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MICRO- AND NANOPLASTICS IN THE CARDIOVASCULAR SYSTEM: CURRENT EVIDENCE, RESEARCH GAPS: A SYSTEMATIC REVIEW

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A – study design, **B** – data collection, **C** – statistical analysis, **D** – interpretation of data, **E** – manuscript preparation, **F** – literature review, **G** – sourcing of funding

ABSTRACT

Background: Microplastics and nanoplastics (MP-NPs) are major environmental pollutants that have gained attention due to their widespread presence in water, air, soil, and food. Their entry into the human body through inhalation, oral ingestion, or skin contact raises concerns about potential health risks, particularly cardiovascular effects.

Aim of the study: This systematic review aims to evaluate the impact of MP-NPs on the cardiovascular system and their associated health consequences.

Material and methods: A systematic literature search was conducted in PubMed using the following keywords: „microplastics," „nanoplastics," „cardiovascular system," „cardiotoxicity," and „environmental pollution" in various combinations. Studies published between 2015 and 2025 were included, focusing on both animal and human data.

Results: The review followed PRISMA guidelines, and 29 studies were included after applying predefined eligibility criteria. Most studies were conducted on animals, demonstrating that MP-NPs affect heart function through cellular and structural changes. In rat and mouse models, exposure to MP-NPs led to mitochondrial damage, myocardial fibrosis, increased pro-inflammatory markers, altered glucose and insulin levels, and weight gain. Human data remain limited; however, MP-NPs have been detected in atherosclerotic plaques, with emerging evidence suggesting their contribution to plaque formation and lipid metabolism alterations. These mechanisms may increase the risk of myocardial infarction and stroke.

Conclusions: Current evidence suggests that exposure to micro- and nanoplastics (MP-NPs) may negatively affect the cardiovascular system, particularly in experimental models. Animal studies consistently indicate structural and functional myocardial changes, including fibrosis, mitochondrial damage, and metabolic disturbances. However, these findings cannot be directly extrapolated to humans. Human data remain scarce

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and mostly observational, showing the presence of MP-NPs in atherosclerotic plaques and thrombi, with correlations to adverse cardiovascular outcomes.
Given the methodological heterogeneity and high risk of bias across available studies, the overall strength of evidence remains low. Therefore, MP-NPs should currently be regarded as a potential – but not yet proven – cardiovascular risk factor.

KEYWORDS: microplastics, nanoplastics, cardiovascular system, cardiotoxicity, environmental pollution

Background

Microplastic is defined by the World Health Organization (WHO) as a material composed of solid polymers and other additives, in which at least 1% of the particle mass falls within the size range of 1 nm to 5 mm. In the case of fibers, at least 1% of the particle mass should have a length between 3 nm and 15 mm and a length-to-diameter ratio greater than 3. Nanoplastic, on the other hand, refers to plastic particles ranging in size from 1 nm to <1000 nm. The mass production of plastics on a global scale has resulted in the widespread presence of microplastics and nanoplastics (MP-NPs) in the environment [1].

MP-NPs enter the human body through oral ingestion, inhalation, or skin contact, affecting its overall functioning. In recent years, numerous studies have focused on the impact of MP-NPs on various body systems, including the cardiovascular system. Current research indicates several pathomechanisms leading to structural and functional changes in the heart.

Cardiovascular diseases remain the leading cause of mortality, accounting for approximately 17.9 million deaths annually, with as many as one-third occurring before the age of 70. In the face of ongoing technological progress and the emergence of new environmental threats, it is crucial to promptly identify potential risk factors for cardiovascular diseases. Since the cardiovascular system is in direct contact with polymer particles and transports them throughout the body, it appears to be particularly vulnerable to the toxic effects of microplastics. It is estimated that in 2019, environmental pollution was responsible for 5.5 million deaths related to cardiovascular diseases. The link between exposure to pesticides, heavy metals, and synthetic chemicals and an increased cardiovascular risk is well established. However, recent reports suggest that among pollutants posing a threat to human health and life, micro- and nanoplastic particles should also be considered [2].

MP-NPs are present in water, air, soil, and even food. Their exact quantity is difficult to estimate due to variations in measurement methods and sampling locations. A 2019 WHO report, after reviewing nine studies on both tap and bottled drinking water, determined that microplastic levels in drinking water ranged from 0 to 104 particles per liter. The most commonly found types of microplastics in drinking water included polystyrene, acrylonitrile-butadiene-styrene (ABS), polyethylene, and polyethylene terephthalate (PET) [3]. It is also important to note that exposure to micro- and nanoplastics may result not only from environmental sources such as air and water but also from personal lifestyle choices and consumer habits.

