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ENDOCRINE-DISRUPTING AND IMMUNOTOXIC EFFECTS OF PESTICIDES ON OFFSPRING UNDER CONDITIONS OF EXPOSURE THROUGH THE MATERNAL ORGANISM

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Abstract

This study investigated endocrine-disrupting and immunotoxic alterations occurring in offspring as a result of pesticide exposure through the maternal organism during pregnancy and lactation. Experiments were conducted on Wistar rats to evaluate the effects of the pesticides lambda-cyhalothrin and fipronil on the development of immune and endocrine system organs. Morphological, immunohistochemical, electron microscopic, and biochemical methods were used to assess changes in the thymus, lymph nodes, spleen, and thyroid gland. The results demonstrated that pesticide exposure leads to an imbalance between cellular proliferation and apoptosis, thereby promoting the development of hypothyroidism and oxidative stress. These pathological processes contribute to impaired development and maturation of immune organs and the thyroid gland in offspring.

Keywords: pesticides, immunotoxicity, endocrine disruption, thyroid gland, thymus, hypothyroidism, oxidative stress.

Introduction

In recent years, environmental pollution and the extensive use of pesticides have become significant public health concerns. Certain pesticides possess immunotoxic and endocrine-disrupting properties, adversely affecting both the hormonal and immune systems of living organisms.



The fetus and newborn are particularly sensitive to toxic environmental factors. Exposure to pesticides through the maternal organism during pregnancy and lactation may interfere with the normal development of the endocrine and immune systems. Consequently, such exposure can increase the risk of chronic diseases, immune dysfunction, and metabolic disorders later in life.

The aim of this study was to identify the structural and functional mechanisms underlying the immunotoxic and endocrine-disrupting effects of

pesticides on offspring exposed through the maternal organism.

Materials and Methods

The study was carried out on female Wistar rats. The animals were divided into three groups:

- **Experimental Group I** – received lambda-cyhalothrin;
- **Experimental Group II** – received fipronil;
- **Control Group** – received physiological saline.

The pesticides were administered orally throughout pregnancy and lactation. Biological samples from offspring were collected on postnatal days 3, 7, 14, 21, and 30.

The thymus, mesenteric lymph nodes, spleen, and thyroid gland were examined using morphometric, electron microscopic, and immunohistochemical techniques. Serum concentrations of T3, T4, and TSH hormones, as well as markers of oxidative stress, were also determined.

Results and Discussion

The findings demonstrated that pesticide exposure significantly impaired the development of the immune and endocrine organs in offspring. The growth rates of the thymus, lymph nodes, and spleen were markedly lower compared with those observed in the control group.

Immunohistochemical analyses revealed a decrease in the proliferation index within T-lymphocyte-rich regions. At the same time, apoptosis levels increased by approximately 3–5 times. These alterations were more pronounced in the group exposed to fipronil.

In the thyroid gland, delayed follicular development, reduced thyrocyte area, and diminished hormone-producing activity were observed. Decreased levels of T3 and T4, accompanied by elevated TSH concentrations, indicated the development of hypothyroidism.

Electron microscopic examination demonstrated degenerative changes in cellular organelles, including reduced mitochondrial size and diminished activity of the endoplasmic reticulum and Golgi apparatus.

The obtained results indicate that the toxic effects of pesticides are mediated not only through direct cytotoxic mechanisms but also through the induction of hypothyroidism and oxidative stress.

Conclusion

1. Exposure to pesticides during pregnancy and lactation causes immunotoxic and endocrine-disrupting effects in offspring.
2. The development of the thymus, lymph nodes, spleen, and thyroid gland is significantly impaired.
3. Pesticides disrupt the balance between cellular proliferation and apoptosis.
4. Hypothyroidism and oxidative stress play key pathogenic roles in pesticide-induced toxicity.
5. Early detection and correction of these disorders in pregnant women and newborns may help reduce the adverse consequences of pesticide exposure.

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