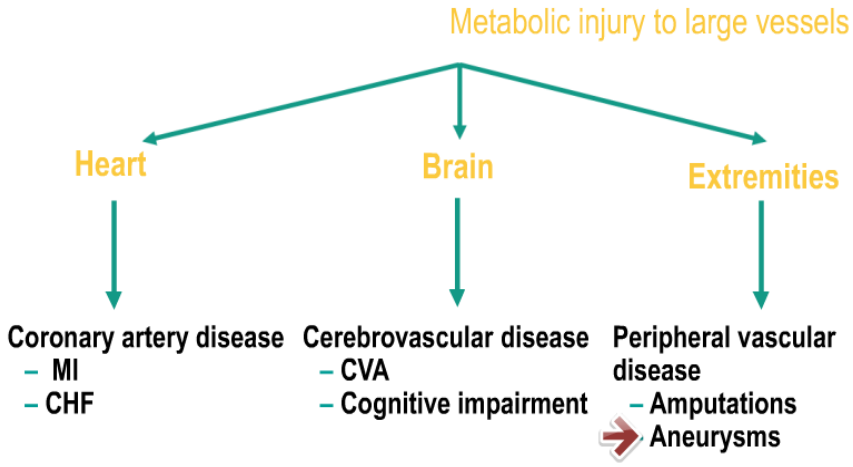

Figure 12 (Macrovascular Complications of Smoking) (18)

First, Second, and Third Hand Smoke Effects

Smoking's risks are well-known. Inhaling nicotine and other hazardous compounds in cigarette smoke, whether as a smoker or a nonsmoker, can lead to ailments such as heart disease, stroke, and lung cancer. The impact of so-called "thirdhand smoke," a term coined to describe the potentially cancer-causing compounds that form when tobacco smoke particles mix with gases in the air and absorb into nearby surfaces such as carpets, rugs, clothes, bed sheets, wall paint, car dashboards, and even toys, is less well understood. Tobacco smoke residue can linger in these materials for years after a cigarette has been extinguished, and many researchers are concerned that this could be damaging to people's health. However, evaluating the risk of thirdhand smoke is challenging, and research on its effects is limited (19).

When particles from a cigarette or other tobacco-burning device infiltrate into and are absorbed by things such as hair, clothes, furniture, carpet, and walls, thirdhand smoke is produced. After then, the compounds are aged, which affects their chemical structure. Nicotine forms carcinogens, or substances that may cause cancer, when it combines with indoor air pollutants like nitrous acid. In a process known as "off-gassing," the gas is repeatedly re-emitted back into the air. (20).

Attempts to diffuse the smoke, such as opening windows or using a fan, do not prevent thirdhand smoke from forming or being inhaled, and the residue may emit harmful chemicals for years, if not decades. "It's not a one-time phenomenon," Dr. Bechara explains of thirdhand smoke. "It's a phenomenon that develops over time as a result of increased exposure." Normal cleaning methods are ineffective against contaminants as well. Most of the time, the only options are to replace carpets or repaint the walls (21).

Thirdhand smoking has been a research focus for decades, even though it is a relatively new word. It was first discovered in 1953 when a scientist from Washington University School of Medicine in St. Louis discovered that painting mice with tobacco smoke condensate—or liquid from gas condensation—caused cancer. Researchers discovered nicotine in the dust of smokers' homes in a 1991 investigation. A further study, published in 2004, discovered that nicotine was still present in residences where smokers attempted to reduce their exposure by smoking outside. A 2008 study comparing smokers' automobiles to non-smokers' cars found comparable results, including detecting tobacco residue on the dashboards of cars driven by smokers who had outlawed smoking in their vehicles (22).

When it was first used in a 2009 research published in Pediatrics, the phrase "thirdhand smoke" became widely known. The authors hypothesized that emphasizing the health risks of thirdhand smoke to children could motivate adults to quit: "Children are more vulnerable to thirdhand smoke exposure because they breathe near, crawl and play on, touch, and mouth polluted surfaces." The American Academy of Pediatricians released suggestions to minimize children's exposure to reduce the danger. "Smokers must be aware of this because it may serve as an

incentive for them to quit for the sake of themselves and their loved ones," Dr. Bechara says. "Becoming aware of thirdhand smoke can help smokers recognize that they are potentially harming others as well as themselves." (23).