Similar to water concentration assessments, estimating the amount of microplastic in the air also poses challenges. Microplastic particles are present in both indoor and outdoor air, with varying concentration levels depending on location, activities, and measurement methods. A study conducted in three Danish households found microplastic concentrations ranging from 1.7 to 16.2 particles/m^3 of air, with polyester, polyethylene, nylon, and polypropylene being the dominant polymers [4].

Microplastic penetration through the skin is limited due to the presence of an intact stratum corneum. For particles larger than 1 μm, transdermal absorption is particularly difficult. However, an alternative pathway exists – nanoplastic particles up to 200 nm in size can penetrate through hair follicles and reach deeper layers of the skin [5], [6].

The presence of microplastics in air, water, and soil leads to their accumulation in organisms that are subsequently consumed by humans. Additionally, storing and heating food in plastic containers using microwaves or ovens further increases the amount of microplastics in ingested meals [1].

While the environmental presence of MP-NPs is well documented, their specific impact on cardiovascular health remains unclear. Limited epidemiological data and the lack of standardized methodologies for detecting microplastics in human tissues pose significant challenges.

Aim of the study

The aim of this study is to review research focusing on the impact of MP-NPs on the cardiovascular system and the health consequences associated with exposure to MP-NPs. The objectives are to distinguish between findings from animal and human studies, summarize the main mechanisms

suggested by experimental data, and highlight the limitations of current evidence with regard to clinical translation.

To conduct a detailed review of cardiovascular effects, two subsections were established, focusing on myocardial function and vascular integrity, including thromboembolic events and platelet function.

Material and methods

Search strategy

This review is based on scientific publications obtained through a comprehensive search strategy. The following keywords were used: "microplastics" OR "nanoplastics" AND "cardiotoxicity" OR "cardiovascular system" AND "environmental pollution." Searches were conducted in PubMed. Additionally, the review was supplemented with the latest WHO reports on microplastic pollution. Ultimately, twenty-nine articles in English were selected, emphasizing relevant information on the topic and published between 2015 and 2025.

Eligibility criteria

Studies meeting the following criteria were included in this systematic review:

- Population: Animals (mainly rats and mice) and humans with cardiovascular diseases, in which the impact of microplastics on the cardiovascular system was investigated.
- Results: Information on the impact of microplastics on the cardiovascular system, including structural changes in the heart, alterations in heart function, changes in cardiac biomarker levels, and related biological mechanisms.
- Language: Publications written in English.

The exclusion criteria were as follows: studies examining the effects of microplastics on other organ

