

Air pollution may affect some plausible biological mechanisms that could explain some of the exacerbation of CVD morbidity and mortality. Air pollution exposure may increase systemic cytokine-mediated inflammation and prothrombotic activity. In susceptible people, ultrafine particles were able to provoke alveolar inflammation, with the release of mediators capable of increasing blood coagulability. Increased plasma viscosity is a potential mechanism explaining why high fibrinogen levels are related to increased CVD risk. Similarly, elevated C-reactive protein, ICAM-1, and VCAM-1 levels have been associated with inflammation and cardiovascular risk. An increase in C-reactive protein may reflect arterial damage from white blood cell invasion and inflammation within the wall due to air pollution exposure, thus inducing cardiovascular events.

PM exposure effect on markers of coagulation, inflammation and endothelial function. This association should be modified by race, sex, and age. The question about the most susceptible people is still not answered.

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DYNAMICS OF INDICATORS ANTIOXIDANT PROTECTION IN PATIENTS WITH CHRONIC HEPATITIS DURING THE COMPREHENSIVE TREATMENT WITH INCLUSION “HEPTRAL” BELONGS TO DISEASE WITH CHRONIC HEPATITIS

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The aim of our study was to study the effect of “Heptral” on the results of treatment of patients with chronic hepatitis non-viral origin. 41 patients with chronic hepatitis aged from 22 to 75 ($51,3 \pm 14,5$) have been explored. According to the treatment, patients are divided into two groups. The basic group consisted of 21 patients, whom together with standard treatment prescribed pills “Heptral” 1 tablet three times a day 30 minute before meal for 15-18 days. The group for compare were 20 patients with chronic hepatitis non-viral origin, who received the standard treatment. The group for check up were 20 practically healthy volunteers. We researched the concentration in the blood of the reaction products thiobarbituric acid content of glutathione in the blood, activity of catalase, glutathione peroxidase.

As a result of research discovered a significant increase in the concentration of reduced glutathione during treatment in patients who additionally received “Heptral”. They had contents of reduced glutathione after treatment higher by 26,1% ($p < 0,05$) in compare with contents before treatment. The trend to reduced activity of glutathione peroxidase observed during treatment in both groups of patients, but it was not credible. Blood catalase activity significantly increased after treatment in patients who took “Heptral” on average by 20,4% ($p < 0,05$) in compare with that before treatment, in patients of the group of compare – by 13,8% ($p < 0,05$). After treatment we could see decrease of concentration of reaction products of thiobarbituric acid in patients of both group, more reduction of their content noted in patients, whom to complex treatment was included “Heptral”.

During two weeks of treatment better antioxidant status was adjusted in patients with chronic hepatitis, whom in addition to standard treatment took “Heptral”. For full correction of the clinical manifestations of the disease and antioxidant status should follow the chosen schemes of treatment as the maintenance dose to begin of stable remission in outpatient stage.

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COLCHICINE EFFICACY AND SAFETY FOR THE TREATMENT WITH ISCHEMIC HEART DISEASE: A SYSTEMATIC REVIEW AND META-ANALYSIS OF RANDOMIZED TRIALS

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Colchicine, an anti-inflammatory drug that has been used in rheumatology for a long time to treat gout and prevent seizures; it was firstly presented in cardiology to reduce the recurrence rate of

pericarditis. Currently, the attention of cardiologists to colchicine has again been attracted due to the study of the role of systemic inflammatory response in the development of atherothrombotic cases.

The aim of the study was to investigate the effectiveness of colchicine in inhibiting the activity of the inflammatory process in the pathogenesis of ischemic heart disease (IHD) and its acute and chronic forms. The study of the PubMed database for relevant research in the literature has been conducted. Articles on the mechanism of action of colchicine and clinical applications in IHD have been identified and reviewed.

The most studied mechanism of action of colchicine is its ability to bind to tubulins, thus blocking the formation and polymerization of microtubules. In addition, studies have shown that the biological effects of colchicine are dose-dependent (different effects occur at different concentrations of colchicine) and are directly related to the effects of colchicine on cell migration, cytokine release and intracellular movement, which play an important role in cell dysfunction which are involved in the development of inflammation. The effectiveness of prophylaxis colchicine for the prevention the risk of cardiovascular complications in patients with chronic and acute ischemic heart disease has been demonstrated in clinical trials LoDoCo (Low-Dose Colchicine trial) and COLCOT (CoLchicine Cardiovascular OuTcomes Trial). According to a randomized placebo-controlled study of LoDoCo2, colchicine at low doses (0.5 mg/day) reduces the incidence of ischemic complications and the need for revascularization in patients with stable ischemic heart disease. According to 5 randomized controlled trials, long-term use of colchicine reduced the risk of cardiovascular cases for patients with atherosclerosis significantly, as well as similar mortality from non-cardiovascular diseases (compared with placebo). On the other hand, the results of COVERT-MI (Colchicine for Left Ventricular Remeling Treatment in Acute Myocardial Infarction) were reported at the European Society of Cardiology 2021, which did not show the effectiveness of colchicine in the size of the infarction area and revealed an unexpected threefold increase in left ventricular thrombus in the group of colchicine which requires further research among this group of patients. A recent meta-analysis evaluated adverse cases in 14,983 patients. The results showed that the use of colchicine for the treatment of cardiovascular disease is associated with an increased risk of gastrointestinal adverse cases (especially diarrhea) and prevention of drugs taking associated with colchicine-associated adverse cases (mainly in relation to gastrointestinal symptoms), compared with placebo. It should be noted that among patients who have been receiving a lower daily dose (0.5 mg/day) of colchicine over a long period of time (> 6 months), the risk of gastrointestinal adverse cases is similar to placebo.

With the continuous understanding of the mechanisms of atherosclerosis, anti-inflammatory therapy is approaching clinical applications. Current studies have shown that colchicine, as an anti-inflammatory drug, is likely to become a first-line treatment for atherosclerosis and other cardiovascular inflammatory diseases in the future.

Thus, colchicine is an affordable, safe and effective drug that can be successfully used for the secondary prevention of atherosclerotic cardiovascular disease if its tolerability and benefits for the cardiovascular system are confirmed in current clinical trials.

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**THE STATE OF THE SYSTEM OF FREE RADICAL OXIDATION AND THE
ANTIOXIDANT DEFENSE SYSTEM OF THE BODY IN NEWBORNS WITH IMPAIRED
FUNCTIONAL STATE OF THE CARDIOVASCULAR SYSTEM**

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According to the literature, damage to body cells, in particular myocardium, due to hypoxic exposure is caused by the activation of free radical oxidation (FRO) processes, which, with insufficient antioxidant defense components, triggers a cascade of generic oxidative stress (OS) reactions, as a result of which pathological changes occur at the molecular, cellular, tissue, organ and systemic levels. Based on a comprehensive study of the main components of the FRO and the antioxidant defense system (ADS) system, we made a conclusion regarding their significant role as