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UPPER RESPIRATORY TRACT EPITHELIUM AND ITS PROTECTIVE FUNCTION

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Abstract. This abstract examines in detail the morphological, histological, and immunobiological characteristics of the upper respiratory tract epithelium, which provides multilevel protection of the body against inhaled pathogens and harmful environmental factors. The upper respiratory tract (nose, nasopharynx, larynx, and initial segments of the trachea) is lined mainly by pseudostratified ciliated columnar epithelium with pronounced structural specialization. It includes ciliated cells, goblet cells, basal stem cells, brush cells, endocrine cells, and areas of stratified squamous epithelium in regions exposed to increased mechanical stress.

The epithelium performs barrier, transport, sensory, immune, and secretory functions, enabling constant interaction between the external environment and intrinsic mechanisms of homeostasis. The study emphasizes that the key protective mechanism of the epithelial lining is mucociliary clearance, formed by coordinated ciliary activity and the tracheobronchial secretion layer consisting of two components: the gel layer (mucus) and the sol layer (periciliary fluid). Histologically, this apparatus is supported by specialized epithelial cells secreting the mucins MUC5AC and MUC5B, as well as ion channel regulation that ensures proper mucus hydration.

The upper respiratory tract epithelium is considered an active immune organ. It contains innate immune receptors (TLR, NOD pathways), secretory IgA, β-defensins, lysozyme, lactoferrin, and other antimicrobial factors. Special attention is given to interactions between the epithelium and local immune system cells — dendritic cells, macrophages, lymphoid follicles, and MALT components (NALT, BALT), which contribute to the formation of local adaptive immunity.

A separate focus is placed on the regenerative potential of the epithelium. Basal cells with stem-cell properties provide continuous renewal of the epithelial lining and physiological plasticity, including the ability for metaplasia and restoration of the ciliated surface after injury. The study examines morphofunctional changes in the epithelium under the influence of risk factors: viral infections (especially respiratory viruses), bacterial toxins, air pollutants, tobacco smoke, allergens, and xenobiotics. Particular attention is given to the toxic effects of organophosphate compounds, including chlorpyrifos, which disrupt mucociliary transport, damage cilia, increase epithelial permeability, intensify inflammatory responses, and induce tissue remodeling.

The abstract emphasizes that the upper respiratory tract epithelium is a highly specialized system integrating mechanical (cilia), chemical (mucins, enzymes), immunological (secretory IgA, antimicrobial peptides), and cellular (dendritic cells, lymphocytes) protective mechanisms. The complexity and synergy of these mechanisms ensure effective filtration, inactivation, and removal of foreign particles, maintaining sterility in the lower respiratory tract. The results highlight the importance of studying epithelial morphological changes under harmful exposures and the need to develop strategies for preventing mucociliary dysfunction and epithelial barrier impairment.

Keywords: Upper respiratory tract epithelium; pseudostratified ciliated epithelium; ciliated cells; goblet cells; mucociliary clearance; MUC5AC; MUC5B; secretory IgA; antimicrobial peptides; innate immunity; epithelial barrier; Toll-like receptors (TLR); respiratory tract; morphofunctional alterations; inflammatory response; organophosphate compounds; chlorpyrifos; epithelial remodeling; basal stem cells; epithelial regeneration; MALT system; NALT; toxic exposure; air pollution; respiratory disease pathogenesis.

INTRODUCTION

The upper respiratory tract epithelium is a highly specialized multicompartmental system providing the body's primary defense against airborne environmental factors. Under constant exposure to microorganisms, allergens, pollutants, xenobiotics, and mechanical particles, the epithelial lining functions as an integrated barrier performing mechanical, secretory, sensory, and immunoregulatory functions. The study of the histological structure of the epithelium is crucial, as cellular organization, intercellular junctions, and functional differentiation determine the efficiency of mucociliary clearance and immune protection.

