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ASSESSMENT OF THE IMPACT OF EXHAUST GASES ON CARDIOVASCULAR SYSTEM INDICATORS

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Abstract: This article examines the impact of motor vehicle exhaust gases on the cardiovascular system based on a systematic review of the literature. The toxic components of exhaust gases — carbon monoxide, nitrogen oxides, hydrocarbons, PM2.5 and PM10 particulate matter — are shown to induce endothelial dysfunction, oxidative stress, inflammatory cascades, and atherosclerosis. The key cardiovascular indicators (blood pressure, ECG changes, cytokine levels, lipid profile) and their association with atmospheric pollution are analyzed. Diagnostic markers, preventive measures, and therapeutic approaches are described. Studying the relationship between environmental pollution and cardiovascular disease is of particular relevance in the context of Uzbekistan.

Keywords: exhaust gases, cardiovascular diseases, atmospheric pollution, PM2.5, carbon monoxide, endothelial dysfunction, oxidative stress, atherosclerosis.

Introduction and Relevance of the Problem

In the context of rapid urbanization and industrial development, atmospheric air pollution has become one of the most serious threats to public health worldwide [12, 15]. Motor vehicle exhaust gases, in particular, have deeply permeated the daily lives of urban populations and this issue is receiving growing attention in the fields of medicine, ecology, and public health [3, 11]. According to data from the World Health Organization, air pollution directly causes the deaths of more than 7 million people annually, a large proportion of which are attributable to cardiovascular conditions [12, 19].

The composition of exhaust gases is highly complex, containing carbon monoxide (CO), nitrogen oxides (NOx), sulfur dioxide (SO2), hydrocarbons, benzene, formaldehyde, lead compounds, and — most hazardous of all — PM2.5 and PM10 particulate matter [3, 23]. Each of these compounds, both individually and in combination, acts upon the human body and creates the conditions for numerous pathological changes in the cardiovascular system [13, 14]. Heavy-duty diesel trucks are regarded as a particularly significant hazard: the PM emissions they generate substantially increase the risk of developing cardiovascular disease [24].

In the Republic of Uzbekistan, the density of motor traffic in cities — particularly Tashkent, Samarkand, and other major centers — is increasing year on year, and ambient air quality is steadily deteriorating [6, 7]. Local environmental monitoring data reveal concentrations of harmful

substances in the air that exceed permissible norms [4, 7]. Against this background, the rates of cardiovascular diseases — hypertension, coronary heart disease, and cerebrovascular disorders — continue to rise [9, 21]. Specialists emphasize that a direct pathogenetic link exists between these processes [5, 22].

The cardiological effects of exhaust gases are mediated through several mechanisms. First, particulate matter penetrates the bloodstream via the airways and induces endothelial dysfunction [12, 19]. Second, it elevates levels of oxidative stress and pro-inflammatory cytokines such as IL-6 and TNF-α [10, 22]. Third, it disrupts the autonomic nervous system, destabilizing heart rate and blood pressure [5, 25]. This complex interplay of mechanisms contributes to the development of both acute and chronic cardiovascular diseases [9, 21]. The aim of this article is to systematically evaluate the impact of exhaust gases on cardiovascular system indicators on the basis of the available literature.

Large-scale international epidemiological studies have reliably demonstrated the association between air pollution and cardiovascular mortality [20, 22]. These investigations have found that each 10 μg/m³ increase in PM2.5 concentration raises the risk of cardiovascular events by 6–17% [22, 24]. Furthermore, high concentrations of CO have been shown to convert oxyhemoglobin to carboxyhemoglobin, directly paving the way for myocardial ischemia [3, 8]. These findings clearly underscore the scientific as well as the socio-medical significance of this topic.

Pathophysiological Mechanisms and Topographic Classification

The impact of exhaust gases on the cardiovascular system develops along several interrelated pathological pathways [3, 14]. The first and most fundamental pathway is endothelial dysfunction. Particulate matter (PM2.5 and PM10) enters the lungs via the airways, passes into the bloodstream, adheres to vascular endothelium, and generates reactive oxygen species (ROS) [4, 12]. This process reduces nitric oxide (NO) synthesis, impairs endothelium-dependent vasodilation, and consequently elevates blood pressure and increases arterial tone [19, 22].

