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HISTOMORPHOLOGICAL CHANGES OF THE LARYNGEAL MUCOSA UNDER THE INFLUENCE OF CYPERMETHRIN AND ITS EFFECT ON THE LARYNGEAL MICROSTRUCTURE AND VASCULAR SYSTEM IN LABORATORY ANIMALS

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Abstract

Cypermethrin is a widely used synthetic pyrethroid insecticide that may cause toxic effects on the upper respiratory tract, particularly the laryngeal mucosa, after inhalational exposure. Damage to laryngeal tissues can impair respiratory and phonatory functions.

This study analyzed histomorphological changes in the laryngeal mucosa based on experimental and literature data. Histological and toxicological findings focused on epithelial degeneration, inflammation, vascular changes, and oxidative stress.

Cypermethrin exposure caused epithelial degeneration, destruction of the ciliary apparatus, inflammatory infiltration, stromal edema, vascular dilation, and microcirculatory disturbances. Chronic exposure led to fibrosis and squamous metaplasia. Oxidative stress played a major role in tissue injury.

Cypermethrin exerts significant toxic effects on the laryngeal mucosa through inflammatory and degenerative mechanisms. These findings highlight the importance of preventive measures and safety standards during pesticide exposure.

Keywords: Cypermethrin, pyrethroid, larynx, laryngeal mucosa, histomorphology, epithelial degeneration, oxidative stress, fibrosis, inflammation, toxicology.

Introduction. Synthetic pyrethroid insecticides are extensively used worldwide because of their high insecticidal activity and relatively low cost. Among these compounds, cypermethrin is one of the most commonly applied pesticides in agriculture and household pest control [1,2]. However, increasing environmental exposure has raised concerns regarding its toxicological effects on human health and biological systems.

The respiratory tract is continuously exposed to environmental toxicants and therefore represents one of the primary targets of inhalational pesticides. The larynx plays an essential role in respiration, phonation, and airway protection. Its mucosal surface consists of pseudostratified ciliated columnar epithelium, glandular structures, connective tissue stroma, and a developed vascular network that collectively ensure mucociliary clearance and local immune defense[3,4].

Cypermethrin enters the body through inhalation, dermal contact, and the digestive tract. Inhalational exposure is particularly important because aerosolized pesticide particles directly contact the mucosa of the upper respiratory tract. Experimental studies indicate that cypermethrin induces oxidative stress, free radical formation, endothelial dysfunction, inflammatory reactions, and structural degeneration within respiratory tissues[1,6,7].

At the cellular level, cypermethrin disrupts membrane permeability and sodium channel function, leading to mitochondrial dysfunction and activation of apoptosis and necrosis pathways. Oxidative damage promotes lipid peroxidation and destruction of intracellular proteins and nucleic acids, thereby impairing epithelial integrity and vascular homeostasis[1,5].

Despite the widespread use of cypermethrin, the histomorphological mechanisms underlying laryngeal injury remain insufficiently studied. Therefore, investigation of cypermethrin-induced changes in the laryngeal mucosa and vascular system is of considerable scientific and clinical significance.

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Aim of the Study. The aim of this study was to analyze histomorphological changes in the laryngeal mucosa caused by inhalational exposure to cypermethrin and to evaluate its effects on the laryngeal microstructure and vascular system in laboratory animals.

Materials and Methods. This study was based on a comprehensive analysis of experimental toxicological and histological data concerning the effects of cypermethrin on the laryngeal mucosa in laboratory animals. Histological observations and morphometric evaluations described in experimental models were systematically analyzed.

The normal structure of the laryngeal mucosa, including epithelial architecture, lamina propria composition, vascular organization, and glandular structures, was compared with pathological alterations induced by cypermethrin exposure.

Histological assessment focused on: epithelial degeneration; destruction of the ciliary apparatus; inflammatory infiltration; stromal edema; vascular alterations; fibrosis and metaplastic transformation.

Special attention was given to oxidative stress mechanisms and their contribution to epithelial and endothelial injury. Morphological changes were evaluated according to previously described toxicological and histopathological criteria.

Relevant scientific literature, histology atlases, toxicology textbooks, and WHO reports on pesticide toxicity were additionally reviewed to support interpretation of the findings.

Results. Under physiological conditions, the laryngeal mucosa is lined predominantly by pseudostratified ciliated columnar epithelium. Regions exposed to significant mechanical stress contain stratified squamous epithelium. The lamina propria contains collagen and elastic fibers, blood vessels, lymphoid cells, and mucous glands responsible for secretion and humidification of the mucosal surface. Coordinated ciliary movement ensures efficient mucociliary clearance and protection against inhaled particles and microorganisms.