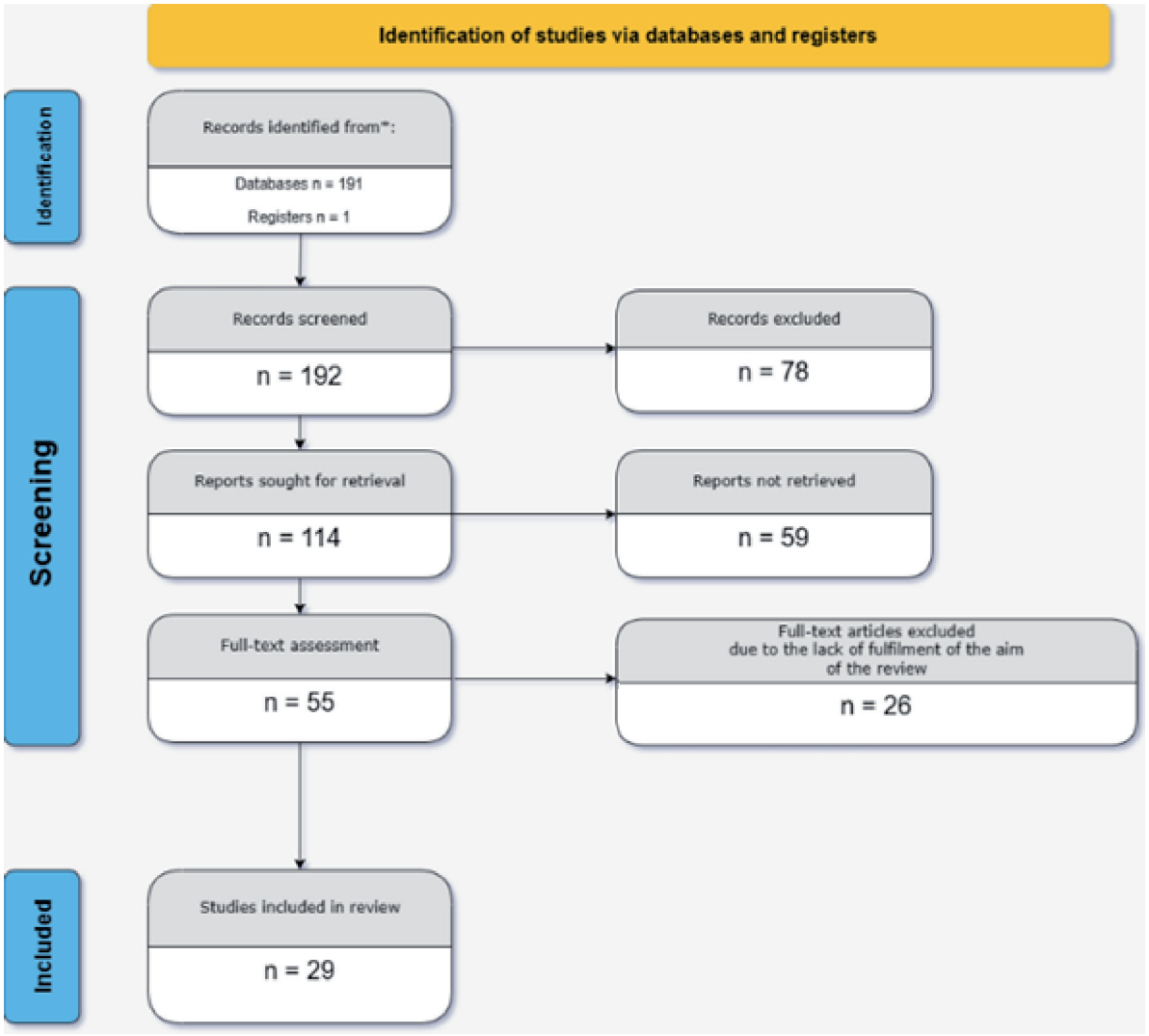

Figure 1. Flow chart of study selection

systems, studies unrelated to microplastics or nanoplastics, and publications without full-text access.

Data collection

The literature review was conducted independently by each author. Initially, titles and abstracts of the identified articles were analyzed to assess their relevance to the topic of the review. Articles meeting the inclusion criteria were selected for full-text analysis. The studies were evaluated based on their content, including the type of microplastics, administration methods, duration of exposure, and outcomes related to the cardiovascular system. The collected data were verified by the authors to ensure consistency and accuracy.

This review was not prospectively registered in PROSPERO or a similar database. However, the inclusion and exclusion criteria, as well as the data extraction and quality assessment methods, were defined a priori and consistently applied.

The risk of bias and methodological quality of included studies were assessed qualitatively. This information was summarized in Table 1.

Results

Impact of microplastics on the heart

The effects of microplastics on the cardiovascular system have been the subject of numerous recent studies. Currently, the vast majority of these investigations have been conducted in animal models, providing mechanistic insights but with limited direct clinical applicability. In contrast, only a few studies have explored the presence and potential role of microplastics in the human heart.

In 2015, Erica Smith et al. conducted a study using polystyrene particles. Among the potential mechanisms increasing cardiovascular risk due to the presence of microplastic particles in the bloodstream, they identified the induction of platelet aggregation. All types of polystyrene particles used in the study exhibited a concentration-, particle size-, and surface modification–dependent effect on platelet aggregation. The strongest effect was observed for 50 nm anionic polystyrene latex nanoparticles modified with carboxyl groups and for 100 nm unmodified nanoparticles. This effect is believed to be mediated by protein kinase C–driven secretion of dense granule contents, extracellular calcium ions, and GPIIb/IIIa receptors, as the GPIIb/IIIa antagonist eptifibatide significantly inhibited aggregation in most cases. This effect was not associated with the loss of cell membrane integrity, as LDH levels did not differ significantly between the thrombin-exposed and polystyrene-exposed groups. In vivo, platelet aggregation was observed only after administering aminated polystyrene particles at low concentrations together with collagen. However, the same dose of microplastics with different surface properties or without collagen did not induce measurable aggregation on its own [7]. This finding suggests that the impact of nanoparticles on platelet function depends on the specific substance and its physicochemical properties – including particle size, surface charge, and concentration. Therefore, when assessing the health effects of microplastics, generalizations should be avoided, and each compound should be analyzed individually [8].