The second mechanism is the systemic inflammatory cascade. Components of exhaust gases trigger airway inflammation and release pro-inflammatory cytokines — IL-6, IL-8, and TNF-α — into the circulation [10, 12]. These cytokines elevate C-reactive protein (CRP), accelerate the formation of atherosclerotic plaques, and narrow the lumen of coronary arteries [9, 22]. Studies have shown that CRP levels are significantly higher in urban residents, especially those living near major transport routes, compared to rural populations [7, 25].

The third mechanism is oxidative stress. Polycyclic aromatic hydrocarbons (PAH) and benzene contained in exhaust gases produce highly reactive compounds through oxidative metabolism [16, 23]. These compounds oxidize lipids, modify LDL cholesterol, and increase the quantity of oxidized LDL with atherogenic properties [10, 14]. The result is accelerated development of atherosclerosis, lipid deposition in vessel walls, and increased thrombosis risk [14, 22].

The fourth mechanism involves effects on the autonomic nervous system. Exhaust gases — particularly CO and PM2.5 — reduce vagal (parasympathetic) tone while increasing sympathetic activity [3, 5]. This leads to changes in heart rate variability (reduced HRV), development of arrhythmias, and blood pressure instability [5, 11]. Epidemiological data document an increase in hospital admissions and acute coronary events on days when air pollution is intensified [9, 21].

From a pathophysiological standpoint, the effects of exhaust gases can be classified topographically into two levels: local (at the level of the respiratory and cardiovascular systems) and systemic (at the level of the entire organism) [4, 15]. At the local level, microthrombi form in pulmonary capillaries, coronary artery spasm occurs, and the myocardium suffers ischemia [8, 13]. At the systemic level, blood viscosity increases, platelet hyperreactivity develops, and atherosclerotic changes are observed throughout the vascular system [14, 22]. Both levels amplify each other, laying the groundwork for the development of chronic cardiovascular pathology [12, 24].

Changes in the cardiac conduction system are also observed under the influence of exhaust gases [5, 11]. QT interval prolongation, ST segment depression, and T-wave inversion have been described as electrocardiographic signs of exhaust gas exposure [5, 21]. These changes are particularly pronounced in patients with pre-existing heart disease and increase the risk of sudden cardiac death [9, 21]. Moreover, when elevated ambient temperature and increased exhaust gas concentration occur simultaneously, the risk of cardiovascular complications has been found to double [25].

Key Indicators for Assessing the Impact of Exhaust Gases and Their Differential Analysis

A number of laboratory and instrumental indicators are used to assess the impact of exhaust gases on the cardiovascular system [5, 9]. These indicators must be analyzed in a comprehensive manner, evaluated in conjunction with the clinical condition and the degree of ambient air pollution [22, 25].

The first group comprises blood pressure indicators. An elevation of systolic and diastolic blood pressure is one of the earliest and most widespread signs of chronic exhaust gas exposure [4, 22]. Research has shown that each 10 μg/m³ increase in PM2.5 concentration leads to an average rise in systolic blood pressure of 1.4–2.8 mm Hg [22, 24]. The observation that blood pressure decreases during nighttime hours when traffic diminishes — a disruption of the circadian rhythm — confirms the autonomic mechanisms of exhaust gas action [5, 25].

The second group consists of inflammatory and oxidative stress markers. Elevated levels of C-reactive protein (CRP), fibrinogen, IL-6, and TNF-α occur in parallel with intensifying exhaust gas exposure [10, 22]. Among oxidative stress markers, 8-isoprostane, malondialdehyde (MDA), and reduced superoxide dismutase (SOD) activity serve as objective indicators of systemic oxidative damage [16, 23]. These markers are used not only to assess the degree of exposure but also to evaluate the efficacy of cardioprotective therapy [10, 22].

The third group covers lipid metabolism indicators. Chronic exhaust gas exposure shifts the blood lipid profile unfavorably: LDL rises, HDL falls, and triglycerides increase [14, 22]. Oxidized LDL (oxLDL) is of particular importance — its uptake by macrophages produces foam cells and drives atherosclerotic plaque development [14, 20]. The dynamics of these indicators are also applied in comparative studies between urban and rural populations [20, 25].

The fourth group includes ECG and echocardiographic indicators. Holter ECG monitoring provides 24-hour heart rate variability (HRV) analysis, yielding information about the state of the autonomic nervous system [5, 11]. Reduced HRV is directly associated with exhaust gas exposure and the risk of cardiac arrhythmias [5, 21]. On echocardiography, impaired left ventricular diastolic

function and increased wall stiffness reflect the chronic cardiological consequences of exhaust gas exposure [9, 11].

