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ANALYSIS AND RESULTS OF MORPHOLOGICAL CHANGES IN THE LUNGS OF SMOKERS...

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Abstract. Smoking, a widespread habit with a serious impact on health, remains a significant public health concern. Despite growing awareness of the risks associated with smoking, many individuals continue to succumb to this addiction. Understanding the facts about smokers and smoking habits can shed light on the challenges faced in addressing this global issue.

In 2022, in collaboration with several research institutes of the Ministry of Agriculture, a study was conducted on the problem of nasvay and cigarettes. It is estimated that there are 4.5 million smokers in Uzbekistan. More than half of them, namely 2.4 million people, smoke cigarettes. The rest (2.1 million people) use nasvay. Smoking is a habit that has numerous harmful effects on human health. Smoking can lead to a multitude of short-term and long-term health problems, especially with regular use. Smoking harms the respiratory system, causes chronic obstructive pulmonary disease (COPD), and increases the risk of respiratory infections.
Key words: smoking, nicotine effects, alveoli, microscopic, lung tissue.
Калит сузлар: чекиш, никотин таъсири, алвеолалар, микроскопик, упка тукимаси.
Ключевые слова: курение, действие никотина, алвеолы,микроскопические, ткань легких.

Introduction: Smoking is a major risk factor for many health problems, including lung cancer, heart disease, respiratory diseases, and stroke.

Smoking can lead to serious lung diseases such as chronic obstructive pulmonary disease (COPD), bronchitis, and emphysema. These diseases are accompanied by symptoms such as shortness of breath, cough, phlegm, and respiratory infections. Smoking can cause various types of cancer, including lung, larynx, esophageal, pancreatic, and bladder cancer. Smoking increases the risk of developing cancer and can lead to death.

COPD is a progressive disease characterized by chronic inflammation of the airways with impaired normal airflow at the level of the distal bronchi and structural changes in lung tissue and vasculature. Today, three main etiological factors for this nosology are identified: smoking, occupational exposure to the respiratory system, and genetic predisposition.

Objective: Analysis and results of morphological changes in the lungs that develop in smokers...

Materials and Methods; For this study, research was conducted using archival materials from the Tashkent City Research Center of Forensic Medicine. Specifically, lung tissue samples from 85 male smokers (aged 25-50 years) who died between 2024 and 2025 were morphologically compared and studied. In COPD, pathological changes are found in all elements of the bronchial tree and exhibit varying spectra and severity. **Macroscopically:** In COPD, the bronchial mucosa develops characteristic enhanced folding, and lumen deformation is noted, characterized by focal constrictions and dilations. In addition to typical mucus deposits, papillary formations composed of connective tissue may be found within the lumen. Microscopic examination of the bronchial mucosa reveals hyperplasia of goblet cells and

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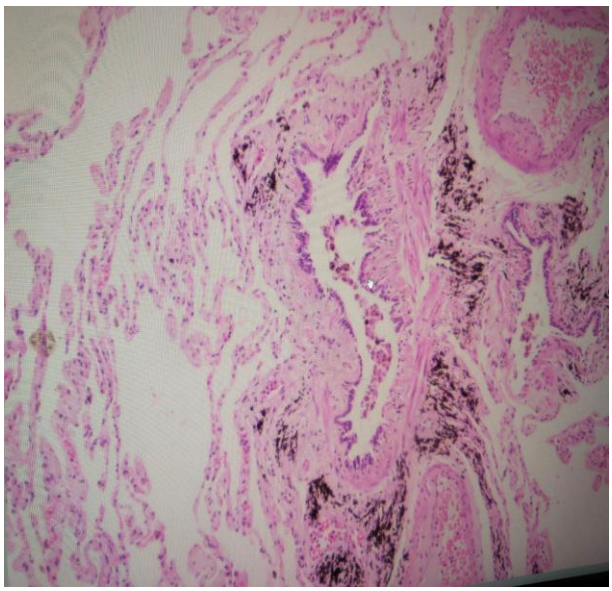
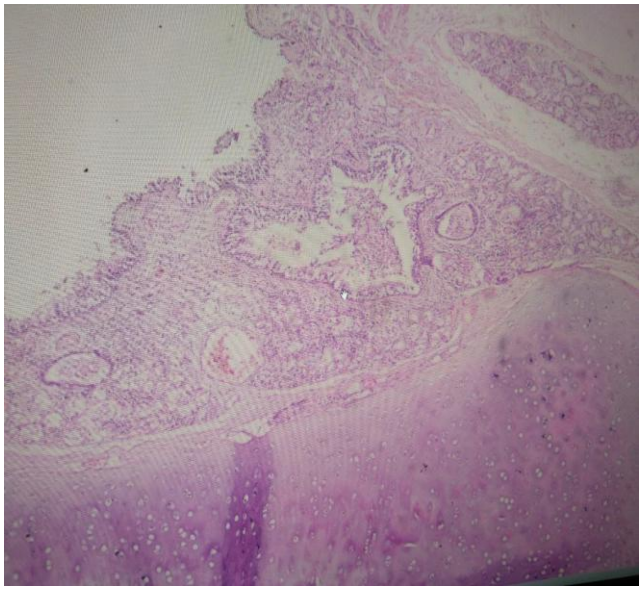
accumulation of PAS-positive granules in the cytoplasm of bronchial epithelial lining cells. In some areas, the epithelium undergoes focal squamous metaplasia. With the addition of acute purulent inflammation, the bronchial mucosal epithelium is intensely desquamated, and a large number of mucus plugs may be found in the lumen. The propria submucosa of the bronchial mucosa undergoes thickening and diffuse infiltration by lymphocytes. In some areas of the bronchial wall, the infiltrate can be so massive that the visual boundary between the epithelium and the muscularis mucosa is lost. Characteristic morphological changes in COPD caused by smoking include neoangiogenesis, thickening, and fragmentation of the mucosal basement membrane.





