

Review

Air Pollution and Vascular Biomaterial Remodeling

Kšela Juš^{1,3,*}, Lakić Nikola¹, Stefanovski Dime¹, Racman Mark¹, Amon Mojca², Knežević Ivan²

¹. University Medical Centre Ljubljana, Division of Cardiovascular Surgery, Ljubljana, Slovenia

². University Medical Centre Ljubljana, Surgery Department, Ljubljana, Slovenia

³. University of Ljubljana, Faculty of Medicine, Department of Surgery, Ljubljana, Slovenia;

* Correspondence: Kšela Juš; ksel.jus@kclj.si,

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Abstract:

Air pollution is increasingly recognised as an independent cardiovascular risk factor. Fine particulate matter and ultrafine particles can transport toxic elements and metalloids, promoting systemic inflammation, oxidative stress, endothelial dysfunction, and vascular injury. Toxic elements such as cadmium, lead, mercury, arsenic, nickel, and chromium may contribute to vascular damage, while essential trace elements, including selenium, zinc, copper, manganese, and molybdenum, support redox balance and cellular homeostasis. However, the role of toxic-essential element imbalance in cardiovascular disease remains insufficiently understood.

Recent translational studies combine environmental exposure assessment, human biomonitoring, and vascular phenotyping. Exposure is typically evaluated using environmental data together with biomarkers measured in blood and urine. Cardiovascular status can be assessed through non-invasive vascular measurements and, where clinically indicated, analysis of vascular tissue obtained during surgery. Advanced analytical methods enable quantification of toxic and essential elements and evaluation of their associations with vascular alterations.

Chronic exposure to air pollution-related toxic elements is expected to be associated with endothelial dysfunction, inflammation, and early vascular remodeling. Imbalances between toxic and essential elements may correlate with the severity of vascular injury. Combining biomonitoring with tissue-level analysis may therefore provide new mechanistic insights and support the identification of early biomarkers of environmentally driven vascular damage.

Overall, integrating environmental exposure assessment, biomonitoring, and vascular tissue analysis offers a promising framework for understanding environmentally related cardiovascular disease. Considering both toxic and essential elements may improve cardiovascular risk assessment, early detection, and the development of targeted preventive strategies.

Keywords: Air pollution; Cardiovascular disease; Toxic elements; Essential elements; Trace elements; Endothelial dysfunction; Vascular pathology; Biomonitoring; Environmental health

1. Environmental Exposure and Cardiovascular Risk

Air pollution is increasingly recognized as an important independent cardiovascular risk factor. Exposure to airborne particulate matter and environmental toxicants may promote systemic inflammation, oxidative stress, endothelial dysfunction, and early vascular injury. Particulate matter (PM) comprises microscopic solid particles and liquid droplets generated from traffic emissions, industrial activities, combustion processes, and environmental dust. According to aerodynamic diameter, PM is categorized into coarse particles ($PM_{10} \leq 10 \mu m$), fine particles ($PM_{2.5} \leq 2.5 \mu m$), and ultrafine particles ($< 0.1 \mu m$). Due to their small size, fine and ultrafine particles can penetrate deeply into the respiratory tract and enter the circulation, where they contribute to vascular and cardiovascular damage.

Airborne particles frequently carry toxic metals and metalloids, including cadmium, lead, mercury, arsenic, nickel, and chromium. Chronic exposure to these elements may result in tissue accumulation and cellular dysfunction, thereby enhancing vascular injury and cardiometabolic risk. In contrast, essential trace elements such as selenium, zinc, copper, manganese, and molybdenum are involved in antioxidant defence, redox regulation, and cellular homeostasis. Evaluating the interaction between toxic and essential elements may therefore improve understanding of environmentally driven cardiovascular disease and support earlier risk detection and prevention.

2. Biomonitoring of Toxic and Essential Elements

Environmental exposure assessment and biomonitoring are increasingly used to investigate the cardiovascular effects of air pollution and toxic elements. Epidemiological evidence suggests that ambient and household air pollution contribute substantially to global cardiovascular morbidity and mortality, accounting for millions of premature deaths annually (Liang et al., 2020; Münzel et al., 2021). These environmental exposures are now considered relevant alongside established cardiovascular risk factors such as hypertension, diabetes, dyslipidaemia, obesity, and smoking.

