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GROWTH AND FORMATION OF THE THYROID GLAND IN OFFSPRING OBTAINED UNDER CONDITIONS OF MATERNAL HYPOTYROIDISM

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Relevance

In recent years, it has been found that small doses of pesticides can have effects that are completely unpredictable when using large doses. In this case, toxic effects proceed secretly, without certain clinical manifestations, which makes it extremely difficult to carry out early diagnosis, effective prevention and treatment of chronic intoxication with small doses of pesticides.

This is explained by the fact that small doses of pesticides, first of all, affect the regulatory systems of the body, which are the endocrine and nervous systems. The endocrine system of a developing organism is one of the most sensitive to the action of various environmental pollutants, including pesticides. The term “endocrine-disrupting chemicals” (EDC) has long been firmly established in the literature, implying their adverse effect on various stages of the synthesis, transport and function of hormones on target cells.

It should be noted that the endocrine-destroying effect of pesticides of new generations has been revealed relatively recently and there are relatively few reports on this. Thyroid hormones play an important role in the embryonic and postembryonic development of organs and tissues. Their deficiency can lead to significant changes in the organs and systems of the fetus and offspring. However, the mechanisms of the adverse effect of maternal hypothyroidism of pregnancy on the pre- and postnatal growth of organs and systems of the offspring remain largely unexplained.

The purpose of the work is to identify the features of the postnatal development of the thyroid gland of offspring obtained under the influence of pesticides through the mother's body.

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Material and Methods

The experiments were carried out on the offspring of female rats obtained from healthy females, as well as females exposed to the widely used pyrethroid pesticides fipronil (FP) and fastokin (FC) during pregnancy and lactation. The thyroid gland (TG) of rat pups was studied on days 3, 7, 14, 21, 30, and 90 after birth. Morphological, morphometric, immunohistochemical, electron microscopic and statistical research methods were used.

Research results

It has been established that exposure to pesticides during pregnancy and lactation contributes to the development of maternal hypothyroidism, which leads to disruption of the process of postnatal growth and formation of the thyroid gland, which manifests itself as secondary hypothyroidism in offspring. The impact of pesticides on offspring through the mother's body. Moreover, the slowdown in the rate of formation of the thyroid gland was more pronounced with FA intoxication compared with the effect of fipronil (FP).

Electron microscopically revealed high functional activity of macrophages and destructive changes in subcellular organelles of lymphoid cells. It has been established that exposure to pesticides leads to inhibition of the proliferative activity of thyrocytes. At the same time, intrauterine and early postnatal exposure to pesticides led to a significant increase in the degree of cell apoptosis in organs.

Conclusion

Thus, maternal hypothyroidism plays a leading role in the pathogenesis of disorders in postnatal development of organs under exposure to pesticides, which contributes to the development of secondary hypothyroidism in offspring. Early detection of hypothyroidism in pregnant women and newborns, and their timely pharmacological correction will help prevent or reduce the negative effects of pesticide exposure on the younger generation.

Chronic intoxication of the mother's body with pesticides leads to a significant slowdown in the rate of postnatal growth and the formation of secretory follicles of the thyroid offspring. Ultrastructural changes in the form of disorganization of the granular endoplasmic network and destruction of other cytoplasmic organelles of thyrocytes are a morphological substrate of thyroid dysfunction that occurs in postnatal ontogenesis of the organ in conditions of chronic intoxication.

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