The severity of these changes directly depends on the person's smoking history. Bronchial glands typically undergo hypertrophy and hyperplasia. Initial changes in the wall of cartilaginous bronchi in the development of COPD involve an enhanced PAS reaction during

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staining of fibrous structures and their thickening. The bronchial wall thickness increases due to edema and neutrophilic-lymphocytic infiltration of the stroma. In the long-term course of the pathological process, there is complete atrophy of the muscularis mucosae with its replacement by connective tissue elements, a decrease in the thickness of the fibrocartilaginous layer of the bronchi, and partial destruction of its wall with the formation of an air cavity. In some cases, edema in the wall of bronchi and bronchioles can be so pronounced that it leads to their significant thickening, destruction, separation of collagen and elastic fibers, and detachment of the basement membrane along with the underlying epithelium. Bronchioles undergo identical changes.

Another zone consists of areas of emphysematous lung tissue expansion, occurring distal to sclerosed and narrowed terminal bronchioles. In this case, changes in the alveolar walls are caused by the fragmentation of intramural elastic fibers. Due to the direct contact of alveoli with adjacent respiratory bronchioles, the stretching of the former leads to a decrease in the tone of smooth muscle cells and an increase in the volume of the latter. A conditionally specific factor in lung tissue damage in COPD, primarily caused by tobacco smoke exposure, is associated with the accumulation of tar elements in alveolar macrophages. The phagolysosomal enzymes of phagocytes are unable to fully lyse the substances that make up tar granules, leading to imperfect phagocytosis. Over time, phagocytes undergo destruction, and the tobacco tar granules are phagocytosed without any changes by another macrophage, after which the process can be repeated multiple times. In this process, enzymes contained in the phagolysosomes of dead macrophages act on surrounding structures, causing their destruction, which ultimately accelerates the progression of COPD.Alveoli, in the case of acute purulent inflammation, undergo focal emphysematous distension and infiltration by neutrophils and lymphocytes. However, acute purulent inflammation in COPD differs from a similar process in intact lungs by the rapid spread of inflammation deep into the walls of bronchi, bronchioles, and alveoli, a tendency to form numerous areas of post-inflammatory pneumosclerosis, and the composition of the leukocytic infiltrate – in addition to neutrophils, lymphocytes constitute its main part. Air cavities are the most frequent morphological finding in the lungs in COPD.Currently, three morphological types of COPD are distinguished: 1) bronchointerstitial type, characterized by a pronounced combination of sclerotic changes in cartilaginous bronchi and lung tissue with the formation of obstructive emphysema; 2) emphysematous type, where obstruction arises from the collapse of highly sclerosed bronchioles without a corresponding level of damage to cartilaginous bronchi; 3) truly obstructive type, where the development of bronchiolitis and bronchiole collapse occur without significant sclerosis of their walls.

Conclusion: The study was conducted based on archival materials from the Scientific Center of Forensic Medical Examination of Tashkent. Specifically, during a comparative morphological study of lung tissue samples from 85 men (aged 25-50) who died in 2024-2025 and were smokers, it was established that in the majority of them, the bronchial epithelium underwent metaplasia, forming precancerous morphological changes.

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