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The influences of smoking on the formation of Atherosclerosis

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Abstract. Smoking is closely related to the occurrence and development of many other cardio cerebral Vascular diseases. Smoking can increase the risk of moderate diseases such as hypertension, coronary heart disease, myocardial infarction, and stroke. Smoking will cause Vasoconstriction, increase the load of the heart, lead to poor blood circulation, and then lead to the occurrence of cardio cerebral Vascular disease. In addition, smoking can also have adverse effects on the human immune system. Smoking can lead to a decrease in immune function and increase the risk of infections. Research shows that smokers are more likely to suffer from upper respiratory tract infection, pneumonia and other diseases. Smoking can also cause pulmonary inflammatory reactions, leading to airway stenosis and obstruction, seriously affecting respiratory function. Studying the role of smoking in promoting the formation of Atherosclerosis has important scientific and social significance, which helps to understand the relationship between smoking and Atherosclerosis, provides scientific basis and guidance, promotes health publicity and education, and improves public health.

Keywords: Smoking, Cardiovascular Disease, Atherosclerosis.

1. Introduction

The official data of the National Bureau of Statistics shows that from 2018 to 2021, the number of deaths due to cardio cerebral Vascular disease in China will increase year by year, and tobacco will kill more than 8 million people every year, of which more than 7 million people are due to the direct use of tobacco, about 1.2 million are non-smokers exposed to second-hand smoke [1], and about 12% of cardiovascular disease deaths are attributable to tobacco use and Passive smoking exposure [2]. The products of organic substances in cigarettes after aerobic combustion include nicotine, tar, Benzopyrene, carbon monoxide and other chemical components. The chemicals in tobacco can significantly promote the formation of Atherosclerosis. The mechanism is closely related to promoting inflammatory reactions, inducing oxidative stress damage, and promoting platelet activation.

The research content of this paper is the mechanism of smoking on the formation of Atherosclerosis, to study the effects of smoking on vascular endothelial function, platelet activation, inflammatory reaction and other aspects, and to explore how smoking can promote the formation of Atherosclerosis through these mechanisms. The study of the promotion of smoking on the formation of Atherosclerosis can provide a deeper understanding of the relationship between smoking and the disease. The lack of designated prevention and intervention strategies provides a scientific basis.

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Table 1. Mortality rate of Cerebrovascular disease in urban centers.

	2021	2020	2019	2018
Urban men's crude mortality rate of heart disease (1/100000)	171.26	159.09	153.46	150.13
Urban women's crude mortality rate of heart disease (1/100000)	159.40	152.52	143.42	142.42

Table 1 shows that the incidence rate of heart disease in the city center is increasing year by year, which is attributed to unhealthy lifestyles, bad eating habits, high-pressure life and work pressure, environmental pollution, and bad social habits, such as smoking and drinking.

2. Analysis of the influences of smoking on humans' health

Smoking could damage the human respiratory system, circulatory system, digestive system, Genitourinary system, and immune system. Smoking will damage the defense mechanism of the lungs and increase the risk of Upper respiratory tract infection, Chronic obstructive pulmonary disease and other diseases; Increase the risk of cardio cerebral Vascular disease, including coronary heart disease, myocardial infarction, stroke, increase blood pressure, and increase heart load; Have symptoms such as male erectile dysfunction, Female infertility, premature delivery, and fetal dysplasia; Causes a decrease in immune function and increases the risk of infection. In addition, smoking can also cause skin aging, trigger oral diseases, increase the risk of osteoporosis, and even increase the risk of cancer.

3. Mechanism of smoking in promoting the formation of Atherosclerosis

3.1. Endothelial dysfunction

Endothelial cells are a layer of cells in the intima of blood vessels, which have functions such as regulating vascular relaxation and contraction, inhibiting platelet aggregation, and white blood cell adhesion [3]. Harmful substances in smoking, such as nicotine and carbon monoxide, can directly damage endothelial cells, leading to abnormal endothelial function. The main manifestations of endothelial dysfunction caused by smoking are: endothelial cell damage, and harmful substances in smoking can cause direct damage to endothelial cells, leading to damage to cell membranes and dysfunction; Endothelial cell dysfunction. Smoking can reduce the ability of endothelial cells to produce nitric oxide (NO), weaken the Vasodilation function, and increase the tension of the vascular wall [4]. Endothelial inflammatory response can be activated by smoking, which increases the release of inflammatory mediators and leads to the occurrence of endothelial inflammatory response. The permeability of blood vessels increases. Smoking can lead to an increase in the permeability of endothelial cells, making the blood vessel wall more prone to leakage, and promoting lipid deposition and plaque formation [5].