Modern research shows that epithelial cells of the upper respiratory tract are not passive anatomical barriers. They actively participate in pathogen recognition via Toll-like receptors, synthesize antimicrobial peptides and secretory IgA, regulate mucus hydration, and interact with innate and adaptive immune cells. Structural disruption of the epithelium reduces mucociliary activity, promotes chronic inflammation, contributes to tissue remodeling, and leads to respiratory diseases.

The introduction emphasizes the relevance of studying morphofunctional changes in the epithelium under exposure to toxic agents, including organophosphates, viral infections, and tobacco smoke, which impair ciliary function, alter mucin synthesis, and increase epithelial permeability.

AIM OF THE STUDY

The aim of this work is a comprehensive morphofunctional investigation of the upper respiratory tract epithelium and analysis of its protective mechanisms, with a focus on:
- cellular composition and features of pseudostratified ciliated epithelium;
- mechanisms of mucociliary clearance;
- humoral and cellular factors of innate immunity;
- regenerative capacity of epithelial cells;
- structural and functional epithelial changes under damaging factors, including organophosphate compounds;
- the role of the epithelial barrier in maintaining respiratory homeostasis.

MATERIALS AND METHODS

The study includes morphological, histological, and immunohistochemical techniques to evaluate the condition of the upper respiratory tract epithelium.

1. Study Objects:
- samples of nasal cavity, nasopharynx, and initial tracheal epithelium;
- isolated ciliated, goblet, and basal stem cells;
- biopsy material from patients with inflammatory airway diseases;
- experimental samples exposed to chlorpyrifos and other pollutants.

2. Histological Methods:

- Hematoxylin–eosin staining for general tissue architecture;
- PAS reaction for mucin visualization;
- Elastin and collagen staining for submucosal structure assessment.

3. Immunohistochemistry:
- detection of mucins MUC5AC, MUC5B;
- markers: CK5/6 (basal cells), β-tubulin (cilia), Ki-67 (proliferation);
- detection of secretory IgA;
- inflammatory markers: IL-8, TNF-α.

4. Electron Microscopy:
- ultrastructural analysis of cilia, tight junctions, and desmosomes.

5. Functional Methods:
- measurement of mucociliary transport rate;
- analysis of mucus viscosity and elasticity;
- biochemical analysis of airway secretions;
- evaluation of epithelial barrier permeability.

RESULTS AND DISCUSSION

1. Structural Organization:
Histological analysis confirmed three major cell populations: ciliated, goblet, and basal cells. Regeneration occurs through basal cell proliferation marked by high Ki-67 expression. Toxin exposure reduced ciliated cell density and disrupted epithelial architecture.

2. Mucociliary Clearance:
Normal cilia beat synchronously to move mucus. Chlorpyrifos exposure significantly reduced ciliary beat frequency, increased mucus viscosity, and disrupted the MUC5AC/MUC5B ratio, causing mucus stagnation.

3. Immune Activity:
Epithelial cells expressed β-defensins, LL-37, lysozyme, and IgA. Toxin-exposed tissues showed reduced IgA and increased IL-8 and TNF-α expression.

4. Regeneration:
Basal cells differentiate into all epithelial cell types. Chronic irritation led to squamous metaplasia, reducing mucociliary efficiency.

5. Toxin-Induced Changes:
Organophosphates caused:
- degeneration of cilia,
- epithelial disorganization,
- increased permeability,
- inflammatory remodeling,
- goblet cell hyperplasia and excessive mucus production.

CONCLUSIONS

1. The upper respiratory tract epithelium is a dynamic, multicomponent barrier essential for protection against airborne pathogens.
2. Mucociliary clearance is the primary mechanical defense.
3. Epithelial cells play an active immune role.
4. Basal cells ensure regeneration; toxins disrupt differentiation.
5. Chlorpyrifos damages epithelium and impairs clearance.
6. Further research is needed to prevent epithelial barrier dysfunction.

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