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«Профілактична медицина: сучасні виклики та перспективи»

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Матеріали конференції відображають широкий спектр сучасних проблем громадського здоров'я, клінічної і профілактичної медицини. У збірнику представлені результати наукових досліджень і практичних розробок з питань профілактики професійних і неінфекційних захворювань, здоров'язбережувальних технологій, психогігієни, гігієни харчування та санітарного забезпечення населення в умовах сучасних викликів.

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Оргкомітет конференції вважав за доцільне залишити авторські тексти без змін

Зміст

THE IMPACT OF OCCUPATIONAL CONTACT WITH FORMALDEHYDE ON THE DEVELOPMENT OF LEUKEMIA

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«Stop evil before it arises»
Laozi, Chinese philosopher, 6th century BC

The relevance is due to the widespread use of formaldehyde in departments and units of morphological profiles, its constant presence in work areas, which determines prolonged chronic exposure to employees.

The aim of the study: To examine and determine the risks of developing dysfunctions of the hematopoietic system under the influence of formalin-containing substances by analyzing their toxicological, carcinogenic, and genotoxic properties.

Materials and methods: Review and analysis of scientific and methodological periodicals.

Results and conclusions: According to research findings and the analyzed literature, it was revealed that formaldehyde is a highly reactive chemical substance used in many fields of activity. According to international classifications of associations studying malignant neoplasms of the nasopharynx and leukemia, it is classified as a Group 1 carcinogen. However, the relationship between formaldehyde exposure and leukemia incidence remains a debatable issue. Genetic changes caused by oxidative stress and formaldehyde may disrupt the function of the hematopoietic system and are likely to lead to leukemia. There are genes believed to be susceptible to formaldehyde exposure and associated with the development of leukemia.

Formaldehyde is a colorless, highly toxic substance with a sharp odor. As the simplest form of aldehyde (H-CHO), formaldehyde is synthesized by catalytic oxidation of methanol and dissolves easily in water. A 37% solution of formaldehyde is used as a preservative, pesticide, and disinfectant [1].

Most commonly, formaldehyde affects individuals through inhalation from the environment and in workplaces where products containing it are widely used. According to WHO recommendations, indoor formaldehyde levels should be limited to 0.1 mg/m³ (0.08%). Under normal biochemical processes such as DNA/RNA demethylation and oxidative deamination, a small amount of formaldehyde is produced. The concentration of endogenous formaldehyde in human blood is approximately 2–3 mg/L (0.1 mM). Exposure to exogenous formaldehyde is usually associated with irritation of the eyes and upper respiratory tract. Formaldehyde has geno- and cytotoxic properties, causing DNA damage and chromosomal changes. Increased genomic instability caused by genotoxic chemicals may enhance the risk of cancer development.

Chronic and subchronic studies of nasal tumors in rats conducted over various years have provided sufficient evidence that inhaled and oral exposure to formaldehyde causes cancer. There is a certain relationship between the frequency of tumor development and exposure to different doses of formaldehyde. The connection between formaldehyde and cancer development has also been identified in many epidemiological studies. Epidemiological investigations of occupational formaldehyde exposure have revealed a significant increase in mortality from malignant nasopharyngeal tumors. It is likely that formaldehyde causes nasopharyngeal cancer and leukemia and contributes to the development of cancer of the paranasal sinuses.

By reacting with DNA, formaldehyde can exert its genotoxicity. In occupational studies of formaldehyde exposure in humans, increased DNA damage, micronucleus formation, sister chromatid exchange, and chromosomal aberrations were observed in peripheral lymphocytes and mucosal cells. Individuals exposed to formaldehyde showed an increased risk of genetic damage, reflected in biochemical blood analyses as changes in the percentages of B cells, T (cytotoxic) cells, and natural killer cells. In vitro studies demonstrated that formaldehyde causes DNA–protein crosslinks in leukocytes and nasal epithelial cells.

In workers exposed to formaldehyde, increased levels of dipeptidylpeptidases in leukocytes were detected; these indicators represent early signs of carcinogenesis, and their elevated levels serve as a biomarker of formaldehyde exposure. The existing genotoxic effects are a probable mechanism for the carcinogenic influence of formaldehyde.

It is possible that formaldehyde causes distant toxicity, as it is rapidly metabolized and highly reactive, and its toxic effects generally act locally.

The probable leukemogenic properties of formaldehyde include:

1. the ability of exogenous formaldehyde to penetrate bone marrow;
2. the ability of formaldehyde to cause hematological toxicity;
3. the occurrence of leukemia in laboratory animals exposed to formaldehyde.

Three possible mechanisms of formaldehyde-induced leukemia have been proposed: direct damage to stem cells; damage to hematopoietic stem/progenitor cells circulating in the blood; damage to primitive pluripotent stem cells located in the nasal and oral cavities.

Considering that formaldehyde and its metabolic pathway are naturally present in all cells, high concentrations of formaldehyde that significantly exceed metabolic levels lead to toxicity. It has been established that exogenous exposure to formaldehyde does not cause distant toxicity beyond the immediate site of exposure. However, there are potential mechanisms related to oxidative stress that emphasize the toxicity of formaldehyde to red bone marrow. [2,3].

It is necessary to consider the processes of oxidative stress and key genes (further research is required) associated with formaldehyde and leukemia to confirm causal relationships. Important factors such as individual genetic background, interspecies differences, and exposure degree must be taken into account. It is important to develop methods that will allow accurate assessment of distant toxicity using carefully selected genomic data for modeling long-term occupational exposure. Undoubtedly, it is also reasonable to consider such possibilities as abnormal activation of key genes and signaling pathways, which will enable the study of genetic factor influences and be used to identify new mechanisms and biomarkers.

References:

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