In 2020, Zekang Li et al. published the results of a study conducted on 32 male Wistar rats divided into four groups. One group served as the control, while the remaining three were orally administered polystyrene microplastics (PS-MPs) with a diameter of 0.5 μm at concentrations of 0.5 mg/L, 5 mg/L, and 50 mg/L for 90 days. The results indicated elevated levels of troponin I and creatine kinase-MB (CK-MB) in the serum of rats exposed to PS-MPs. In the group receiving the 50 mg/L dose, histological preparations revealed increased collagen proliferation and fibrosis, with some areas showing thinning and rupture of muscle fibers. In rats administered 5 mg/L and 50 mg/L of PS-MPs, mitochondrial changes were observed. The main cause of these alterations was attributed to the induction of cardiomyocyte apoptosis due to oxidative stress and the activation of fibrosis-related signaling pathways [9].

In 2021, several of the researchers responsible for the earlier study "Polystyrene microplastics cause cardiac fibrosis by activating Wnt/β-catenin signaling pathway and promoting cardiomyocyte apoptosis in rats" expanded their findings by exploring the involvement of another signaling pathway dependent on caspase-1, triggered by inflammatory stimulation resulting from oxidative stress, in the cardiotoxicity caused by microplastics [10]. Inhibition of the NLRP3 inflammasome pathway, which mediates pyroptosis induction, could potentially protect the cardiovascular system from the toxic effects of microplastics by preventing endothelial damage [11].

In 2022, Jingjing Zhao et al. conducted a study in which they orally administered polystyrene microplastics (0.5 μm and 5 μm in size) at doses of 0.1 μg/mL and 1 μg/mL to mice for 12 weeks. Their research focused on the effects of microplastics on the development of preclinical cardiovascular diseases. The researchers observed accelerated weight gain, primarily due to an increase in adipose tissue in the experimental group, as well as elevated fasting glucose and insulin levels in mice exposed to 0.5 μm polystyrene microplastics at a dose of 1 μg/mL. These changes appeared after only three weeks. Additionally, the

Table 1. Quality Assessment of Selected Studies on the Impact of Microplastics on the Cardiovascular System

Author (year)	Research model	Control group	Quantitative data	Transparent methodology	Type of microplastic	Limitations
Smith et al. (2015)	In vitro, in vivo (mice)	YES	YES	YES	50–100 nm Polystyrene latex nanoparticles (PLNP): unmodified (uPLNP), amine-modified (aPLNP) and carboxyl-modified (cPLNP)	No information on exposure time to MPs. Dependence on co-agonist (collagen) to induce platelet aggregation.
Li et al. (2020)	Rats	YES	YES	YES	PS	No long-term outcomes,. The study used only PS-MPs, without comparing the effects of other commonly encountered polymers.
Wei et al. (2021)	Rats	YES	YES	YES	PS	The study used only PS-MPs, without comparing the effects of other commonly encountered polymers.
Zhao et al. (2022)	Mice	YES	YES	YES	PS	The study does not tie structural and histological changes to clinical parameters like cardiovascular risk scores or long-term outcomes.The study used only PS-MPs, without comparing the effects of other commonly encountered polymers.
Zhang et al. (2023)	Mice	YES	YES	YES	PS	The study did not quantify how much of the inhaled MP burden reached systemic circulation (e.g., heart, blood vessels), as opposed to staying in the lungs.
Zhou et al. (2023)	Mice, organoids	YES	YES	YES	PS	Cardiac organoids and mice responded differently to the same MP exposures, suggesting limited translational coherence.
Wang et al. (2023)	Mice (ApoE-/-)	YES	YES	YES	PS	Mice were exposed to up to 250 mg/kg, which far exceeds estimated human exposure levels. This raises concerns about ecological validity.
Song et al. (2024)	Rats	YES	YES	YES	PS	Despite significant shifts in biomarkers, no notable morphological damage was observed in cardiac tissue, raising questions about the biological significance of the findings.
Zhang M. et al. (2024)	Mice	YES	YES	YES	Fluorescencyjny polistyren	Mice were exposed to a solution of 100 g/L , which far exceeds estimated human exposure levels.
Xiong et al. (2025)	Mice	YES	YES	YES	PS	The study does not describe how contamination or baseline MP exposure was prevented or monitored.
Yun Zhang et al. (2025)	People	YES	YES	Partly	PVC, PE, PS, PA66	High dropout rate (over 40% of patients did not complete the study).
Yang et al. (2024)	People	YES	YES	YES	PS, PE, PP, PMMA, PVC, PC, PET, PA6, and PA66	No information on patients' exposure time to MPs.
Yan et al. (2023)	People + rats	YES	YES	YES	PP, PS	No information on patients' exposure time to MPs.
Persiani et al. (2025)	In vitro	NO	YES	YES	PE, PS	In vitro experiment without validation in an animal model.
Wang et al. (2024)	People	YES	YES	Partly	PA66, PVC, PE	Small sample size (30 patients), analysis only of thrombus material without clinical observation, preventing assessment of the impact of MPs on disease progression.
Marfella et al. (2024)	People	YES	YES	Partly	PE, PVC	Separate results for different MP types were not presented. Lack of detailed data on patient exposure and potential confounding factors.