Different Types of Smoking in Asian Countries

Hookah (sheesha), bidi, and cigars were mentioned as additional country-specific possibilities; bidi was mentioned in India, Pakistan, Nepal, and Bangladesh; cigars in the Philippines; hand-rolled tobacco in Timor Leste; Pan Masala and Gutkha in India; and nuswar in Pakistan. If a respondent answered 'yes' to the first question, 'pipe' to the fourth question, or smoked hookah, bidi, cigars, or hand-rolled tobacco, they were classed as 'current smokers.' If the fourth question was answered with any kind of SLT, such as 'chewing' tobacco, 'Pan Masala,' 'Gutkha,' and snuff,' the respondents were classed as 'current SLT users.' (23).

Methods to Reduce Exposure

Behavioral and pharmacological treatments, which dramatically boost rates of long-term abstinence from smoking, are examples of evidence-based approaches for treating smokers. Despite this, absolute abstinence rates are low; they range from 8% to 25%, depending on the study group and therapy. Furthermore, at any given time, only a tiny percentage of smokers are interested in treatment. Patients with CVD have a higher interest in quitting smoking and have a better chance of succeeding at long-term abstinence than the overall population of smokers. Despite this, abstinence rates remain dismally low, especially given the significant health benefits for this demographic when they quit smoking (23).

Due to poor treatment outcomes, researchers are interested in evaluating strategies that could reduce the risk of smoking among people who continue to smoke. Although research on whether harm is truly decreased with reduced cigarette exposure is scarce, these tactics are sometimes referred to as "harm reduction" approaches. To present, only a few clinical trials, prospective cohort studies, and epidemiologic studies have looked at the impact of approaches for lowering exposure on CVD risk factors and development (23).

Measures of tobacco constituent exposure (e.g., nicotine and CO); biomarkers of inflammation (e.g., CRP, leukocyte counts, and fibrinogen); thrombosis (e.g., fibrinogen and PAI-1); lipid abnormalities (e.g., total cholesterol, HDLc, LDLc, triglycerides, APOs A-I and B, and HDLc to LDLc ratio); and oxidative stress, which (blood pressure, heart rate, angina, exercise tolerance, MI, other adverse events, and death) (24).

The INTERHEART study

Background

Although low- and middle-income nations account for more than 80% of the worldwide burden of cardiovascular disease, understanding of the importance of risk factors is mostly derived from

rich countries. As a result, in most parts of the world, the impact of such factors on the risk of coronary heart disease is unknown (24).

Methods

A systematic case-control study of acute myocardial infarction was constructed in 52 nations that represented every inhabited continent. A total of 15152 patients and 14820 controls were enrolled in the study. This study looks at the link between smoking, hypertension, diabetes, waist/hip ratio, dietary patterns, physical activity, alcohol use, blood apolipoproteins (Apo), and psychosocial factors and myocardial infarction. The association of risk variables to myocardial infarction and their population attributable risks (PAR) were calculated using odds ratios and their 95% confidence intervals (25).

Findings

Smoking (odds ratio 2·87 for current vs never, PAR 35·7% for current and former vs never), raised ApoB/ApoA1 ratio (3·25 for top vs lowest quintile, PAR 49·2% for top four quintiles vs lowest quintile), history of hypertension (1·91, PAR 17·9%), diabetes (2·37, PAR 9·9%), abdominal obesity (1·12 for top vs lowest tertile and 1·62 for middle vs lowest tertile, PAR 20·1% for top two tertiles vs lowest tertile), psychosocial factors (2·67, PAR 32·5%), daily consumption of fruits and vegetables (0·70, PAR 13·7% for lack of daily consumption), regular alcohol consumption (0·91, PAR 6·7%), and regular physical activity (0·86, PAR 12·2%), were all significantly related to acute myocardial infarction ($p<0·0001$ for all risk factors and $p=0·03$ for alcohol). These associations were noted in men and women, old and young, and in all regions of the world. Collectively, these nine risk factors accounted for 90% of the PAR in men and 94% in women (26).

