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Tobacco Smoking and Respiratory Diseases

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Annotation: The endothelial cells of the capillaries of the small circulatory circle are damaged – the phenomenon of endothelial dysfunction. It is difficult to imagine the scale of the harmful effects of tobacco smoke, to which a person exposes himself throughout his life.

Keywords: tobacco smoke, pathological process, human body.

Reactive forms of aldehydes and quinones (quinon) play a leading role in the formation of oxidative stress. Tobacco smoke causes not only acute damage to the lung tissue, but is also the direct cause of the inflammatory process; macrophages, neutrophils, dendritic cells and CD+8 T–lymphocytes migrate from the vascular lumen into the lung tissue. In local inflammatory foci, the level of pro-inflammatory mediators increases. As you know, cell division is a highly regulated process. Copies of DNA and chromosomes and their distribution in dividing cells are controlled by the cyclin/cyclin–dependent kinase complex. This family of proteins plays an important role in regulating the process of cell proliferation, which is especially evident in genotoxic stressesCell aging is associated with these biological processes. This hypothesis formed the basis of the study by Yao H. et al. (2008), which showed that the ablation of the p21 protein gene significantly modifies the inflammatory response to the penetration of tobacco smoke into the respiratory tract of an experimental animal. The data are extrapolated to the human population, and they are cited as an argument for the greater sensitivity of a certain part of people to the damaging effects of tobacco smoke.

A relatively new direction is the study of the genotoxicity of tobacco smoke and its role in accelerating the aging process of the human body. The biological process of cell aging occurs due to an irreversible stop of cell division and growth, which, in turn, occurs due to DNA damage. Tobacco smoking leads to oxidative and genotoxic stress, a special role is assigned to active forms of oxygen, the latter are associated with the process of DNA damage. Naturally, this process is more complicated. However, it should be emphasized that for the first time in the history of studying the effect of tobacco smoke on the human body, molecular mechanisms affecting the process of cell division, DNA damage, disruption of the process of phosphorylation, regeneration, proliferation and apoptosis are described.

The functional activity of the cyclin/cyclin kinase complex also plays an important role in the formation of the inflammatory process. A decrease in phagocytic activity and the number of alveolar macrophages may be the biological cause of a smoking person's tendency to infectious diseases of the respiratory tract. It should be emphasized that COPD patients have low sensitivity to the action of inhaled glucocorticosteroids, which explains the low anti-inflammatory activity of existing inhaled medications used in treatment programs for patients suffering from COPD. A

person who smokes tobacco products has a peculiar situation: on the one hand, he is susceptible to respiratory infection, on the other – he has reduced the anti-inflammatory activity of many medicines.

During the formation of this imbalance, which is characterized as oxidative stress, damage occurs to the biological membranes of cells that are part of the structure of lung tissue. Acute damage to lung tissue in chronic tobacco smoking is transformed into a chronic inflammatory process of the respiratory tract. The result of the inflammatory process is a slow but progressive decrease in the ventilation function of the lungs. The clinical picture of this pathological process is known as chronic obstructive pulmonary disease. The balance in the oxidants–antioxidants system can be maintained by an increased content of antioxidants in food (exogenous sources of antioxidants) and increased synthesis of endogenous substances with antioxidant activity. The main endogenous antioxidant capacity is represented by glutathione (GSH). The epithelial cover of the respiratory tract contains high concentrations of glutathione, significantly exceeding other organs and systems of the human body in these parameters. In people who smoke, the concentration of glutathione increases even more, which reflects endogenous adaptive processes aimed at combating oxidative stress. Glutathione transferase plays an important role in neutralizing xenobiotics entering the human body in different ways. De novo glutathione synthesis occurs in two stages.

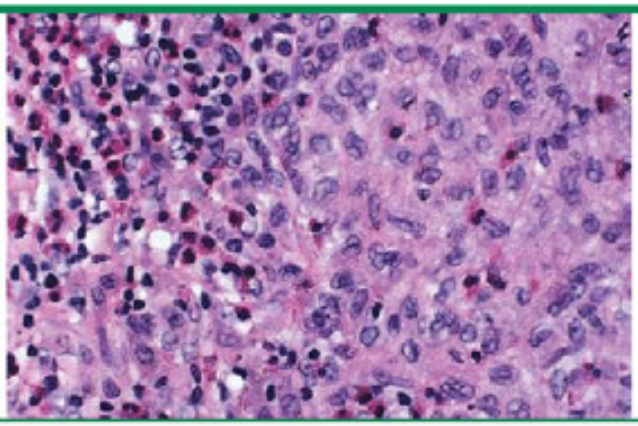
COPD occurs and develops in humans under the influence of a complex of factors that can be characterized as clinical and, on the other hand, molecular (such as genetic). This circumstance explains why only one of the two individuals develops COPD. It should be emphasized that smoking is an aggressive risk factor for the development of this form of lung pathology in humans. As is known, the functional development of the lungs in humans ends after the age of 20 years. Tobacco smoking leads to a reduction in the ventilation function of the lungs, the parameters of FVD decrease prematurely, respiratory failure increases at an accelerated rate [Tager I.B. et al., 1976]. The damaging effect of tobacco smoke increases dramatically in people with atopic reactions, deficiency of antioxidants. It should be emphasized that in a cold climate and strenuous physical work, inhalation of tobacco smoke provokes the early development of emphysema of the lungs. The genetic factors predisposing to the development of COPD were discussed above, to which should be added the polymorphism of the TNF–a gene, metalloproteinases, microsomal epoxy hydrolase. Genetic markers have historically been studied after Eriksson S. (1964) described a1–antitrypsin deficiency in essential pulmonary emphysema. The clinical manifestations of COPD arising from exposure to tobacco smoke are of a different nature. A typical manifestation is the development of emphysema, the so-called emphysematous type of COPD. Phenotypically, it is characterized by underweight, muscle hypotrophy, pronounced hyperinflation of lung tissue. The second phenotype is clinically manifested by cough, increased sputum production, the development of diffuse cyanosis; in this category of patients, signs of decompensated pulmonary heart develop relatively early. This COPD phenotype is designated as bronchitic.

