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Toxic Effects of Pesticides on Human and Animals

***Corresponding Author(s): Yunus Salimov**

Doctor of veterinary sciences, Researcher Institute of Veterinary Medicine of Samarkand Uzbekistan.

Email: salimov.yunus61@mail.ru

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Abstract

The review addresses issues related to the toxic effect of synthetic pyrethroids (permethrin, deltamethrin, cypermethrin, alpha-cypermethrin, zetacypermethrin, esfenvalerate), neonicotinoids (imidacloprid and thiacloprid) and pesticides from the group of organophosphorus compounds. In the pathogenesis of poisoning with these pesticides, local and systemic effects on organs and tissues, general intoxication, and a detrimental effect on the immune and reproductive systems of mammals can be distinguished. The occurrence of disorders on the part of the central and peripheral nervous system is associated with a direct or indirect effect on M and (or) N cholinergic receptors. For a long period of time, organophosphorus compounds were actively used in agriculture and in everyday life. Poisoning with this group of pesticides is characterized by neurotoxic and psychotropic effects. Modern pesticides-synthetic pyrethroids also have a toxic effect on the nervous system, causing damage to various organs and systems. Neonicotinoids, which have replaced pyrethroids, are also unsafe to use. They have a detrimental effect on various organs and tissues, mainly on the liver and kidneys. Literature data, presented in the review make it possible to assess the degree of risk of exposure to the studied pesticides on the human body and other mammals.

Introduction

The widespread use of pesticides is inextricably linked not only with agricultural production, but our everyday life makes us constantly contact with these far from harmless chemical compounds.

They can be found in food, in the environment, and contact with them in the modern world is inevitable. Nevertheless, these funds are far from safe and, if used improperly, storage and transportation rules are not followed, they can lead to negative consequences, ranging from poisoning, allergic reactions, chronic deviations from the functions of body systems and ending with death. Such long-term consequences of exposure to pesticides, such as carcinogenesis and oncogenesis, are also possible [1].

We can also observe serious consequences of the use of pesticides in the field of environmental well-being. These include problems such as a disturbance in the ratio of chemicals in the soil, which leads to an increase or decrease in the number of certain populations of animals and plant varieties. The mechanisms of action of modern and most commonly used pesticides are complex and important to study in order to prevent their harmful impact on the environment and directly on the human body.

Characteristics of the main classes of pesticides. The most common combinations of modern pesticide classes used for pest control are synthetic pyrethroids, organophosphorus compounds (OPs) and neonicotinoids.

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Synthetic pyrethroids are effective in killing insect pests, have low toxicity to mammals and, due to this advantage is actively used in agriculture [12]. However, experiments were carried out on rats, proving the negative effect of pyrethroids on immunity [7]: When applying permethrin to the outer covers, in particular, a decrease in the size of the thymus was noted [32]. For the first time, pyrethroid substances were obtained from the flowers of Persian, Dalmatian and other species of chamomile of the genus Pyrethrum. Before the discovery of this substance, people used pyrethrins (completely natural insecticides). Subsequently, pyrethroids became their synthetic counterpart. However, natural pyrethroids are unsuitable for use in the field, since they are destroyed under the action of sunlight [28].

The most commonly used in agriculture are pyrethroids such as Arivo (active ingredient - ypermethrin), Denis (deltamethrin), "Karate" (lambda-cyhalothrin) [11]. Pyrethroids are esters of cyclopropanecarboxylic acids with various radicals [13]. They are rapidly hydrolyzed by esterases [15,18] in the mammalian liver, and due to the lability of the ether bond, they are rapidly metabolized and eliminated from the body [15].