Fine particulate matter and ultrafine particles represent particularly harmful components of polluted air because they contain complex mixtures of organic compounds, reactive gases, and metal-associated contaminants. Following inhalation, these particles may directly translocate into the bloodstream or trigger pulmonary inflammatory and oxidative responses that subsequently affect the vascular system. Such mechanisms have been associated with endothelial dysfunction, vascular inflammation, autonomic imbalance, arterial stiffness, thrombosis, and accelerated atherosclerosis (Finch & Conklin, 2016; Liang et al., 2020).

Biomonitoring approaches based on blood, urine, and tissue analysis enable quantitative assessment of cumulative exposure to toxic and essential elements. Combined with vascular phenotyping and environmental exposure data, these methods may provide valuable mechanistic insight into environmentally related cardiovascular injury and facilitate the identification of early biomarkers of vascular dysfunction.

3. Vascular Remodeling and Endothelial Dysfunction

Endothelial dysfunction is increasingly considered one of the earliest and most important pathophysiological mechanisms linking environmental exposure to cardiovascular disease. The vascular endothelium plays a central role in maintaining vascular homeostasis through regulation of vasodilation, inflammation, thrombosis, oxidative balance, and vascular remodelling. Under physiological conditions, endothelial cells preserve nitric oxide (NO) bioavailability, inhibit inflammatory activation, and prevent platelet aggregation and leukocyte adhesion. However, exposure to air pollutants and toxic metals may disrupt endothelial homeostasis through excessive reactive oxygen species (ROS) production, impaired endothelial nitric oxide synthase (eNOS) activity, and activation of pro-inflammatory signalling pathways. Reduced NO bioavailability together with increased oxidative stress contributes to impaired vasodilation, vascular stiffness, inflammation, and progressive vascular injury (Liang et al., 2020; Finch & Conklin, 2016; Münzel et al., 2018; Bayo, 2023).

4. Translational Approaches to Environmentally Driven Cardiovascular Disease

Experimental and clinical studies have demonstrated that exposure to $PM_{2.5}$ is associated with increased arterial stiffness, elevated blood pressure, impaired endothelial-dependent

vasodilation, progression of carotid intima-media thickness, and enhanced atherosclerotic plaque formation. Air pollution has also been linked to elevated circulating inflammatory mediators, endothelial adhesion molecules, and oxidative stress biomarkers, which promote monocyte recruitment, macrophage activation, lipid accumulation, and vascular remodelling, thereby contributing to the initiation and progression of atherosclerosis (Liang et al., 2020; Finch & Conklin, 2016).

Toxic elements represent an additional and increasingly recognized environmental cardiovascular risk factor. Unlike essential trace elements required for normal physiological processes, non-essential metals such as cadmium, mercury, arsenic, and lead have no known biological role and may exert harmful effects even at low concentrations. Chronic exposure may lead to tissue accumulation and disruption of antioxidant defence systems, mitochondrial function, lipid metabolism, and endothelial signalling pathways. These mechanisms have been associated with hypertension, coronary artery disease, stroke, heart failure, and accelerated vascular ageing, while recent systematic reviews and meta-analyses further support the relationship between toxic element exposure and cardiovascular risk (Nucera et al., 2024; Cheema et al., 2025).

In contrast, essential trace elements including selenium, zinc, copper, manganese, and molybdenum contribute to antioxidant defence, cellular metabolism, and redox homeostasis. Disturbances in the balance between toxic and essential elements may therefore influence vascular integrity and susceptibility to cardiovascular injury. Despite increasing evidence linking environmental exposures with cardiovascular disease, the complex interactions between toxic and protective trace elements remain insufficiently understood.

5. Conclusions

Current evidence highlights the need for integrated translational approaches that combine environmental exposure assessment, biomonitoring, vascular phenotyping, and direct tissue-level analysis. Such strategies may improve understanding of the mechanisms underlying environmentally driven vascular injury and support the identification of early biomarkers of endothelial dysfunction and vascular remodeling. Ultimately, a deeper understanding of environmental cardiovascular risk factors may contribute to earlier detection, improved risk stratification, and the development of targeted preventive and therapeutic strategies for environmentally exposed populations.

Conflicts of Interest: The authors declare no conflict of interest.

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