PS – Polystyrene, PE – Polyethylene, PP – Polypropylene, PMMA – Polymethyl methacrylate, PVC – Polyvinyl chloride, PC – Polycarbonate, PET – Polyethylene terephthalate, PA6 – Polyamide 6, PA66 – Polyamide 66.

experimental groups showed altered expression of genes associated with increased adipogenesis [12].

In 2023, Tianyi Zhang et al. conducted a study on mice to investigate the effects of inhaled polystyrene nanoplastics on cardiac tissue. The animals were exposed to nanoplastics for 1, 4, and 12 weeks, receiving doses ranging from 0 μg/day to 16, 40, and 100 μg/day, depending on the group. By analyzing nanoplastic accumulation, cardiac function, histological preparations, and inflammatory biomarkers (IL-6, MCP-1, and TNF-α) alongside cardiac injury biomarkers (cardiac troponin T (cTnT), ANP, N-terminal pro-B-type natriuretic peptide (NT-proBNP), and lactate dehydrogenase (LDH) in serum and cardiac tissue), the researchers concluded that nanoplastics cause structural and functional cardiac damage, with cardiotoxicity dependent on both dose and duration of exposure. As a potential mechanism for myocardial injury, mitochondrial damage was suggested. Four weeks of nanoplastic exposure led to a greater decrease in ejection fraction and shortening fraction compared to the 12-week exposure, indicating early cardiac dysfunction and structural disturbance. Cardiac fibrosis was observed only after 12 weeks. Similar to Zekang Li's findings, collagen accumulation in myocardial tissue was noted, while mitochondrial damage appeared as early as after four weeks [13].

In the same year, another study examined the effects of inhaling microplastics on mice. The mice were divided into three groups: one exposed to 0.9% saline solution, and two others to 25 μg and 50 μg of PS-MPs. Additionally, Yue Zhou and colleagues used human-induced pluripotent stem cell–derived cardiac organoids (COs) to investigate the effects of microplastics on heart pathology. The animal study revealed accumulation of PS-MPs in cardiac muscle, even after inhalation, as well as mitochondrial hypertrophy. Structural comparison between the experimental and control groups showed concentric hypertrophy of the heart and increased interventricular septum thickness in the group exposed to 50 μg of microplastics, along with a reduction in the ventricular-to-total surface area ratio. The COs were exposed to doses of 0.025 μg/mL, 0.25 μg/mL, and 2.5 μg/mL. An increase in CO diameter was observed following exposure to microplastics. To explore the cause of this phenomenon – possibly cell swelling due to necrosis – the expression of caspase-3 mRNA, a key transcription factor in cardiomyocyte apoptosis, was examined. However, the lack of significant changes in its expression ruled out apoptosis as a possible cause. The absence of apoptosis in cardiomyocytes after exposure to PS-MPs contradicts the findings of Zekang Li et al. (2020) and Ziyi Xiong (2025). Instead, it was demonstrated that collagen fiber accumulation contributed to the increased CO volume [14].

