Morphological Bases of Adaptation Respiratory Departments of Lungs at an Elevated Temperature and Hypoxia.

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ABSTRACT

This article provides a basis ulstrukturnye adaptation respiratory department of lungs in conditions of high temperature and hypoxia.

Keywords: hypoxia, pinocytic bubbles osmiophil calf fugalizosomy, surfactant.

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Introduction

It is known that exposure to intense heat leads to the development of hyperthermia with structural changes in the lungs [1-4] at the same time morphological works devoted to the study of long-term targeted kombinorovannogo influence of high temperature and hypoxia on the structural and functional components of the lung using transmission and scanning methods eletronomikroskopicheskogo study and identify mechanisms of adaptation, in the available literature, we have not met. Objective-examine by transmission and scanning microscopy email character of ultrastructural changes in the respiratory department of lungs in conditions of high temperature and hypoxia. Material and methods. The experiments were carried out on 15 adult male rats, 5 of them were controlled. Animal hour for 30 days in a thermostat kept at a temperature of 360 °C, a relative humidity of 35% and in case of insufficient ventilation. For transmission electron microscopic study of lung tissue pieces were fixed in 2.5% glutaraldehyde solution at Millonga phosphate buffer (pH 7.4-7.6) for 2.5 hours with postfiksatsiey for 2 hours in a 1% solution of Os O4, then the pieces of tissue were performed through a series of ethanol solutions of ascending concentrations of absolute acetone and embedded in epon-812. Ultrathin sections were contrasted with uranyl acetate and lead citrate according to Reynolds. The study and survey of ultrathin sections were performed on the computer 100L electron microscope at an accelerating voltage 75kV. For scanning electron microscopy, pieces of lung size 5h3h3mm digidrotirovali alcohols of increasing strength, a mixture of alcohol - acetone. Drying of the material was carried out at the critical point of the liquid CO2. After the deposition of gold samples examined in electron probe microanalyzer in the raster mode, the instrument research Superprobe 733. The samples were photographed with an increase of 800 X-H4000. Transmission electron microscopic examination revealed that the majority alveolocytes type I was in a state of severe labor hypertrophy. The amount of cells increased. Large hyperchromic nucleus possessed scalloped jagged kontrollers nuclear envelope. Geterohromatin distributed primarginalno. Perinuclear protransvso been extended and is often reported with the lumen of the endoplasmic reticulum tubules granular. Located on the periphery of the granular endoplasmic reticulum canals were enlarged by taking an irregular shape. Reticulum lumen was filled flaky sodererzhimy moderate density email. Membranes were provided many fixed ribosomes. In the cytoplasm there is a large, poorly razlechimy on a dark background, mitochondria Matrix moderate elektronny potnosti and partly vacuolated mezhrkristnymi intervals. Dark background cytoplasm was due to a high content of free ribosomes and fine fibrillar structures. The apical surface was provided with numerous thin, long, often branching cytoplasmic processes that shape the whole plexus (Figure 1). they contributed to the formation of large vacuoles. In the interior processes and on the periphery of the cytoplasm has numerous small pinocytic bubbles form the mouth of which points to the disclosure of their cytoplasm and towards the interstitial.

Figure 1: TEM. Increased temperature and hypoxia. Alveolotsit I type with marked hypertrophy of the core (I) and long cytoflazmatic processes (CO). Uv.H 12500

Peripheral departments alveolocytes type I also showed increased cytoplasm completely electronic. Uneven "fringed" surface tonekimi cytoplasmic outgrowths contributed to the formation of large vacuoles. Closer to perikaryon were seen numerous small penotsitzonye puzyrki.Kletki located on a thickened basement.
membrane and loosened. Single alveolar type I cells in a state of severe hydropic degeneration and partial necrosis. Under the pathological changes of cells "creeping" processes reginiruyuschego thin alveolar epithelium. Alveolocytes type II were in working hypertrophy. They contained the core of irregular shape with primarginalnym distribution of heterochromatin and expanded perinuclear space. Number of granular endoplasmic reticulum tubules was increased. They formed a dense network of extended space filled flake content of moderate electron density. The number and size of mitochondria also increased. They had hung the electron density matrix, often located Kristen mezhrkristnye intervals selected were vacuolated. In the cytoplasm, sharply increased the number of osmiophil plate cells (Figure 2).