3.2. Promoting lipid deposition

The effects of smoking on lipid metabolism mainly include the following aspects: improving the level of low-density lipoprotein (LDL) cholesterol, smoking can increase the level of LDL cholesterol in the blood, making it deposit in the vascular wall, and form the basis of Atherosclerosis; Reduce the level of High-density lipoprotein (HDL) cholesterol. Smoking can reduce the level of HDL-positive sterol in the blood. HDL is a kind of lipoprotein with a vascular protection effect. Its reduction will reduce the clearance and transport of cholesterol, further promoting the deposition of lipids in the vascular wall. The harmful substances in smoking can cause oxidative stress, produce a large number of free radicals and oxides, disrupt the stability of lipids, leading to lipid oxidation [6], promote the deposition of lipids in the vascular wall, and increase the uptake of lipids by endothelial cells. Smoking can increase the uptake of lipids in endothelial cells, making it easier for them to deposit on the vascular wall.

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3.3. Platelet activation

Smoking can activate platelets, thus promoting the formation of Atherosclerosis [7]. The influence of smoking on platelets is mainly reflected in the following aspects: platelet aggregation. Smoking can activate platelets and increase the degree of platelet aggregation, so that platelets can aggregate into thrombus on the inner wall of blood vessels, which will lead to stenosis and obstruction of the vascular lumen, thus promoting the formation of Atherosclerosis; Platelet activation, harmful substances in smoking can directly stimulate platelets, making them in an activated state; Activated platelets will release a series of bioactive substances, such as Platelet-derived growth factor (PDGF), platelet activating factor (PAF), etc [8]. These substances will cause inflammatory reactions and vascular endothelial cell damage, thereby promoting the occurrence of Atherosclerosis, and changes in platelet function. Smoking can alter platelet function, making it easier to bind to damaged areas on the inner wall of blood vessels, thereby promoting platelet activation and aggregation, and ultimately forming thrombi.

3.4. Oxidative stress response

Harmful substances produced by smoking, such as nicotine and carbon monoxide, can cause oxidative stress reactions [9]. The deposition of low-density lipoprotein (LDL) in the artery intima is the initiating factor of Atherosclerosis. Under the effect of reactive oxygen species secreted by vascular cells, "primitive" LDL becomes oxidized LDL (ox LDL), stimulates endothelial cells to secrete a variety of inflammatory factors, induces monocytes to adhere to and migrate into the artery intima, and transforms into macrophages. Ox LDL can also induce macrophages to express scavenger receptors and promote their uptake of lipoproteins to form Foam cells. Meanwhile, ox-LDL is an activator of NADPH oxidase, which can enhance its activity, promote the production of reactive oxygen species, and is more conducive to the oxidation of LDL to ox-LDL. In addition, ox LDL can inhibit the production of nitric oxide and its biological activity, making Vasodilation function abnormal. Specifically, oxygen free radicals can damage vascular endothelial cells, causing them to produce inflammatory reactions and proliferation, and then trigger inflammatory reactions and damage to the vascular wall. At the same time, oxygen free radicals can also oxidize low-density lipoprotein (LDL), making it easier to be absorbed and deposited on the vascular wall, forming the basis of Atherosclerosis [10].

3.5. Inflammatory reactions

Harmful substances in smoking, such as nicotine, carbon monoxide, and polycyclic aromatic hydrocarbons, can cause inflammatory reactions [11]. These substances can stimulate vascular endothelial cells and immune cells to produce inflammatory mediators and cytokines, such as Interleukin 6 (IL-6), tumor necrosis factor (TNF), etc. These inflammatory mediators and cytokines can trigger inflammatory reactions, leading to inflammation and damage to the vascular wall. The inflammatory reaction is one of the important links in the formation of Atherosclerosis. Inflammatory reactions can cause damage and dysfunction of vascular endothelial cells, rendering them ineffective in protecting the vascular wall. At the same time, inflammatory reactions will also attract and activate immune cells, such as monocytes and macrophages, which will ingest and oxidize low-density lipoprotein (LDL) to form foam cells. The accumulation of foam cells can lead to the formation of Atherosclerosis plaque.