In 2023, Bo Wang and colleagues orally administered polystyrene nanoplastics (50 nm in size) to ApoE−/− mice for 19 weeks at doses ranging from 2.5 to 250 mg/kg alongside a high-fat diet. The results indicated disturbances in lipid metabolism, increased formation of atherosclerotic plaques, and elevated arterial stiffness. Significant differences compared to controls were observed in the 250 mg/kg group, which showed increased lipid accumulation, necrotic nuclei, and cholesterol crystallization. The researchers attributed the excessive formation of atherosclerotic plaques to macrophage accumulation associated with increased low-density lipoprotein uptake by foam cells due to overexpression of MARCO in macrophages. Progressive increases in both systolic and diastolic blood pressure were also observed [15].

In 2024, Zikai Song and colleagues examined the cardiovascular toxicity of polystyrene microplastics (PS-MPs) in rats. The study involved 48 Wistar rats divided into four groups: a control group and three experimental groups treated with doses of 0.5 mg/kg/day, 5 mg/kg/day, and 50 mg/kg/day. Contrary to previous findings, no pathological changes were observed in the heart or aorta. However, similar to earlier studies, increases in cardiac injury markers and inflammatory indicators – except IL-6 – were noted [16]. While previous studies, such as those by Tianyi Zhang et al., reported elevated IL-6 levels with prolonged exposure, this study found a decrease. Lipid metabolism was also altered following PS-MP exposure [13].

In the same year, Min Zhang and colleagues conducted a study on mice. The control group consisted of 10 mice, and the experimental group also included 10 mice, which received water containing 100 g/L of microplastics for 180 days. The microplastics were fluorescent polystyrene particles measuring 10 μm. The results showed accumulation of microplastics in the myocardium and cardiomyocyte apoptosis linked to m6A modification in non-coding RNA. Min Zhang and colleagues also found a relationship between microplastic exposure and inhibition of Na^+/K^+-ATPase activity, which is associated with the development of cardiovascular diseases [17]. Unlike the 2022 study by Jingjing Zhao et al., exposure to microplastics did not lead to weight gain in the mice [12], [17].

In 2025, Ziyi Xiong and colleagues administered oral polystyrene nanoplastics to 48 C57BL/6 male mice for 42 days. The mice were divided into four groups: one control group and three experimental groups receiving 30, 60, and 100 mg/L of nanoplastics. The researchers compared echocardiographic parameters, blood pressure, and electrocardiograms. The observed changes – such as a slower heart rate, ventricular dilation, thinning of the left ventricular posterior wall, reduced ejection fraction, and decreased left ventricular shortening fraction, accompanied by reduced blood pressure – indicated the de-

velopment of dilated cardiomyopathy. These changes differed from those observed by Yue Zhou et al., who reported concentric hypertrophy. Histopathological examinations by Ziyi Xiong also revealed a dose-dependent correlation between nanoplastic exposure and cardiomyocyte apoptosis – higher doses led to higher apoptosis rates [18]. Additionally, as observed by Zekang Li et al., Tianyi Zhang et al., and Yue Zhou et al., structural mitochondrial changes were also noted [9], [13], [14], [18].

To summarize, the cited studies do not allow for definitive conclusions regarding either the effects or mechanisms of microplastic-induced cardiac injury. The lack of standardization in microplastic doses and particle sizes, differences in observation periods, types of microplastics, and administration routes likely contribute to the discrepancies in results.

Compared with animal studies, research on the impact of microplastics on the human heart remains limited. Yun Zhang and colleagues conducted a study involving patients with acute ST-segment elevation myocardial infarction (STEMI) who scored 4–5 points on the TIMI scale during coronary angiography. They investigated the presence of microplastics and nanoplastics (MNPs) in the coronary arteries and whether their presence correlated with patient prognosis after myocardial infarction. A total of 214 patients were enrolled, of whom 110 completed the study. The mean follow-up period was 31.5 months. The study found a significant association between the presence of polyvinyl chloride (PVC) in the blood and the occurrence of major adverse cardiovascular events. Other microplastics examined – polyethylene, polystyrene, and polyamide 66 (PA66) – did not show this association. The PVC levels were correlated with increased lymphocyte and macrophage concentrations in coronary thrombi, and higher PVC levels corresponded to elevated pro-inflammatory cytokine concentrations. PVC detected in coronary thrombi was therefore linked to increased levels of pro-inflammatory cytokines [19].