One of the rare forms of pulmonary pathology is eosinophilic granuloma; other designations of this pathology are: pulmonary granulomatosis by Langerhans cells, pulmonary histiocytosis X (the latter term is used most often). The nature of the disease remains not fully understood, however, it is emphasized that smoking is a risk factor in the development of granulomatous process in the lungs. The proof is the development of bronchiolitis, macrophages containing characteristic inclusions of tobacco smoke elements in phagolysosomes (tobacco smoke markers) migrate into the walls of the terminal respiratory tract. The disease affects people at a young age, is characterized by repeated pneumothorax and increasing shortness of breath. The diagnosis is confirmed after image diagnostics, but for the final diagnosis it is necessary to conduct a lung biopsy, which allows identifying characteristic morphological changes.

Thus, modern scientific research on the mechanisms of the damaging effect of tobacco smoke on the human body has allowed us to identify three most important areas. Tobacco smoking is a factor associated with the process of biological aging of cells of the human body. Tobacco smoke disrupts the balance in the oxidants–antioxidants system and leads to the formation of oxidative stress. High production of reactive oxygen species, as well as certain chemical compounds (aldehydes and others) induce a chronic inflammatory process of the respiratory tract. Finally, the complex chemical composition of tobacco smoke has pronounced genotoxic properties. The body's response to the damaging effect of tobacco smoke largely depends on the expression of genes of the epithelial cover of the respiratory tract both in the formation of defense mechanisms and in the process of oncogen amplification.

The clinical forms of respiratory damage in tobacco smoking are very diverse. The most common pathology is the development of chronic obstructive pulmonary disease. It should be noted that phenotypically the clinical forms of COPD vary: emphysematous type, bronchitis, bronchiolitis of a smoker.

Pulmonary Langerhans cell histiocytosis



The cellular infiltrate of pulmonary Langerhans cell histiocytosis is characterized by Langerhans cells, eosinophils, lymphocytes, neutrophils, and alveolar macrophages. *From Colby, TV, Koss, MN, Travis, WD. Tumors of the Lower Respiratory Tract. Armed Forces Institute of Pathology, Washington, DC.*

Inhaling smoke is accompanied by vasoconstriction, an increase in blood pressure and the release of the hormones adrenaline and norepinephrine. The consequence of this is a violation of blood circulation, which leads to a deterioration in the nutrition of all organs and tissues – as a result, the cardiovascular and respiratory systems suffer.The changes that occur with the respiratory system under the influence of tobacco smoke are steadily progressing.

In total, a cigarette contains several thousand toxic compounds that destroy the body with each puff. A person may feel the effects of harmful substances as compression in the sternum. This feeling is a manifestation of oxygen deficiency and an increase in carbon monoxide levels.

Not only active, but also passive smoking is dangerous. Tobacco addiction during pregnancy leads to an increased risk of developing bronchial asthma in a child. Smoking is also associated with respiratory disorders during sleep, which increase the risk of sudden death syndrome. This is due to the effect of nicotine on the nervous system and, in particular, on the respiratory center.

The lungs are directly exposed to oxygen, an oxidation inducer, as well as other oxidants contained in polluted air (ozone, nitrogen and sulfur dioxide). In addition, lung tissue contains unsaturated fatty acids, which are also a substrate for lipid peroxidation.

Under the influence of smoking, the number of neutrophils increases tenfold. Neutrophils secrete various proteases, including neutrophilic elastase, neutrophilic proteinase-3, neutrophil cathepsin, matrix metalloproteinases that destroy their own elastic backbone of the lungs, which leads to the destruction of the lung parenchyma. On the other hand, neutrophil elastase causes mucus secretion and hypertrophy of the mucous glands. Neutrophil proteinase-3 can also contribute to the induction of increased bronchial secretion production.

On the one hand, protease activity increases, and on the other hand, antiprotease protection decreases. Under the influence of tobacco smoke in smokers with initially normal levels of a1-antitrypsin, its activity decreases. As a result of chronic exposure to tobacco smoke oxidants, methionine oxidation is disrupted, which in turn leads to the inactivation of a1-antitrypsin.

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