In addition, inflammatory reactions can promote the proliferation and fibrosis of blood vessel walls, forming the solidification and stability of plaques. Stable plaques can trigger thrombosis, further leading to vascular stenosis and obstruction.

3.6. Interference with blood coagulation function

Smoking can produce harmful substances such as nicotine and carbon monoxide, which can affect the normal function of the blood coagulation system [12]. Specifically, smoking can lead to increased platelet aggregation and activation, promoting the formation of blood clots. Smoking also affects the function of vascular endothelial cells, making them produce coagulation factors and substances that inhibit Plasmin, further increasing the risk of thrombosis

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The formation of thrombus is one of the important links of Atherosclerosis. In Atherosclerosis plaque, coagulation factors such as platelets and fibrin will aggregate and form thrombus, further blocking blood vessels. The formation of thrombus will lead to insufficient blood supply, aggravate the inflammatory reaction and damage of blood vessels, and promote the progress of Atherosclerosis.

In addition, smoking will also reduce the activity of the Plasmin system, reducing the ability of thrombolysis. This can also lead to the accumulation of blood clots and narrowing of blood vessels.

4. Measures taken by the government and the public

Government should promulgate and implement strict anti-smoking laws and regulations, including prohibiting smoking in public places, restricting tobacco advertising and sales, etc.; strengthen tobacco regulation and law enforcement efforts, and crack down on illegal tobacco trading and smuggling; Raise tobacco taxes to increase the price of tobacco products, thereby reducing the number of smokers. Support smoking cessation projects and services, provide smoking cessation medication and counseling support. Strengthen anti-smoking education and publicity for young people, including conducting relevant activities in schools and communities.

For the public, they should stop smoking or minimize the frequency and quantity of smoking as much as possible, actively participate in smoking cessation plans and support organizations, seek help and support for quitting smoking; actively maintain smoking ban orders in public places and remind others not to smoke, promote the importance of smoking cessation through social media and personal influence, and encourage those around you to quit smoking.

5. Conclusion

This paper analyzes the mechanism of smoking in promoting the formation of Atherosclerosis, and concludes that there is a significant positive correlation between smoking and arterial drowning-like sclerosis. The mechanism of smoking promoting the formation of Atherosclerosis is multifaceted. Firstly, smoking can cause arterial endothelial damage and inflammatory reactions. The chemicals and harmful substances in tobacco can directly contact and damage vascular endothelial cells, disrupting the integrity of vascular walls. This type of injury can trigger an inflammatory response, prompting white blood cells and inflammatory cells to aggregate on the blood vessel wall, forming plaques. Secondly, smoking increases oxidative stress and platelet activation. The chemicals and harmful substances in tobacco produce a large amount of free radicals, leading to an increase in oxidative stress. Oxidative stress can damage the vascular wall and promote the formation of plaques. In addition, smoking can also cause platelet activation and aggregation, further exacerbating the formation of plaques. In addition, smoking can also reduce the level of High-density lipoprotein cholesterol in the blood and increase the oxidation and deposition of low-density lipoprotein. High-density lipoprotein cholesterol helps to remove cholesterol from the blood vessel wall and protect the health of blood vessels. Smoking will reduce the level of High-density lipoprotein cholesterol, making the cholesterol on the vascular wall easier to be oxidized and deposited by low-density lipoprotein, forming plaque. To sum up, there is a significant positive correlation between smoking and Atherosclerosis. Smoking can cause arterial endothelial damage and inflammatory reaction, increase oxidative stress and platelet activation, reduce the level of High-density lipoprotein cholesterol, increase the oxidation and deposition of low-density lipoprotein, and further promote the development of Atherosclerosis. Therefore, the public and health departments should take effective measures to reduce smoking behavior to reduce the occurrence and development of Atherosclerosis, thereby reducing the risk of cardiovascular disease.

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