Pesticides of the FOS group have been actively used for a long period of time. They were used in forestry and agriculture, in animal husbandry, as well as in everyday life [17]. FOS are able to penetrate cell membranes, have a high degree of reabsorption through the epithelial integument, easily overcome the hematoencephalic barrier and are able to suppress the activity of not only extracellular, but also intracellular acetylcholinesterase [10]. Neonicotinoids are a new generation of insecticides. The need to use neonicotinoids arose in connection with the emerging resistance to synthetic pyrethroids and FOS [2]. In terms of structure and mechanism of action, neonicotinoids are similar to nicotine and belong to a new generation of pesticides. Therefore, they have the name "Neonicotinoids" [3]. More than 50 drugs based on five active ingredients are registered in Russia: Imidacloprid, thiacloprid, acetamiprid, thiamethoxam and clothianidin [11, 22]. According to their chemical structure, neonicotinoids are divided into two groups-nitroso-containing compounds and cyan-containing ones. Nitro-containing ones include imidacloprid, dinotefuran, clothianidin, and cyanide-containing ones-acetamiprid, thiacloprid. In the chemical structure of imidacloride, acetamiprid and thiacloprid, the common thing is the presence of a pyridine ring with one chlorine atom in the 6th position [3].

Mechanism of action of pesticides from the groups of pyrethroids, neonicotinoids and FOS The mechanisms of action of pyrethroids, neonicotinoids and FOS are inextricably linked with direct or indirect (through acetylcholinesterase) effects on M and (or) N cholinergic receptors. FOS are irreversible anticholinesterase agents. They block acetylcholinesterase by forming covalent bonds with the esterase the center of the enzyme. The bonds are formed strong, and their hydrolysis proceeds extremely slowly. That is why the inhibition of acetylcholinesterase is practically irreversible [21]. A large amount of acetylcholine accumulates in the synoptic gap, which leads to increased excitability of the nervous system, seizures and other symptoms of poisoning with this pesticide.

Pyrethroids are capable of causing functional changes in the postsynaptic membrane of a neuron. These substances act on chemically excitable ion channels [33], have a fairly high affinity for nicotinic acetylcholine receptors [20]. Cyanide-containing pyrethroids interact with Gamma-Aminobutyric Acid (GABA) receptors in the brain substances, causing functional disturbances in the extrapyramidal system and spinal intermediate neurons [20,11]. The blocking of acetylcholine receptors and the anticholinesterase activity of an external factor is carried out due to a violation of calcium metabolism in synapses and in sodium-potassium channels. As a result, acetylcholine is produced in excess and leads to overexcitation of the body, increased motor activity and other clinical manifestations associated with excessive release of this neurotransmitter. In addition, it can disrupt intracellular metabolic processes and nonspecific reactions characteristic of a stressful state. According to S.Kh. Haidarliu choline and GABAergic systems are directly or indirectly involved in the formation of the stress response of the body to environmental factors [13]. From here it can be assumed that pyrethroids can change not only the functions of the nervous system, but also adversely affect the adaptive capabilities of the body to changes in conditions external environment. Neonicotinoids have both a direct (through receptors) effect on the nervous system of insects, and an indirect one. They, like FOS, reduce activity acetylcholinesterase, which leads to an increase in the amount of acetylcholine in the synaptic cleft. Excitation of N-cholinergic receptors occurs [4,16,14], since neonicotinoids are their agonists. In addition, they significantly increase the opening of sodium channels in the postsynaptic membrane. All this leads to the onset of clinical symptoms. poisoning with this substance, similar to the symptoms of poisoning with FOS and pyrethroids. The neurotoxic effect of neonicotinoids is due to the development of tissue hypoxia, hepatopathy, toxic encephalopathy, and nephropathy [4]. Peculiarities of the toxic effect of pesticides on mammals In recent years, personal household plots have used preparations based on permethrin, deltamethrin, cypermethrin, alpha-cypermethrin, zeta-cypermethrin, and esfenvalerate. Acute poisoning with substances such as "Arivo" (cypermethrin), "Decis" (deltamethrin), "Karate" (lambda-cyhalothrin) tend to increase [11]. Moreover the clinical picture largely depends on the structure of the substance: One type of pyrethroid cause tremors, increased activity, agitation (aggressive behavior), and other - muscle contractures. The specific features of the toxic effect of type II pyretroids on mammals-cyanopyretroids - are convulsions and recurrent convulsive seizures, hypersalivation, choreoatetosis, and hyperkinesis [20].