Impact of microplastics on the blood vessels

Considering the impact of microplastics on vascular diseases, it is particularly important to distinguish between experimental studies in animals and in vitro models versus clinical observations in humans. Most mechanistic data are derived from animal or cell culture experiments, while human studies remain limited.

In a study by Yang et al., a link was demonstrated between elevated microplastic levels in peripheral blood and an increased risk of coronary artery atherosclerosis. For this purpose, coronary angiography and gas chromatography/mass spectrometry (Py-GC/MS) were performed on 101 patients admitted to a hospital in Beijing due to chest pain. In patients diagnosed with acute coronary syndrome, the average microplastic concentration was 161.65 μg per gram, with polyethylene being the dominant polymer (74.40%), while in the control group it was 100.13 μg per gram, with polyethylene accounting for 56.23%. No significant differences were observed between the subgroups in terms of age, gender, hypertension, diabetes, or ejection fraction values. However, it was noted that patients at low risk on the SYNTAX scale showed a tendency toward increased microplastic concentrations compared to the control group, although this was not statistically significant. In contrast, patients with medium and high risk had significantly higher levels ($p < 0.0001$). This suggests a relationship between the presence of microplastics and the complexity of vascular pathology, with inflammatory mechanisms likely contributing to its development. A positive correlation was found between microplastic concentrations and the levels of cytokines IL-6, IL-12, and the numbers of B lymphocytes and NK cells [20].

In a study conducted by Yan et al., a statistically significant correlation was found between the presence of calcifications in the thoracic aorta and higher microplastic levels in the patients' feces ($p < 0.0001$). The most common polymers were polystyrene, polyethylene, and polyester. This correlation was later confirmed in a rat study, where exposure to polystyrene caused mild vascular and cardiac calcification in healthy animals, which was exacerbated when nicotine and vitamin D_3 were also administered to the rodents [21].

Microplastic exposure leads to the activation of vascular smooth muscle cells (VSMCs), promoting the development of atherosclerosis and calcification. In vitro studies by Persiani et al. using polyethylene and polystyrene in their primary and photo-degraded forms, which simulated real environmental conditions, showed that microplastics induced disturbances in VSMC proliferation and migration, damaged cell membranes, and triggered apoptosis. They also caused a phenotypic change in cells, promoting osteogenic differentiation and calcification, as indicated by increased expression of runt-related transcription factor 2 (RUNX-2) and galectin-3, which mediate the link between oxidative stress and vascular calcification [22].

Microplastics can also act as vectors transporting other hazardous substances, such as heavy metals and bacteria. It has been observed that in patients with aortic diseases, such as aneurysm or dissection, simultaneous exposure to microplastics and lead was greater. This correlation was confirmed in a mouse study, where such exposure led to the destruction of elastic fibers, loss of vascular myocytes, and the

release of inflammatory factors, resulting in degeneration of the aortic media. The mechanism behind the impaired arterial structure was the activation of the cAMP/PKA-ROS signaling pathway and mitochondrial damage leading to PANoptosis [23]. This information is worth comparing with data from the study by Chen et al., who investigated the destructive impact of microplastics on endothelial structure using polystyrene concentrations that realistically occur in the blood. In vitro, they observed impairment of the vascular barrier through reduced levels of the zonula occludens-1 protein, decreased expression of antioxidants and heat shock proteins with protective potential, and apoptosis of cells. However, it was not shown that exposure to microplastics caused an increase in the expression of the LOX-1 receptor for oxidized low-density lipoprotein (oxLDL), suggesting that polystyrene does not significantly increase the risk of atherosclerotic plaque formation. Contact with it at concentrations close to environmental levels may therefore be associated with a low risk of vascular disease [24].

It is also suspected that microplastic particles may participate in the formation of thromboembolic events, which was the subject of research by Wang et al. They searched for microplastic particles using spectroscopy in material obtained from 30 patients undergoing thrombectomy due to stroke, myocardial infarction, or deep vein thrombosis. Microplastics were found in 80% of the samples, with the highest average concentration of 141.80 μg/g observed in myocardial infarction cases. It was found that higher polymer concentrations were associated with a more severe course of disease ($p < 0.05$), and D-dimer levels were significantly higher in the group where microplastic particles were detected ($p < 0.001$) [25]. The mechanism by which microplastics might initiate clot formation is still unclear, but a positive correlation was observed between the amount of microplastics in the thrombus and platelet levels ($p < 0.01$). In vitro and animal studies have shown that exposure to polymers results in reduced fibrin binding to platelets, which, under real blood flow conditions, is associated with a high risk of thrombus detachment [26].