Exposure to cypermethrin caused pronounced degenerative alterations in epithelial cells. Histological examination demonstrated hydropic degeneration, cytoplasmic vacuolization, pyknosis of nuclei, and focal necrosis. In several regions, disruption of epithelial continuity was observed, indicating severe toxic injury.

Cypermethrin exposure induced shortening and destruction of cilia, leading to impaired mucociliary transport. Dysfunction of the ciliary apparatus reduced the ability of the respiratory tract to eliminate foreign particles and pathogens, thereby promoting inflammatory processes.

Marked inflammatory infiltration consisting predominantly of lymphocytes and neutrophils was observed in the lamina propria. Blood vessel dilation and stromal edema accompanied inflammatory reactions. Chronic exposure resulted in progressive thickening of the laryngeal wall and increased stromal remodeling.

Significant microcirculatory disturbances were identified within the vascular system. Capillary dilation, endothelial dysfunction, increased vascular permeability, and hemodynamic abnormalities were evident. These alterations contributed to tissue hypoxia and exacerbation of inflammatory damage.

Long-term cypermethrin exposure promoted fibrosis and epithelial metaplasia. Respiratory ciliated epithelium was gradually replaced by stratified squamous epithelium. Although metaplasia represents an adaptive response to chronic irritation, it significantly impairs normal physiological functions of the laryngeal mucosa. Fibrotic remodeling reduced tissue elasticity and predisposed to chronic pathological conditions.

Oxidative stress was identified as a major pathogenic mechanism underlying tissue injury. Cypermethrin-induced generation of reactive oxygen species enhanced lipid peroxidation and caused mitochondrial dysfunction. These processes activated apoptotic and necrotic pathways, contributing to progressive epithelial and stromal damage.

Discussion. The present study demonstrates that cypermethrin exerts pronounced toxic effects on the laryngeal mucosa and microvascular system. Histomorphological changes induced

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by cypermethrin are characterized by a combination of degenerative, inflammatory, vascular, and proliferative processes.

One of the primary mechanisms of injury involves oxidative stress and free radical formation. Enhanced lipid peroxidation damages cellular membranes and intracellular organelles, particularly mitochondria. These findings are consistent with previous toxicological studies reporting oxidative tissue injury after pyrethroid exposure [1,2,7].

Destruction of the ciliary apparatus significantly impairs mucociliary clearance, thereby weakening local defense mechanisms of the respiratory tract. The resulting accumulation of inhaled particles and microorganisms intensifies inflammatory reactions and contributes to chronic mucosal injury.

Vascular disturbances represent another important component of cypermethrin toxicity. Endothelial dysfunction and increased vascular permeability promote stromal edema and microcirculatory insufficiency, ultimately leading to tissue hypoxia. Persistent hypoxic conditions stimulate fibrogenesis and chronic remodeling processes [5,7].

The development of squamous metaplasia during chronic exposure reflects an adaptive but functionally inadequate response of the respiratory epithelium to continuous toxic irritation. Similar metaplastic transformations are commonly observed in chronic inflammatory diseases of the respiratory tract and are associated with impaired physiological function [4,5].

Overall, the obtained findings indicate that cypermethrin toxicity extends beyond its neurotoxic effects and significantly affects respiratory tissues. These morphological changes may contribute to chronic laryngeal dysfunction and respiratory pathology in individuals exposed to pesticides over prolonged periods.

Conclusion. Cypermethrin is a synthetic pyrethroid insecticide capable of inducing significant histomorphological alterations in the laryngeal mucosa and vascular system following inhalational exposure. The toxic effects are manifested by epithelial degeneration, destruction of the ciliary apparatus, inflammatory infiltration, vascular dysfunction, fibrosis, and epithelial metaplasia.

Oxidative stress and free radical formation play central roles in the pathogenesis of tissue injury. Chronic exposure leads to progressive structural remodeling that may impair the respiratory, protective, and phonatory functions of the larynx.

These findings emphasize the necessity of: strengthening occupational safety standards; minimizing inhalational exposure to pesticides; using personal protective equipment; implementing preventive toxicological monitoring.

Further experimental and clinical studies are required to better understand the long-term effects of cypermethrin on respiratory tissues and to develop effective protective strategies.

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