Symptoms of poisoning form a vivid neurotoxic syndrome. Electrophysiological experimental studies suggest that the action of pyretroids causes functional changes in the postsynaptic membrane of the neuron. These substances act on chemically excitable ion channels, have a fairly high affinity for nicotinic acetylcholine receptors [11]. Cyanide-containing pyretroids interacting with the GABA receptors of the brain can cause functional impairments in the work of the extrapyramidal system and spinal intermediate neurons [34,20]. The way the toxic substance enters the human body also affects the manifestation of these or other symptoms. The active substances can enter through the respiratory pathways, gastrointestinal tract, intact skin [11]. In the liver, pyretroids are subject to oxidation and hydrolysis with the formation of glucuronates. Acute pyretroid poisoning manifests itself most often in the form of headache, burning and itching of the facial skin, dizziness, general weakness, in the first 2–3 days of an increase in body temperature [30]. At oral admission after 10-60 minutes, pain in the stomach, nausea, vomiting, headache, dizziness, increased gastric secretion, muscle twitching appear. In the most severe cases, convulsions develop, shortness of breath with moist wheezing, indicating the development of pulmonary edema, loss of

consciousness [20]. Experiments on animals have revealed that acute intoxication with deltamethrin causes hypersecretion of glucocorticoids, hyperglycemia during the development of insulin resistance [6]. Studies by E.A. Chigrinski (2017) found abnormalities in the synthesis of corticosteroids by the adrenal glands of rats exposed to high doses deltamethrin, which indicates the development of a stress response in experimental animals to the action of synthetic pyrethroids [26].

Synthetic pyrethroids interfere with reproductive function [29]. This is associated with impaired function of the antioxidant system in the gonads [8,19] Deltamethrin helps to reduce the concentration of glutathione and impair the activity of glutathione dependent enzymes, which contributes to the development of oxidative stress in the testes of rats [25,8]. Acute poisoning with a new generation of insecticides, neonicotinoids [33], is rarely encountered in everyday life, the acute toxicity of which is more pronounced with oral ingestion of this insecticide and, to a lesser extent, with transcutaneous and inhalation exposure [34].

It is known that in high doses imidacloprid (a structural analogue of nicotine) activates the central nervous system similar to the action of nicotine [35,27], causing tremor, pupillary dysfunction, and hypothermia [11]. The highest concentration of neonicotinoids is registered in the liver and kidneys, while the mass of the liver increases and the enzymatic activity increases. Acute neonicotinoid poisoning is manifested by drowsiness, disorientation, dizziness, while gastrointestinal erosion, hemorrhagic gastritis, fever, leukocytosis and hyperglycemia are also observed. Patients with imidacloprid poisoning recover without complications within 2-3 days.

Additional examinations carried out every month, as a rule, do not reveal pathologies on the part of organs and systems [34]. One of the manifestations of the adrenergic syndrome in case of poisoning with imidacloprid and thiacloprid-containing pesticides is intestinal atony, as evidenced by lack of disease in animals during the day after acute poisoning. During the autopsy of corpses and animals killed at the end of the experiments, an acute expansion of the stomach and the blind intestine is noted, confirming the development of transient intestinal paresis [5].

In case of acute poisoning of animals with nonicotinoids, the following effects are noted: Neurotoxicity, immunotoxicity, hepatotoxicity, nephrotoxicity and reproductive cytotoxic effect [35]. One day after acute poisoning with “Confidor®” extra and “Calypso®” in animals, neutrophilia, lymphocytopenia and monocytopenia are recorded in the background of leukopenia, and after seven days - relative lymphocytosis, decrease in erythrocytopenia and decrease in the amount of erythrocytopenia. Indicators of total bilirubin, aminotransferases, urea, creatinin and thymol test increase, indicating the development of toxic hepatopathy and nephropathy. In chronic poisoning with nonicotinoids, anemia, leukopenia, increased activity of serum aminotransferases and total bilirubin are noted [5].