Interpretation

In both sexes and at all ages in all areas, abnormal lipids, smoking, hypertension, diabetes, abdominal obesity, psychosocial variables, consumption of fruits, vegetables, and alcohol, and regular physical activity contribute to the majority of the risk of myocardial infarction. This data implies that prevention strategies can be based on comparable principles over the world and have the ability to prevent the majority of myocardial infarctions before they occur (27).

Discussion

Tobacco kills about one person every six seconds, killing 1 in 10 adults. Up to half of the current consumers will eventually die of tobacco-related diseases. About 80 percent of the world's more than 1 billion smokers live in low- and middle-income countries, where the burden of tobacco-related diseases and deaths is much higher. Every year an estimated 6 million people worldwide die from the effects of smoking. About half of children regularly breathe air polluted with cigarette smoke in public places. More than 40% of children have at least one parent who

smokes. Secondhand smoke causes 600,000 premature deaths each year. According to the 2012 Eurobarometer report "European Attitude Tobacco", the prevalence of smoking among Europeans aged 15 years and older is 28%, with the highest prevalence in Greece (40%) and the lowest in Sweden (13%). Italy (like the Netherlands) is in fourth place with a frequency of 24% from last time. The 2012 Italian DOXA survey, which is commissioned every year by the Istituto Superiore di Sanita, found a low rate of 21% (28).

Specifically, in Egypt, 37.7% of men, 0.5% of women, and a total of 19.4% (9.6 million adults) currently smoke. In the country, 35.8% of men and 0.5% of women smoke every day. 6.2% of men, 0.3% of women, and a total of 3.3% (1.6 million adults) currently use hookah. 6.5 million adults (60.7 percent of adults whose workplace includes home) are exposed to secondhand smoke. Another study of high school students in the suburbs of Egypt found that 19% of people ever smoked hookah. Three studies of older men living in the valleys found that 11-15% smoked in pipe groups. The percentage of smokers is also high among healthcare professionals and medical students. According to a study published in 2010, the prevalence of smoking among healthcare workers in Italy is 44%, more than double the general population, and this is only due to an increase in the number of nurses (48.2%). Doctors (33, 9%), medical students. (35%) and graduate students (52.9%) (29).

Three-quarters of cigarette smoke has received much attention contributing to CVD: nicotine, carbon monoxide (CO) and oxidizing gases, and other cigarette smoke components, including atherogenesis in the pathology of CVD. Nicotine, which is rapidly absorbed in cigarette smoke, is found in blood levels of 40 to 100 ng/ml after each cigarette is consumed. The usual amount of nicotine taken systematically per cigarette is 1 to 2 milligrams (mg). This pattern of deposition is associated with the end of a half-life of nicotine in two hours. In smokers, nicotine levels in venous plasma reach the plateau early in the morning and remain at this level until bedtime. Thus, these results show that the average smoker receives significant nicotine levels 24 hours a day. Nicotine is sympathetic to the release of catecholamines locally from neurons and the adrenal gland. In studies of the pharmacodynamics of nicotine, the severity of its main effects is more rapid (30).

Summary and Conclusion

Smoking, with its parent ingredients, nicotine, and CO, increases oxidative stress, endothelial damage, and dysfunction, is associated with higher concentrations of total cholesterol and serum triglycerides, lowers cardioprotective HDL and, by promoting intravascular inflammation development is an important risk factor for Atherosclerosis and heart disease. Nicotine decreases self-regulation cardiac control, increases sympathetic activity, and increases resting HR, while sustained exercise slows HR levels and reduces elevated HR gains. Overall, smoking is a very important factor in the ever-increasing risk of heart disease, a major risk factor for heart disease and death, and is recognized as the leading cause of preventable death worldwide. Smoking causes endothelial damage and dysfunction of the coronary and peripheral arteries. Exposure to

secondhand smoke increases the risk of thrombosis, stroke, which is a major factor in the pathogenesis of cardiovascular events.

