It was not possible to identify statistically significant differences in the concentration of Zn, Ca and Fe when was comparing the results of the content of trace elements in the hair in patients with coxarthrosis and gonarthrosis. Although it showed an increase in Cu (p = 0,048) in patients with coxarthrosis compared with gonarthrosis.

A statistically significant inverse correlation was found between the index of body mass index and concentration Zn (Rs = –0.89, p = 0,0005) in coxarthrosis. It was found that the higher the body mass index, the lower the concentration of Zn in the hair during coxarthrosis, which may indicate obesity, increased load on the joints, and, consequently, the appearance and progression of joint diseases.

The results can be used to assess the level of trace elements in patients with musculoskeletal pathology and correct their nutrition.

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ANTIOXIDANT ACTIVITY OF THE EXTRACTS OF CHESTNUT FLOWERS (*AESCULUS HIPPOCASTANUM* L), ROWAN (*SORBUS AUCUPARIA* L.), ACACIA (*ACACIA*) AND DIFFERENT TYPES OF LILAC (*SYRINGA*)

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The comparative study of the antioxidant activity of extracts of acacia, rowan, chestnut and different types of lilac flowers has been conducted. The dependence of the fluorescence intensity of fluorescein from the logarithm of the concentration of extracts of flowers was obtained, where the indicators IC50 were determined graphically. Extracts of flowers of acacia, rowan and chestnut restored fluorescence of fluorescein to 98-100% at a concentration of samples of 10-3-10-2%. Extracts of lilac flowers restored the fluorescence of fluorescein to 86-95% at a concentration of samples of 10-1-1%. The IC50 of extracts of flowers of acacia, rowan and chestnut were within 2÷5,3·10-5%, extracts of flowers of lilac - within 1,26÷7,31·10-4%. The maximum antioxidant activity for acacia extract is determined.

Keywords: antioxidant activity, extracts of flowers of acacia, rowan, chestnut and different types of lilac, fluorescein.

Excessive concentration of free radicals in the body is a central risk factor for cardiovascular, cancer and other pathologies. Flavonoids have strong antioxidant properties and can be used to prevent various diseases. Biologically active substances that are a part of the flowers of acacia, chestnut, Rowan and lilac, determine their pharmacological properties, which allows them to be used as a raw source for the pharmacological industry. The flowers of acacia white contain glycoside robinin, as well as a number of other flavonoids [1].

A comparative study of antioxidant activity (AOA) of extracts of acacia flowers, mountain ash, chestnut and 6 different species of lilac was carried out. The method for determining AOA with respect to activated oxygen species (ROS) is based on measuring the fluorescence intensity of the oxidized compound and its decrease under the influence of ROS.

Extract of flowers of mountain ash starts to show the antioxidant activity at a concentration of 10^{-9} %. Chestnut flower extract begins to exhibit AOA at a concentration of 10-9%. Acacia flower extract begins to exhibit AOA at a concentration of 10^{-9} %. Lilac flower extract begins to exhibit AOA at a concentration of 10^{-6} %. Lilac flower extract dark lilac begins to show AOA at a concentration of 10-7%. White lilac flower extract begins to exhibit AOA at a concentration of 10^{-7} %.

Maximum AOA is obtained for the extract of dark lilac lilac flowers. Suppression of free radicals up to 95 % is achieved at the lowest concentration of 10^{-2}%. Extracts of lilac flowers showed lower AOA compared to ex-

tracts of acacia, mountain ash and chestnut flowers. Antioxidant activity began to appear at the concentration of these extracts two orders of magnitude higher (10^{-7}%) than the concentration of extracts of acacia flowers, mountain ash and chestnut (10^{-9}%). Obviously, the lower content of phenolic and glycoside compounds included in the extracts of lilac flowers explains their weaker antiradical activity compared to extracts from acacia flowers, mountain ash and chestnut.

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EFFECTS OF NITROGENOUS BASE ANALOGUES AND NUCLEOSIDES IN TUMOR CELLS

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Understanding the differences in metabolism, pharmacodynamics, and tumor biology between treatable and non-treatable patients provides the scientific basis for improving therapy and integrating this therapy into medicine.

Keywords: chemoresistance, sensitization, combination therapy, antimetabolites, nucleoside analogues, nucleobase analogues.

The peculiarity of cancer [1] is the metastatic potential, so the treatment of malignant diseases usually requires systemic treatment in order to prevent and treat the spread of the tumor. Combined chemotherapy is still a modern model for achieving systemic disease control in clinical oncology, although immunotherapeutic approaches are becoming an important supplement, at least for many patients [2, 3].

Analogues of nitrogenous bases and nucleosides are the main subgroup of antimetabolites, cytotoxic drugs against tumor cells.

Starting from a small area of pediatric oncology, in combination with other chemotherapeutic agents, analogues of nitrogen bases and nucleosides revolutionized clinical oncology and turned cancer into a treatable disease.

All nitrogenous and nucleoside chemotherapy drugs are prodrugs that require chemical modification. Because of this, these compounds interact with many cellular targets and disrupt many cellular processes. Thus, the mode of action of these compounds is multifaceted.

Getting into cancer cells and converting their active forms into metabolites, analogues should be of sufficient concentration and in undecomposed form. They have a direct impact on cell replication and DNA synthesis. Thus, analogues of nitrogenous bases and nucleosides slow down the growth of tumor cells.

However, factors such as deficiency of intracellular delivery of nucleoside analogues, toxicity of nitrogen base analogues and production of protective devices by cells reduce the effectiveness of nitrogen base and nucleo-side analogues against tumor cells.

To increase the resistance of nitrogen bases to tumor cells, it is necessary to glycosylate them using purine and pyrimidine pathways.

So the lack of efficacy can be caused by pharmacokinetic, metabolic, and pharmacodynamic levels and be further complicated by underlying internal tumor biochemistry, there are many possible treatment regimens or a change in therapeutic modality. However, understanding the net effect of a predicted therapy outcome specific to the patient and tumor is a huge challenge.

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