In case of poisoning with imidacloprid-and thiacloprid-containing pesticides, the pathomorphological picture is characterized primarily by vascular disorders and dystrophic changes in the parenchymal organs. A common symptom of poisoning is metheorism with enlargement of the stomach and blind intestine. Poisoning with imidacloprid is characterized by the development of hydropic dystrophy of the liver and kidneys [35] and cardiomyocytes; for poisoning with thiacloprid - development of granular dystrophy, infiltration with mononuclears and proliferation of connective tissue in organs. In birds, the drugs cause enlargement of the goiter and fatty degeneration of the liver [5]. In the experimental work of J. Kim [31], it was stated Assumption of the role of nonicotinoids in the onset of type II diabetes mellitus. When studying the effect of small doses of imidacloprid (0.5; 2; and 8 mg / kg of weight for three months) on the organs of the reproductive system of male rats by a group of Turetsky Researchers have established a slowdown in mobility and a change in the morphology of spermatozoa, as well as a significant decrease in testosterone levels and an increase in the apoptosis index in the reproductive cells of the genital tubules, and in the composition of DNA from rats. It has been experimentally proven that imidacloprid and thiacloprid provoke abortions in pregnant females [9], and in the work of A. Anadon [35] data on a negative effect on the reproductive system of male rats are given. In particular, with the introduction of imidacloprid, a decrease in the viability was observed sperm. The author points out the probable genetic danger posed by nonicotinoids and emphasizes the importance of protective measures and rules of technology safety when working with them. T. Green based on the results of chronic experiments carried out on in males and females of mice, notes that thiamethoxam can be attributed to probable carcinogens for humans due to the increased morbidity of animals from hepatocellular adenoma and carcinoma [5]. Insecticides from the FOS group, cholinesterase inhibitors are highly toxic [9]. The study of the structure of pesticides for 20 years showed that 80% of severe poisonings, which are accompanied by disruption of vital functions and require intensive therapy, are related to cases of poisoning with OPs (65%), including 15% of cases where pesticides are not present.

FOS can enter the human body through the respiratory system, mouth or through the skin. They have psychotropic and neurotoxic effects. There are three stages in the development of FOS poisoning: The first is psychomotor agitation, tightness in the chest, shortness of breath, moist wheezing in the lungs (bronchorea), sweating, increased arterial pressure; the second - separate and involuntary twitching of muscles, convulsions, respiratory failure due to increasing bronchorae, rare pulse, involuntary loose stools, frequent urination; the third - disruption of the respiratory center to complete cessation of breathing, paralysis of the muscles of the extremities, a drop in arterial pressure, disturbance of the rhythm and conduction of the heart [21]. There are known lesions proceeding according to the type of allergic dermatitis, asthmatic bronchitis and other diseases. It is believed that allergic reactions are associated with the ability of FOS to affect functional groups of different proteins [11].

Conclusion

Thus, pesticides cause negative effects in humans and animals. Since pesticides are biologically active substances in the environment, certain requirements are made that ensure the greatest efficiency of their use and the least harm to humans and animals. Considering the great work carried out in the field of creating new pesticides and selection of the assortment, one can hope that the harmful effects will decrease and the selectivity of the action of pesticides on various living organisms will increase. To the drugs used, which is currently managed by the use of mixtures of pesticides with different mechanisms of action. For example, the use of mixtures of contact and systemic fungicides, insecticides - pyretroids with organophosphate insecticides. Acquisition of resistance causes the need for system-

atic replenishment of the assortment with drugs with different mechanisms of action, which requires high costs means and time.

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