Smoking produces a chronic inflammatory condition that contributes to atherogenic disease processes and increases the level of biological symptoms of inflammation, powerful predictors of cardiovascular events. Smoking produces an atherogenic lipid profile mainly due to an increase in triglycerides and a decrease in high-density lipoprotein cholesterol. The deleterious effects of smoking on the cardiovascular system and other organs must be addressed throughout the medical community. Cardiologists must collaborate with other professionals to promote integrated legal measures, economic measures, and public education as we work to end tobacco use.

Tobacco smoke is made up of more than 4,700 different compounds. At least 250 of them are considered dangerous and more than 50 cause cancer. Specifically, tobacco smoke contains aromatic and non-aromatic hydrocarbons (dioxene and benzopyrene), alpha and beta aldehydes (acrolein), heavy metals (cadmium, zinc, and iron), and a wide range of gases. Toxic (nitrogen dioxide, nitric oxide, and carbon monoxide) and bacterial compounds (lipopolysaccharide [LPS]) have important biological effects on the innate and resilient immune system. The effect of these many components is obvious. Most importantly, some of the effects produced by some of these ingredients were unlike those of secondhand smoke. Thus, it can be assumed that the effects of tobacco smoke cannot be predicted by studying the individual components, but are better estimated using tobacco smoke.

Ethical Approval:

As per international standard or university standard ethical approval has been collected and preserved by the authors.

Conflict of Interest

There is no conflict of interests to declare.

References:

1) Li B, Li D, Liu JF, Wang L, Li BZ, Yan XJ, et al. (June 2021). ""Smoking paradox" is not true in patients with ischemic stroke: a systematic review and meta-analysis". Journal of Neurology. 268 (6): 2042–2054.
2) Arafa A, Mostafa A, Navarini AA, Dong JY (August 2020). "The association between smoking and risk of skin cancer: a meta-analysis of cohort studies". Cancer Causes & Control. 31 (8): 787–794.

3) Lai O, Recke A, Zillikens D, Kasperkiewicz M (August 2018). "Influence of cigarette smoking on pemphigus - a systematic review and pooled analysis of the literature". Journal of the European Academy of Dermatology and Venereology. 32 (8): 1256–1262.
4) Wijarnpreecha K, Lou S, Panjawatanan P, Cheungpasitporn W, Pungpapong S, Lukens FJ, Ungprasert P (November 2018). "Cigarette smoking and risk of celiac disease: A systematic review and meta-analysis". United European Gastroenterology Journal. 6 (9): 1285–1293.
5) To N, Ford AC, Gracie DJ (July 2016). "Systematic review with meta-analysis: the effect of tobacco smoking on the natural history of ulcerative colitis". Alimentary Pharmacology & Therapeutics. 44 (2): 117–26.
6) Chen H (2018). "The changing landscape of Parkinson epidemiologic research". Journal of Parkinson's Disease. 8 (1): 1–12.
7) Ascherio A, Schwarzschild MA (2016). "The epidemiology of Parkinson's disease: risk factors and prevention". The Lancet. Neurology. 15 (12): 1257–1272.
8) Oertel W, Schulz JB (October 2016). "Current and experimental treatments of Parkinson disease: a guide for neuroscientists". Journal of Neurochemistry. 139 (Suppl 1): 325–337.
9) Hopper CP, De La Cruz LK, Lyles KV, Wareham LK, Gilbert JA, Eichenbaum Z, et al. (December 2020). "Role of Carbon Monoxide in Host-Gut Microbiome Communication". Chemical Reviews. 120 (24): 13273–13311.
10) Weinblatt E, Shapiro S, Frank CW, Sager RV (August 1968). "Prognosis of men after first myocardial infarction: mortality and first recurrence in relation to selected parameters". American Journal of Public Health and the Nation's Health. 58 (8): 1329–47.
11) Sagud M, Mihaljevic Peles A, Pivac N (September 2019). "Smoking in schizophrenia: recent findings about an old problem". Current Opinion in Psychiatry. 32 (5): 402–408.
12) Bray F, Tyczynski JE, Parkin DM (January 2004). "Going up or coming down? The changing phases of the lung cancer epidemic from 1967 to 1999 in the 15 European Union countries". European Journal of Cancer. 40 (1): 96–125.
13) Shah RS, Cole JW (July 2010). "Smoking and stroke: the more you smoke the more you stroke". Expert Review of Cardiovascular Therapy. 8 (7): 917–32.
14) Mähönen MS, McElduff P, Dobson AJ, Kuulasmaa KA, Evans AE (2004). "Current smoking and the risk of non-fatal myocardial infarction in the WHO MONICA Project populations". Tobacco Control. 13 (3): 244–250.
15) Saha SP, Bhalla DK, Whayne TF, Gairola C (Autumn 2007). "Cigarette smoke and adverse health effects: An overview of research trends and future needs". The International Journal of Angiology. 16 (3): 77–83.

