



SMOKING-RELATED LUNG DISEASES

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Abstract: The compounds in cigarettes are harmful to all living organisms. We have all heard that 1 gram of nicotine can kill a horse. Besides the clear health risks, the positive effects of smoking are minimal. There is an occurrence where nicotine has a beneficial effect on the intestinal mucosa. Through circulation, the substance reaches an area that can protect against ulcerative colitis. Nicotine found in cigarette smoke has anti-inflammatory properties. The effect of smoking on blood vessels is negative, but it helps to balance neurotransmitters. This reduces the likelihood of the onset and progression of Alzheimer's and Parkinson's diseases. The process of smoking constricts blood vessels, which enhances mental abilities and improves memory, but still negatively affects the heart.

Keywords: Lung diseases, Smoking complications, Cigarette smoke, Genetic and exogenous factors.

Smoking-related lung diseases encompass various lung conditions and injuries where the etiological risk factor is tobacco smoking. Tobacco smoke contains over 7,000 different chemicals, which can have direct and indirect toxic effects, triggering immune responses. Together, these can lead to irreversible damage to the airways and lung parenchyma. At least 250 of these substances are known to be toxic or carcinogenic. Smoking can cause lung cancer and chronic obstructive pulmonary disease (COPD), as well as a range of interstitial lung diseases, acute eosinophilic pneumonia, desquamative interstitial pneumonia, and respiratory bronchiolitis associated with interstitial lung disease.

The most common causes of death from smoking are lung cancer and COPD. All smokers experience inflammatory changes in their airways, with smokers always showing a histological form of respiratory bronchiolitis, though some smokers may develop changes that lead to diseases. The development of these diseases is influenced by genetic and exogenous factors, as well as allergies and infections. Many smoking-related lung diseases usually have similar pathological processes, and pathologists often find combinations of different histological forms of inflammation, making any definitive diagnosis challenging. Smoking damages the mucous membranes of the airways, alveolar sacs, and airways, eventually leading to difficulty in providing sufficient air to the lungs.









Smoking increases the likelihood of developing lung cancer by about 22 times compared to non-smokers and COPD is 3-4 times more common among smokers than non-smokers. When inhaled, tobacco smoke first enters the mouth, then passes through the upper respiratory tract, enters the lower respiratory tract, and finally reaches the alveoli. As we inhale deeper into the respiratory tract, more soluble gases are adsorbed, and the particles entering the lungs settle in the airways and alveoli. As a result, significant doses of carcinogens and toxins enter the lungs.

The dose of toxic particles and gases entering the respiratory tract varies depending on the tobacco, the volume of inhalation, the number of puffs per cigarette. and the particle size. Large particles are expelled from the inhaled air through the upper respiratory tract, while small particles less than 2.5 microns in diameter reach and settle in the lungs. The average diameter of the particles inhaled with cigarette smoke is 0.3-0.4 microns - over 60% of the particles inhaled with tobacco smoke settle in the lungs. Mucosal apparatuses and alveolar macrophages should remove the particles that enter the lungs, but not all of them are removed due to their large quantity in a long-term smoker's lungs. Cigarette smoke is a strong trigger of inflammation, mainly marked by an increase in the number of macrophages recruited and activated by cigarette smoke. Macrophages, in turn, attract other immune cells - neutrophils, monocytes, eosinophils, and lymphocytes - to the lungs. The branches of Langerhans cells, a type of dendritic cell, form on the surface of the airways, and these cells proliferate upon exposure to cigarette smoke, enhancing inflammation and causing airway dysfunction. Consequently, emphysema, peribronchial and alveolar wall damage, fibrosis, and hardening develop, leading to breathing difficulties and bronchial diseases.

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