-9.59 (-14.10–-5.88). However, the differences between chronotypes were significant only between the morning and evening types (p=0.025). In addition, among male students of the morning type, as well as among females, there were no vagotonics (0%), among the arrhythmic type there were 43%, and among the evening type – 44%. On the contrary, among the Russian-speaking male students of the evening type there was no sympathotonic person (0%), among the morning type there were 25% of them, and among the evening type: 29%.

Conclusions. During the study of blood pressure, pulse rate and KI in domestic and international second-year medical university students at 8.00-10.00, morning type demonstrated higher HR and KI compared to evening type. That is, the morning type developed a more pronounced activation reaction, and the evening type showed more elements of dysregulation of the cardiovascular system in the morning hours (an increase in systolic and diastolic pressure against the background of a decrease in heart rate and a vagotonic reaction). Statistically reliable gender and ethnic differences in cardiovascular parameters and autonomic tone of second-year medical university students with different chronotypes were reviled. Despite these differences, both sexes demonstrate a predominance of sympathetic tone in students with the morning chronotype before noon. Since all measurements were carried out at 8.00-10.00 a.m., we consider it appropriate to carry out further 24-Hour ambulatory blood pressure and pulse monitoring in students with different chronotypes.

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BIOMARKERS OF OXIDATIVE STRESS AND ITS IMPACT ON THE DEVELOPMENT OF CARDIOVASCULAR DISEASES

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Relevance. Oxidative stress (OS) is a central pathophysiological mechanism underlying endothelial damage and the development of cardiovascular diseases (CVDs) such as atherosclerosis, hypertension, heart failure, and ischemic heart

disease. OS biomarkers have emerged as essential tools to advance cardiovascular precision medicine. Overall, OS is a central mediator in vascular pathology, and progress in biomarker validation and targeted therapies will be essential to translate current knowledge into effective prevention, diagnosis, and treatment of cardiovascular diseases [1].

The purpose: briefly analyze scientific publications devoted to OS biomarkers and its role in development of CVDs.

Research methods. The electronic database PubMed was used to search for information (https://pubmed.ncbi.nlm.nih.gov). PubMed comprises more than 37 million citations for biomedical literature from MEDLINE, life science journals and online books. Citations may include links to full text content from PubMed Central and publisher web sites. The publications that best matched the research topic were selected for analysis. The search data are presented as of March 31, 2026.

Research results and their discussion. During the literature search in the PubMed database, more than **7** 573 publications on the topic of the study were identified. Over the past 5 years, 2 339 works have been published, including 745 in the last year. The presented data convincingly confirm the relevance of the research topic and the high publication activity of the authors.

Below is a brief overview of the articles selected for analysis.

Oxidative stress (OS) occurs when the concentration of oxidants exceeds the effectiveness of antioxidants in the body, simultaneously forming reactive nitrogen species. OS has been extensively studied in recent years, with a particular emphasis on OS markers and their association with cardiovascular diseases (CVDs), a leading cause of death worldwide. Multiple pathophysiological mechanisms are involved in the development of CVDs. However, OS plays an important role in the formation of reactive oxygen species (ROS). Increased ROS production reduces the bioavailability of nitric oxide (NO), which increases vasoconstriction in arteries and contributes to the development of hypertension. ROS formation is also associated with the development of atherosclerotic plaques [2].

Cardiovascular diseases (CVDs) are the leading cause of global morbidity and mortality, with oxidative stress and redox imbalance playing pivotal roles in their initiation and progression. In this comprehensive review, authors explore the molecular mechanisms by which reactive oxygen and nitrogen species disrupt vascular homeostasis, promote endothelial dysfunction, and drive adverse cardiac remodeling. Authors additionally highlight key redox-sensitive pathways, including NADPH oxidase activation, mitochondrial dysfunction, endothelial nitric oxide synthase uncoupling, and endoplasmic reticulum stress. Emerging OS biomarkers, such as 8-iso-prostaglandin F2α, oxidized low-density lipoprotein, and myeloperoxidase, are critically examined for their diagnostic and prognostic relevance [3].

In recent years, genetic methods have been widely used to study OS biomarkers. Li M. et al. aimed to identify biomarkers of OS and immune-mediated stress in atherosclerosis. Differential gene expression analyses were based on the GSE100927 dataset in the Gene Expression Omnibus. The authors identified 74 differentially expressed genes related to OS. To compare the differences in immune infiltration

levels between atherosclerosis and control samples, the CIBERSORT and WGCNA R packages were used. As a result, 27 differentially expressed immune-related OS genes were identified. This study shows that genes MMP9, ALOX5, NCF2, NCF1, and NCF4 were diagnostic genes of atherosclerosis and associated with OS. The ALOX5 and NCF2 genes may be involved in the formation of the necrotic core in atherosclerosis by regulating macrophage ferroptosis [4].

Oxidative stress is a major contributor to the pathogenesis of cyanotic congenital heart disease (CCHD), yet data in neonates are limited. This study aimed to assess OS markers, thiol/disulfide homeostasis, and heme oxygenase-1 (HO-1) levels in neonates.

Neonates with CCHD exhibit significant OS and impaired antioxidant defense early in life. Suppressed HO-1 expression and disturbed thiol/disulfide balance suggest increased vulnerability to oxidative injury. These biomarkers may serve as early indicators for clinical risk stratification and could guide antioxidant-based therapeutic strategies. This study investigates a comprehensive panel of OS biomarkers, thiol/disulfide balance, offering potential insights for early risk stratification and therapeutic planning. Neonates with CCHD exhibit significant OS and impaired antioxidant defense early. Suppressed HO-1 and disturbed thiol/disulfide balance suggest vulnerability to oxidative injury [5].

Growth Differentiation Factor 15 (GDF-15) is a stress-inducible cytokine that is abundantly expressed in cardiomyocytes, adipose tissue, macrophages, endothelial cells, and vascular smooth muscle cells. The levels of this biomarker increase in response to tissue injury, inflammation, and metabolic stress and are elevated in the presence of primary cardiometabolic diseases, such as type 2 diabetes, obesity, hypertension, atherosclerosis, heart failure, and chronic kidney disease [6].

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