16) Narkiewicz K, Kjeldsen SE, Hedner T (2005). "Is smoking a causative factor of hypertension?". Blood Pressure. 14 (2): 69–71.
17) American Cancer Society (2004). "Questions About Smoking, Tobacco, and Health". Journal of the National Cancer Institute. 96 (11): 853–861.
18) Dietrich T, Maserejian NN, Joshipura KJ, Krall EA, Garcia RI (April 2007). "Tobacco use and incidence of tooth loss among US male health professionals". Journal of Dental Research. 86 (4): 373–7.
19) Al-Bayaty FH, Wahid NA, Bulgiba AM (February 2008). "Tooth mortality in smokers and nonsmokers in a selected population in Sana'a, Yemen". Journal of Periodontal Research. 43 (1): 9–13.
20) Davies PD, Yew WW, Ganguly D, Davidow AL, Reichman LB, Dheda K, Rook GA (April 2006). "Smoking and tuberculosis: the epidemiological association and immunopathogenesis". Transactions of the Royal Society of Tropical Medicine and Hygiene. 100 (4): 291–8.
21) Jha P, Jacob B, Gajalakshmi V, Gupta PC, Dhingra N, Kumar R, Sinha DN, Dikshit RP, Parida DK, Kamadod R, Boreham J, Peto R (March 2008). "A nationally representative case-control study of smoking and death in India". The New England Journal of Medicine. 358 (11): 1137–47.
22) Goedert JJ, Vitale F, Lauria C, Serraino D, Tamburini M, Montella M, Messina A, Brown EE, Rezza G, Gafà L, Romano N (November 2002). "Risk factors for classical Kaposi's sarcoma". Journal of the National Cancer Institute. 94 (22): 1712–8.
23) Kendirci M, Nowfar S, Hellstrom WJ (January 2005). "The impact of vascular risk factors on erectile function". Drugs of Today. 41 (1): 65–74.
24) Dechanet C, Anahory T, Mathieu Daude JC, Quantin X, Reyftmann L, Hamamah S, Hedon B, Dechaud H (2011). "Effects of cigarette smoking on reproduction". Human Reproduction Update. 17 (1): 76–95.
25) Hamer M, Stamatakis E, Batty GD (August 2010). "Objectively assessed secondhand smoke exposure and mental health in adults: cross-sectional and prospective evidence from the Scottish Health Survey". Archives of General Psychiatry. 67 (8): 850–5.
26) Anstey KJ, von Sanden C, Salim A, O'Kearney R (August 2007). "Smoking as a risk factor for dementia and cognitive decline: a meta-analysis of prospective studies". American Journal of Epidemiology. 166 (4): 367–78.
27) Jacobsen LK, Krystal JH, Mencl WE, Westerveld M, Frost SJ, Pugh KR (January 2005). "Effects of smoking and smoking abstinence on cognition in adolescent tobacco smokers". Biological Psychiatry. 57 (1): 56–66.
28) Cataldo JK, Prochaska JJ, Glantz SA (2010). "Cigarette smoking is a risk factor for Alzheimer's Disease: an analysis controlling for tobacco industry affiliation". Journal of Alzheimer's Disease. 19 (2): 465–80.

29) Fratiglioni L, Wang HX (August 2000). "Smoking and Parkinson's and Alzheimer's disease: review of the epidemiological studies". Behavioural Brain Research. 113 (1–2): 117–20.
30) Cataldo JK, Prochaska JJ, Glantz SA (Jul 2010). "Cigarette smoking is a risk factor for Alzheimer's Disease: an analysis controlling for tobacco industry affiliation". Journal of Alzheimer's Disease. 19 (2): 465–80.