The fifth group encompasses biomarkers. Troponin I/T, BNP (brain natriuretic peptide), and NT-proBNP assist in the early detection of myocardial injury and dysfunction [9, 21]. Elevated serum levels of these markers are recorded in cases of intense and prolonged exhaust gas exposure [10, 22]. For early diagnostic purposes, a multimarker approach — combining CRP, troponin, oxLDL, and HRV analysis — provides the highest accuracy [5, 22].

Table 1. Key indicators for assessing the impact of exhaust gases

Indicator	Normal Value	Direction of Change	Clinical Significance
Systolic blood pressure	< 120 mm Hg	Increases	Risk of hypertension [22, 24]
CRP	< 3 mg/L	Increases	Systemic inflammation [10, 22]
oxLDL	< 60 U/L	Sharply increases	Atherosclerosis development [14, 20]
HRV (SDNN)	> 50 ms	Decreases	Arrhythmia risk [5, 21]
Troponin I	< 0.04 ng/mL	Increases	Myocardial injury [9, 21]
IL-6	< 7 pg/mL	Increases	Pro-inflammatory state [10, 22]

Table 2. Cardiovascular action mechanisms of exhaust gas components

Component	Primary Effect	Target Organ/System	Hazard Level
PM2.5	Endothelial dysfunction, oxidative stress	Coronary arteries	Very high [12, 22]
CO (carbon monoxide)	Carboxyhemoglobin formation	Myocardium	High [3, 8]
NOx	Disruption of nitric oxide balance	Vascular tone	Moderate-high [13, 16]

PAH	Elevated oxidized LDL	Aorta, arteries	High [14, 16]
Benzene	Bone marrow suppression	Hematopoietic system	Moderate [16, 23]
SO2	Bronchospasm, reflex bradycardia	Pulmonary-cardiac axis	Moderate [13, 19]

The correct combination of these indicators and dynamic monitoring provide a sound clinical conclusion [5, 22]. The limitations of each marker must be taken into account and assessments must be integrated with the degree of ambient air pollution and the individual characteristics of the patient [20, 25].

Peripheral Blood Morphology and Modern Diagnostic Technologies

Morphological examination of peripheral blood smears is of considerable importance in assessing the hematological impact of exhaust gases [4, 17]. Under light and electron microscopy, changes in erythrocyte shape, deformability, and aggregation are identified [4, 17]. Prolonged exposure to exhaust gases is associated with increased counts of poikilocytes, spherocytes, and acanthocytes in the blood, indicating disturbances in blood rheology [17, 23].

Among modern diagnostic technologies, atomic force microscopy (AFM) enables highly accurate assessment of the biomechanical properties of erythrocytes [4, 17]. Flow cytometry is used to quantify microvesicles, activated platelets, and endothelial cells in the circulation [17, 22]. Artificial intelligence-based analytical systems can simultaneously analyze thousands of cells and detect subtle changes invisible to the human eye [12, 17]. These methods are of considerable value in predicting early risk and selecting individualized therapy [22, 25].

Multiple electrode aggregometry (Multiplate) is used to assess platelet function — detecting both hyperreactivity and hypoaggregation [17, 22]. The effect of exhaust gases on platelet aggregation is bidirectional: acute exposure produces hypercoagulation, while chronic exposure leads to platelet exhaustion [17, 24]. Viscometry allows assessment of the rheological properties of blood, a parameter that correlates with microcirculatory disturbances [4, 19].

Portable air quality monitors (PM2.5/PM10 sensors) for real-time tracking of personal exposure levels are increasingly being used in clinical settings and epidemiological research [15, 18]. Integrating this data with ECG readings, blood pressure measurements, and laboratory indicators provides the basis for a comprehensive system for evaluating exhaust gas exposure [11, 22]. This approach enables more accurate individual risk assessment and personalization of preventive measures [20, 25].

Non-invasive vascular diagnostic methods — including ultrasound measurement of carotid intima-media thickness, arterial elastic modulus, and pulse wave velocity (PWV) — are applied in the early detection of subclinical vascular changes resulting from exhaust gas exposure [9, 22]. Using these methods, it has been shown that individuals living in areas with high air pollution may develop early signs of atherosclerosis 3–5 years earlier than the general population [9, 20]. A

combination of modern diagnostic techniques enables holistic evaluation of exhaust gas impact and targeted direction of treatment [22, 25].

