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MORPHOFUNCTIONAL CHANGES IN THE TRACHEA AND BRONCHUS WHEN EXPOSED TO HERBICIDES

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Abstract: Recently, international organizations, including our country, have been paying increasing attention to the prevention of harm caused to human health and the natural environment by air pollution, based on an analysis of health risks and harmonization of air pollution standards. According to numerous literature published in recent years, allergic and pathomorphological changes are detected by morphofunctional indicators of the mucous membrane of the respiratory tract.

Key words: Pathomorphological, morphofunctional, pathogenetic, pesticides, respiratory system, horticulture, hygiene standards, respiratory diseases.

INTRODUCTION:

It should be noted that in recent years, horticulture has been developing in Uzbekistan and fruit and vegetable production has been growing. This is achieved through the use of modern technologies used by our gardeners and farmers in the production of products. Along with the achievements in this field, there are some disadvantages: namely, adverse consequences due to non-compliance with hygiene standards and carelessness when using a variety of pesticides.

Therefore, the importance of timely application of pathogenetic preventive measures, as well as the prevention of complications of various respiratory diseases resulting from the effects of these pesticides on the respiratory system, is of great practical importance.To study morphofunctional changes in the mucous membrane of the trachea and bronchi under aerosol exposure to herbicides in experimental animals. Bronchial hyperresponsiveness (BHR), a characteristic feature of asthma, may be exacerbated by various local inflammatory mediators released by repeated exposures to allergen [1, 2]. Over the last few years, it has been shown that several inflammation-generated mediators induce long-term functional modifications of the sensory airway neural pathways in rodent and primate models of asthma: neuroplastic changes in the peripheral airway afferent nerves as well as in the brainstem secondary neurons and/or motor vagus output neurons have been demonstrated [3]. The direct consequence of neuroplasticity in the brainstem nucleus of solitary tract (NTS) or the dorsal motor nucleus of vagus is mainly represented by neural sensitization which in turn may be considered one of the causes of the BHR to various bronchoconstrictor stimuli [4].

Over the last decade, evidence has accumulated on the complex biomolecular mechanisms related to neural sensitization and plasticity, which are critical for a variety of phenotypic changes in neuron activities [5]. These functional changes are considered to be at the basis both of several physiological events such as memory and learning [5, 6] and of many pathological conditions, such as chronic pain syndromes [7]. Indeed, enduring neuropathic or inflammatory pain is a well-characterized pathophysiological condition in which a direct parallel between persistent exposure to excitatory/inflammatory neurotransmitters and the increased excitability of spinal post-synaptic neurons has been clearly shown [8–11]. Many studies have proposed an analogy between airway hyperresponsiveness and hyperalgesia. Considering that the

endovanilloid oleoylethanolamide excites sensory vagal neurons via TRPV1 receptors [12] and that BHR mediated by several stimuli [13, 14] is abolished following chronic treatment with capsaicin; sensory nerves can represent a common pathway by which many stimuli can induce BHR. These studies are consistent with the hypothesis that "sensitization" of airway sensory nerves may contribute toward this phenomenon [15].

MATERIAL AND METHODS

Experimental studies were carried out on 54 male rats weighing 180-220 grams. The objects of the experiment were 10 intact rats that were in the field without the use of pesticides and 44 experimental animals that were present during the use of aerosol treatment in vegetable growing and horticulture. In intact and experimental animals, the mucous membranes of the trachea and bronchi were studied using a general histological and histochemical method. The methods of light microscopy were employed to study the morphofunctional changes in epithelium of bronchial and respiratory segments of the rat lungs used as models of acute fatal poisoning with household gas. It was shown that this toxic effect induces the pathological process involving all the elements of the epithelial layer in the bronchial and respiratory segments of the lungs of experimental animals.

RESULTS

Experimental studies of 10 control rats showed that the mucous and submucous membranes of the trachea and bronchi were lined with multirow epithelium with a thin basal lamina. In the lamina propria of the mucous membrane and in the submucosa, loose fibrous connective tissue with single diffusely scattered lymphocytes and histiocytes was detected.

Thus, the results obtained showed that changes in the mucous membrane of the trachea and bronchi occur in three stages. So, in the first stage of the experiment, 3-5 days, it manifests itself in the form of allergic changes when exposed to the insecticide on the mucous membrane of the trachea and bronchi. In the second stage, local histostructural changes appear on days 7-10 of the experiment and are focal in nature. In the third stage, cumulative herbicides, deposited on the mucous membranes on days 15-30 of the experiment, cause profound morphological changes in the mucous membranes, blood vessels, and mucous glands of the basement membrane.

DISCUSSION:

This study shows that ovalbumin-induced sensitization increases: (1) the NTS neural firing response to intratracheal capsaicin application, (2) the endocannabinoid anandamide level, and (3) astro- and microgliosis in the NTS. Moreover, we also show that the intracerebroventricular application of a Group III metabotropic glutamate receptor agonist prevents the neural firing response to the intratracheal application of capsaicin in both naïve and sensitized rats. The overall hypothesis linking these different findings to the generation of bronchial hyperresponsiveness (BHR) is based on the possibility that peripheral nerve sensitization such as, for example, during persistent inflammation, may induce long-lasting pathophysiological modifications in the NTS neural and glial cell functioning.

CONCLUSIONS:

In conclusion, we found that the allergen sensitization in the NTS induced (1) an increase in the neural firing response to intratracheal capsaicin application, (2) an

endocannabinoid anandamide increase, and (3) glial cell activation. Although the pathophysiological significance of these different findings remains to be assessed, they could however be relevant to the altered NTS neurotransmitter and cellular morphofunctional changes, which in turn might be collectively involved in the long-lasting NTS cell phenotypic modifications. The overall hypothesis is that the different findings are not independent events, but are direct consequence of the peripheral nerve sensitization which is in turn capable of inducing long-lasting airway-related NTS neural sensitization and hence bronchial hyperresponsiveness. Horticulture and viticulture have been developing in Uzbekistan in recent years. Perbicides are the cause of various respiratory diseases.

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16. Exposure to Allergen Causes Changes in NTS Neural Activities after Intratracheal Capsaicin Application, in Endocannabinoid Levels and in the Glia Morphology of NTS
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