![TEM Image](image)

Figure 2. TEM. Increased temperature and hypoxia. Alveolotsit type II. Large osmiophil lamellar bodies (OPT). Uv.H 21000

It should be noted their polymorphism and the presence of small cells with dense homogeneous elements near the center of the Golgi complex. Large osmiophil calf plate located near the plasma membrane that contains an ordered lamellar structure is formed of surfactant. In the cytoplasm of hyperplastic tank also has the Golgi complex, numerous free ribosomes. The apical surface was sklazhena isolated and contained cytoplasmic processes. With deep invaginations into the cytoplasm plunged large vacuoles.

Occasionally it was marked alveolocytes small undifferentiated type II with initial signs of differentiation.

Endotelial vystikla pulmonary capillaries was in sleeping or expanded state. It was characterized by thickening of the walls of capillaries, and enhanced mikropinotsitozom vakuoleformation. Vacuole contributed to numerous irregularities and cytoplasmic apical surface microvessel outgrowths. In the cytoplasm hung electron density have large mitochondria condcenated type slightly expanded tubules granular endoplasmic reticulum with abundant ribosomes and fixed flake material in the lumen.

In the interstitial space there are signs of fibrosis. Kolagennovye fibrils were collected in bundles of various orientations and were characterized by a clear cross-ischerechennostyu. An interesting feature was the presence of myofibroblasts and playing an important role of the contraction of the alveolar lung linings and fibroklastov carrying resorption "excessive" collagen. In the cytoplasm fibroklastov housed numerous small primary lysosomes.

The ultrastructure of alveolar macrophages was characterized moderate phagocytic reaction. In the cytoplasm, located osmiofil small primary lysosomes, elektronno transperancy large vacuoles, single phagolysozomes with fagotsiterated plate material. Absorbing phase involved the foreign particle attachment to the cytoplasmic outgrowths macrophage.
Scanning Electron microscopic study of the respiratory surface of the lungs showed that when exposed to elevated temperature and hypoxia observed increase over the alveolar surface roughness control, reflecting the functional state of the polymorphism of pulmonary capillaries and vacuolization alveolocytes apical surface of type I (Figure 3). alveolocytes type II were in the active secretion of alveolar surfactant.

Figure 3: SEM. Increased temperature and hypoxia. The roughness of the alveolar surface. Uv. X 940

Figure 4: SEM. Felt-like structure of the surfactant (SF) in the respiratory department of lungs. Increased temperature and hypoxia. Uv. X 1600

Type II was covered with numerous microvilli, central part occupied by nodules and convexity, see fit sekpetsii alveolarnogo surfactant (Figure 4). however, we noticed a lack of alveolar surfactant “washout” it from the alveoli during hyperventilation. The process of hyperventilation showed “deep” the pores of Kohn.

CONCLUSION

The electron microscopic studies we have identified the development of compensatory adaptive changes expressed by all cellular elements resperimentalnyh animals in response to exogenous high temperature and hypoxia. Only a few alveolocytes type I is the most sensitive in a state of hydropic
degeneration and partial necrosis. However, in this case collapsing epitelalnyh rejection of cells from the basement membrane accompanied by reduction lining crawling flattened portion adjacent cells. As alveolocytes of I and type II were in a state of hypertrophy with increased labor and tissue-specific secretory synthesis. Alveolocytes appearance of type II with initial signs of a specific differentiation svidetlstvovalo their proliferation. Alveolyarny surfactant complex for individual iskyucheniem neraskruchenyny lamellar bodies was absent. A paradox between enhanced functional activity alveolocytes type II and apparent lack of surfactant, we like, and (2) relating to the destruction and the "washout" of alveol surfactant during hyperventilation and increased convection teploodachi. Close contact myofibroblasts spaverhnostyu epithelial cells was probably aimed at optimum stabilization of the changing surface alveol. Sharp uselenie micro pinocytosis with thickening of the endothelial layer of aero-hematic memrannoy system prevents the development of hydration epitelyalnogo and interstitial layers of aero-gemotitseskogo barrier. Fibrollo-educational function of fibroblasts was replaced by active fibroklaziey redundant fibrils kollogena. Thus the ultra-structural changes in the lungs indicate the important role of hypoxia in this experimentation.

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