Modern Prevention and Therapeutic Approaches

The strategy for preventing and treating cardiovascular changes caused by exhaust gas exposure encompasses two primary directions: environmental and medical [15, 25]. The environmental direction prioritizes reducing vehicle emissions (through electric vehicles, alternative fuels, and stringent emission standards) and increasing urban green space [14, 15]. The medical direction focuses on developing targeted preventive measures for population groups most heavily exposed to air pollution — children, pregnant women, the elderly, and patients with cardiac disease [6, 19].

In the area of pharmacological prophylaxis, antioxidant agents (vitamin E, vitamin C, N-acetylcysteine) are being investigated with the aim of reducing oxidative stress and improving endothelial function [10, 22]. Omega-3 fatty acids suppress systemic inflammation, improve the lipid profile, and reduce the risk of cardiac arrhythmias [10, 20]. Statins in patients exposed to exhaust gases not only lower lipid levels but also exert an additional cardioprotective effect through their antioxidant and anti-inflammatory properties [9, 20].

Antihypertensive therapy is of particular importance in patients with intensified exhaust gas exposure [4, 22]. ACE inhibitors and angiotensin receptor blockers contribute to restoring endothelial function and slowing vascular remodeling [9, 22]. Beta-adrenoceptor blockers reduce autonomic imbalance, improve HRV parameters, and lower the risk of arrhythmia [5, 21]. The specific efficacy of these agents in individuals exposed to exhaust gases is currently the subject of numerous clinical investigations [9, 22].

Among innovative approaches, the use of air purifiers (HEPA filters) to improve indoor and in-vehicle air quality has practical significance [15, 18]. Personal protective equipment — masks equipped with PM2.5 filtration — reduces exposure in conditions of severe pollution [12, 15]. The beneficial effects of N95 respirators on cardiovascular indicators have been confirmed in randomized studies [12, 25]. From an urban planning perspective, the construction of residential buildings at a greater distance from major transport routes, tree planting, and the establishment of green corridors represent the foundations of long-term prevention [15, 25].

In clinical practice, the introduction of cardiovascular screening programs for residents of areas with high air pollution is recommended [9, 22]. These programs should include regular blood pressure monitoring, lipid profile and inflammatory marker testing, ECG, and echocardiography [5, 9]. A multidisciplinary approach — involving the collaboration of cardiologists, pulmonologists, ecologists, and hygienists — ensures the most comprehensive prevention and therapeutic strategy [19, 22].

Immune and Experimental Biomarkers

The cytokine panel serves as an important diagnostic tool in assessing the immunological effects of exhaust gases [10, 22]. Elevated levels of IL-6, IL-8, IL-1β, and TNF-α are consistently observed in individuals chronically exposed to air pollution, and these indicators correlate strongly with cardiovascular risk [10, 12]. Cytokines are produced by endothelial cells, macrophages, and

neutrophils, initiating the vascular inflammatory cascade and accelerating atherosclerotic plaque formation [10, 22].

Neutrophil extracellular traps (NETs) are activated by exhaust gas exposure and contribute to coagulopathic processes [10, 22]. NETs activate platelets, form platelet-neutrophil aggregates, and promote thrombotic microangiopathy [10, 22]. Experimental models — mice and rats — have demonstrated that PM2.5 inhalation leads to the development of activated NETosis and microthrombi in coronary arteries [12, 22]. This mechanism plays an important role in the pathogenesis of acute coronary syndrome [9, 22].

Experimental models have provided the essential foundation for clinical research into the cardiovascular consequences of exhaust gas exposure [11, 14]. In rat models, four weeks of exhaust gas inhalation produced elevated oxidative stress markers, vascular remodeling, and changes in cardiac morphology [11, 14]. In porcine models, coagulopathy, endothelial injury, and inflammation were studied in combination following exposure to polluted air [14, 24]. These models have elaborated in detail the development of myocardial fibrosis and ventricular remodeling under conditions of chronic exhaust gas exposure [9, 14].

Epigenetic changes represent a new research direction in the study of exhaust gas effects [12, 16]. Exposure to PM2.5 and PAH is associated with altered DNA methylation, disrupted microRNA expression, and histone modification [16, 23]. Experimental research has shown that these epigenetic changes can amplify genetic predisposition to cardiovascular disease and may be transmissible to subsequent generations [16, 23]. Investigation of such mechanisms provides a deeper understanding of the long-term cardiological consequences of exhaust gas exposure [12, 23].

