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# DESTRUCTIVE EFFECTS OF PESTICIDES ON THE ENDOCRINE SYSTEM OF OFFSPRING UNDER THE CONDITIONS OF THEIR EXPOSURE THROUGH THE MOTHER'S BODY DURING PREGNANCY AND LACTATION

**Islomova Shoxista Abdixalilovna**

https://orcid.org/0009-0008-9498-9476

Tashkent Medical Academy

## Annptation

The aim of the study was to identify the toxic effect of intrauterine and early postnatal exposure to pesticides on the development of the endocrine system of offspring. The thyroid gland of the offspring obtained from mothers with chronic poisoning with pesticides vigor and fastkin was examined on 1-30 days after birth using electron microscopic methods. It was revealed that intoxication of the mother's body significantly slows down the pace of postnatal formation of secretory follicles. Ultrastructural changes were found in the form of disorganization of the granular endoplasmic network and destruction of other cytoplasmic organelles of thyrocytes. These changes are a morphological substrate of thyroid dysfunction that occurs in the postnatal ontogenesis of the organ in conditions of chronic intoxication.

## Key words

Pesticides, thyroid gland, chronic intoxication.

**Introduction.** Pesticides are one of the most common environmental pollutants. The widespread use of pesticides in agriculture, in everyday life and public health causes their inevitable ingestion, albeit in small quantities, into the human body and animals. Of the pesticides allowed for use in the republic, more than half belong to the groups of pyrethroid and pyrazole preparations. Despite the relatively low toxicity of these compounds, the probability of their negative effects on the body remains very high. In this regard, pesticides pose the greatest danger to pregnant women and their offspring due to the high sensitivity of the developing endocrine systems of the fetus and newborn to toxic effects. It has been shown that the introduction of even small doses of pesticides into the body of pregnant and breast-feeding females negatively affects the state of the thyroid gland of their offspring [3; 4; 5]. Exposure to small doses of pesticides can negatively affect the morbidity of the population as a whole, and especially on the health indicators of women and children [9]. In this regard, the problem of studying the mechanisms of the toxic effect of pesticides, the search for means and methods for detecting preclinical negative changes in the human body or their offspring that occur under the influence of small doses of pesticides is currently very relevant.

Materials and methods of research.

Experiments were conducted on white adult virgin female Wistar rats weighing 150-170 g, and mature male rats were used only for fertilization. Then the female rats were divided into three groups of 45 rats each. The first (experiment 1) group of rats was injected with fastokin through the mouth using a probe at the rate of 8 mg/kg/ daily. The second (experiment 2) group of rats was similarly injected with FP at the rate of 3.6 mg/ kg / daily, which corresponded to 1/100 of the LD50 of the

VOLUME 7, ISSUE 1, 2023 SPECIAL ISSUE

drugs. The third group of rats, which received the same volume of sterile saline solution, served as a control. The administration of both pesticides to experimental groups of rats was carried out daily for 75 days until the end of the experiments. On the 31st day of the experiments, females of all groups were joined with males for fertilization. The onset of pregnancy was controlled by the presence of sperm in vaginal smears. After pregnancy, the females are separated from the males and placed in separate cells for further research. Offspring from all groups of animals were killed on the 7th, 14th, 21st and 30th days after birth, under mild anesthesia with ether. After killing, the concentrations of thyroxine (T4), triiodothyronine (T3) and thyrotropic hormone (TSH) in the blood serum were determined by immuno-enzyme analysis using special kits of the company "Human" (Germany) and the spectrophotometer "Singl" (Germany). The thyroid gland (thyroid gland) and thymus gland (thymus, Tm) were studied using morphometric, immunohistochemical and electron microscopic methods. All digital data were statistically processed using a computer software package, differences satisfying $P<0.05$ were considered reliable.

Results and their discussion. Exposure to pesticides through the mother's body during pregnancy and lactation significantly reduced the growth rate and formation of thyroid in offspring [1; 2]. Our morphometric studies have shown that the growth rate of the total area of the follicle epithelium under the influence of fastokine is 10-17%, and when exposed to AFN - 15-30% lags behind the control parameters. However, the negative effect of pesticides was not limited only to inhibition of follicle formation. It was found that exposure to pesticides leads to a decrease in the area of the follicle as a whole due to a decrease in the areas of the follicle epithelium and thyrocyte. The slowing of growth and formation of the thyroid gland was accompanied by a significant decrease in the functional activity of the organ. Despite the high level of thyroid-stimulating hormone (TSH), the concentration of free thyroxine (T4), triiodothyronine (T3) remained significantly lower compared to the control. The most pronounced hypothyroidism was observed in offspring under the influence of AFN compared to LST. Electron microscopy revealed a decrease in the size of mitochondria, as well as components of the endoplasmic network and the Golgi complex of thyrocytes, which indicated a decrease in the secretory activity of cells.

The data obtained show that the toxic effect of pesticides on the development of the endocrine and immune systems of offspring is due to a number of metabolic changes in organs and tissues. First of all, it should be noted the endocrine-destroying, more precisely, thyroid-destroying effect of pesticides, which leads to hypothyroidism in the mother and offspring [4; 5]. In addition, it is necessary to emphasize the role of oxidative stress as the main inducer of cell apoptosis [6; 7]. Hence, it can be concluded that the induction of apoptosis in offspring is due not only, and not so much to the direct toxic effect of pesticides. The increase in apoptosis is largely mediated by the weakening of the anti-apoptotic function of thyroid hormones due to hypothyroidism, as well as an increase in the number of free radicals as a result of oxidative stress [8].

## Conclusions

1. The toxic effect of exposure to pesticides through the mother's body during pregnancy and lactation manifests itself in the form of slowing down the growth and formation of the thyroid gland of the offspring.

VOLUME 7, ISSUE 1, 2023 SPECIAL ISSUE

2. In the mechanism of the toxic effect of pesticides on the endocrine system, an imbalance between cell proliferation and apoptosis plays a leading role, caused by both the direct toxic effect of drugs and developing hypothyroidism and oxidative stress in the mother and offspring.

3. This necessitates the early detection of hypothyroidism and oxidative stress in pregnant women and newborns, followed by their pharmacological correction.

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VOLUME 7, ISSUE 1, 2023 SPECIAL ISSUE

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