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## FEATURES OF POSTNATAL DEVELOPMENT OF THE LIVER OF THE OFFSPRING UNDER CHRONIC EXPOSURE TO AIR POLLUTANTS ON THE MOTHER'S BODY

Азизова Феруза Хусановна

доктор медицинских наук, профессор кафедры гистологии и медицинской биологии Ташкентский государственный медицинский университет, Ташкент, Узбекистан

Абдусаломов Гайрат Абдусаломович

Ташкентский государственный медицинский университет, Чирчикский филиал, Ташкент, Узбекистан

Нурматов Бахриддин Қўзибойевич

PhD. Ташкентский государственный медицинский университет, Чирчикский филиал

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### Abstract

The study presents the results of an experimental investigation of the effects of chronic exposure to atmospheric air pollutants on the postnatal liver development of offspring. Morphological, immunohistochemical, and morphometric changes were identified, characterized by decreased hepatocyte proliferation, increased apoptosis, disruption of sinusoidal architecture, and early activation of fibrogenesis. The obtained data indicate the formation of prerequisites for metabolic disorders in later stages of life.

**Keywords:** Air pollution, liver, offspring, postnatal development, proliferation, apoptosis, fibrosis, oxidative stress.



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## **Introduction**

Atmospheric air pollution is a significant risk factor for the development of chronic diseases. Fine particulate matter (PM<sub>2.5</sub> and PM<sub>10</sub>), nitrogen oxides, sulfur dioxide, and volatile organic compounds are capable of crossing the placental barrier and affecting developing fetal organs. The liver, as the central organ of detoxification and metabolism, is particularly sensitive to toxic exposure during critical periods of development.

## **Aim of the Study**

To investigate morphological and immunohistochemical changes in the liver of offspring during the postnatal period under chronic exposure of the maternal organism to polluted air.

## **Materials and Methods**

The experiment was conducted on 40 female Wistar rats and 120 offspring. Chronic exposure was modeled for two months prior to pregnancy and throughout gestation. The offspring were examined on postnatal days 1, 7, 14, 21, and 30. Histological staining methods (H&E, Masson's trichrome, Van Gieson) were applied. Immunohistochemical markers included Ki-67, Caspase-3, CD68, and  $\alpha$ -SMA. Biochemical determination of IL-6 and TNF- $\alpha$  levels was performed. Statistical analysis was conducted using Student's t-test with significance set at  $p < 0.05$ .

## **Results**

In the experimental group, a significant decrease in the Ki-67 index by 45–60% was observed, indicating suppression of hepatocyte proliferative activity. The number of Caspase-3-positive cells increased 1.8-fold compared to the control group.

Sinusoidal dilation, thinning of the endothelial layer, and disorganization of hepatic cords were noted. Increased expression of  $\alpha$ -SMA indicated early activation of fibroblasts and initiation of fibrotic processes.



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## Discussion

The findings confirm the role of oxidative stress and pro-inflammatory cytokines in the pathogenesis of the observed changes. Elevated levels of IL-6 and TNF- $\alpha$  contribute to the maintenance of chronic inflammation and liver tissue remodeling. Activation of the TGF- $\beta$  signaling pathway triggers fibrogenesis.

Thus, chronic exposure to air pollutants during gestation forms structural and functional prerequisites for the development of metabolic disorders later in life.

## Conclusion

Chronic exposure of the maternal organism to polluted air leads to impaired postnatal liver development in offspring, characterized by reduced proliferation, increased apoptosis, and early activation of fibrotic processes.

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