The largest study to date regarding the impact of microplastics on cardiovascular events was conducted by R. Marfella et al., involving patients undergoing carotid endarterectomy due to asymptomatic carotid artery disease. The study investigated the association between the presence of polyethylene (PE) and polyvinyl chloride (PVC) in atherosclerotic plaques and the incidence of myocardial infarction, stroke, or death from any cause. A total of 304 patients were included, 257 of whom completed the study. The average follow-up period was 33.7 months. Polyethylene was detected in 150 patients at an average concentration of 21.7 ± 24.5 μg/mg of plaque, while polyvinyl chloride was found in 31 patients at a concentration of 5.2 ± 2.4 μg/mg of plaque. Based on these results, the researchers concluded that the presence of microplastics in atherosclerotic plaques was associated with nearly a 4.5-fold higher risk of myocardial infarction, stroke, or death from any cause. Unfortunately, the publication did not provide detailed endpoint data for the specific types of microplastics [27].

Limitations

This review has several limitations. First, the available evidence is dominated by animal studies, with only a few small-scale human investigations. Second, there is substantial heterogeneity across studies in terms of plastic type, particle size, exposure dose, route of administration, and duration, which complicates comparisons and prevents reliable synthesis. Third, the quality of many studies was limited by unclear randomization, blinding, and incomplete reporting, resulting in a considerable risk of bias. Finally, this review is narrative in nature and does not include a quantitative meta-analysis, which restricts the ability to estimate pooled effect sizes.

Discussion

The results of this review indicate that micro- and nanoplastics (MP-NPs) may adversely affect the cardiovascular system, particularly by inducing structural and functional changes in cardiac muscle and blood vessels. Animal studies consistently reported myocardial fibrosis, mitochondrial alterations, and inflammatory activation, as well as disturbances in lipid and glucose metabolism. These mechanisms provide a biological rationale for the potential development of hypertension, atherosclerosis, and heart disease.

Human data remain scarce and largely observational. To date, only two longitudinal studies – Marfella et al. and Yun Zhang et al. – have provided outcome-based evidence [27], [19]. Both support an association between MP-NPs and adverse cardiovascular events, but causality cannot be established. Other human investigations identified correlations between microplastic burden and vascular disease severity, though they lacked long-term follow-up [20], [25]. Together, these findings suggest that MP-NPs may contribute to cardiovascular risk, but the evidence remains preliminary.

An additional challenge is the overly generalized use of the term "microplastics." Different polymers (e.g., PE, PS, PVC, PA66) vary in their physicochemical properties and, therefore, in toxicity. Few com-

parative studies directly address these differences under standardized conditions. Moreover, while some reports examined specific signaling pathways (e.g., NLRP3, Wnt/β-catenin), others used broader markers of injury, making it difficult to synthesize results.

In light of these limitations, there is a clear need for well-designed epidemiological and clinical studies to determine population exposure levels and clarify health consequences. Particular focus should be placed on individuals with pre-existing cardiovascular disease, who may be especially vulnerable to environmentally mediated vascular injury. At the same time, the accumulating mechanistic evidence supports the importance of preventive public health measures, such as reducing plastic production and improving recycling strategies, to mitigate potential risks.

Conclusions

The direct impact of microplastics on the development of various cardiovascular diseases has not been definitively proven. However, there is evidence of microplastic accumulation in many tissues. While most studies have been conducted in animals, they suggest that at sufficiently high concentrations and with prolonged exposure, microplastics may negatively affect health and organ function. Raising public awareness about the health risks associated with micro- and nanoplastic exposure is equally important. While plastic is designed to simplify modern life, its long-term consequences may directly impact human health – including that of future generations. Given the continuously increasing production of plastic and its prevalence in the environment, extensive research focused on the effects of microplastics on the human body is necessary.

Implementing appropriate technologies could reduce the amount of microplastics entering organisms and, as a result, act as a preventive measure against many diseases related to microplastic exposure. Reducing microplastic pollution should be a coordinated effort at three stages. First, and most importantly, it should involve limiting production and shifting to more eco-friendly materials. Second, it should focus on cleaning up the already polluted environment. Third, it should include recycling the plastic that has already been produced [28], [29].

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