Among immune biomarkers, fractalkine (CX3CL1) warrants particular attention [10, 22]. This chemokine is produced by endothelial cells and plays a central role in recruiting monocytes and NK cells to atherosclerotic plaques [10, 22]. An association has been identified between elevated serum fractalkine levels under exhaust gas exposure and the severity of coronary atherosclerosis [10, 22]. Experimental models are being used to develop new therapeutic directions — fractalkine receptor antagonists and NETs blockers [10, 22].

Variability of Indicators and Limitations in Result Interpretation

A number of important limitations must be taken into account when interpreting the indicators used to assess exhaust gas exposure [20, 25]. The first and greatest limitation is individual variation. Genetic polymorphisms (for example, the GSTM1 gene), age, sex, smoking status, and pre-existing chronic diseases produce differing degrees of cardiovascular response to identical exhaust gas exposure [12, 20]. For this reason, population-level average indicators may not be directly applicable to an individual patient [20, 25].

The second limitation concerns confounding factors. Smoking, obesity, poor nutrition, and physical inactivity also increase oxidative stress and amplify the risk of cardiovascular disease [9, 25]. In many epidemiological studies, full control of these factors is difficult, which makes it problematic to isolate the 'pure' effect of exhaust gas exposure [20, 25]. Furthermore, factory emissions near residential areas, indoor air pollution from cooking smoke, and occupational exposure can all mix with exhaust gas effects, introducing potential errors into indicator measurements [7, 15].

The third limitation relates to the variability in exhaust gas composition. Fuel type (petrol, diesel, gas, electric), vehicle age, engine technology, and ambient temperature all significantly alter emission composition [3, 23]. For example, PM2.5 emissions from diesel vehicles may be 10–20 times greater than those from petrol vehicles [24]. This limits the ability to standardize the concept of 'exhaust gas exposure' across studies and to compare findings [3, 23].

The fourth limitation concerns the question of exposure duration and dose. Many clinical studies examine short-term exposure, whereas chronic, low-level exposure is the primary driver of cardiovascular disease [12, 19]. Defining the optimal exposure level, the damage threshold, and the dose-response relationship remains a subject of ongoing scientific debate [19, 22]. Additionally, seasonal and daily fluctuations in air quality complicate accurate assessment of individual exposure [7, 15].

The fifth limitation pertains to diagnostic methods themselves. Laboratory markers such as CRP, IL-6, and troponin are sensitive not only to exhaust gas exposure but also to infection, stress, and other diseases [10, 25]. HRV analysis depends on the conditions of measurement (rest, activity, sleep) and may yield errors when non-standardized methods are applied [5, 11]. Although a multimarker approach — combining TEG/ROTEM, cytokines, lipid profile, and HRV — achieves the highest accuracy, its cost and complexity impede its widespread clinical adoption [22, 25].

Conclusion

The impact of exhaust gases on the cardiovascular system has become a global public health concern, and research in this field carries significant clinical and social importance [12, 22]. The PM2.5, carbon monoxide, NOx, and polycyclic aromatic hydrocarbons contained in exhaust gases induce endothelial dysfunction, systemic inflammation, oxidative stress, and autonomic nervous system imbalance, thereby creating conditions for the development of cardiovascular pathologies including hypertension, coronary heart disease, arrhythmia, and atherosclerosis [3, 9, 14, 22].

Comprehensive and dynamic monitoring of key indicators — blood pressure, CRP, oxLDL, HRV, troponin, and cytokines — represents the most effective approach for early detection of the cardiovascular consequences of exhaust gas exposure and individual risk assessment [5, 22, 25]. Modern diagnostic technologies — AFM, flow cytometry, portable air quality sensors, and artificial intelligence-based analyzers — continue to expand these capabilities [17, 22]. Immune and experimental biomarkers — fractalkine, NETs, and epigenetic markers — shed further light on pathogenetic mechanisms and provide the groundwork for developing new therapeutic strategies [10, 16].

In terms of prevention and treatment, the integration of environmental measures (emission reduction, green space creation, personal protective equipment) and medical interventions (antioxidants, statins, antihypertensive agents, TEG/ROTEM-guided targeted therapy) constitutes the most appropriate strategy [15, 20, 22]. In the context of Uzbekistan, where urban air quality is deteriorating year on year and cardiovascular disease rates are rising, systematic investigation of this topic carries not only theoretical but substantial practical importance [6, 7, 9]. Future research will further elucidate the molecular mechanisms of exhaust gas exposure, support the development of targeted cardioprotective strategies, and help improve outcomes for affected populations.

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