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Microplastic and Nanoplastic Contamination as a Novel Cardiovascular Risk Factor: A Narrative Review of Pathophysiological Mechanisms and Public Health Consequences

Katarzyna Stróżna https://orcid.org/0009-0007-7614-1108
Medical Center of Postgraduate Education, [1]Professor Witold Orłowski Independent Public Clinical Hospital, Warszawa, Poland
Czerniakowska 231, 00-416 Warszawa
e-mail: katarzyna.strozna1@gmail.com

Łukasz Kowalewski ORCID https://orcid.org/0009-0008-4005-2610
University Clinical Centre, Gdańsk, Poland
ul. Dębinki 7, 80-952 Gdańsk
e-mail: lek.lukasz.kowalewski@gmail.com

Amir Naziri ORCID https://orcid.org/0009-0009-4465-1685
University Clinical Centre, Gdańsk, Poland
ul. Dębinki 7, 80-952 Gdańsk
E-mail: amirnaziri@o2.pl

Martyna Zielnik, ORCID https://orcid.org/0009-0007-9787-0625
The John Paul's II Western Hospital in Grodzisk Mazowiecki, Poland
ul. Daleka 11, 05-825 Grodzisk Mazowiecki
E-mail: martynazie@icloud.com

Maria Aleksandra Styczyńska ORCID https://orcid.org/0009-0005-3583-1271
University Clinical Centre, Gdańsk, Poland
ul. Dębinki 7, 80-952 Gdańsk
E-mail: mstyczynska00@gmail.com

Julianna Rozalia Chyl ORCID https://orcid.org/0009-0003-1115-1226
6th year medical student
Medical University of Gdańsk, Poland
ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk
e-mail: julianna.chyl@gmail.com

Corresponding Author

Maria Aleksandra Styczyńska, e-mail: mstyczynska00@gmail.com

Abstract

Background. Microplastics (MPs) and nanoplastics (NPs), collectively referred to as MNPs, are widespread environmental pollutants detected in numerous human tissues, including blood, placenta, liver, lungs, and brain. Their potential impact on cardiovascular health has recently become an important research focus, particularly given that cardiovascular disease (CVD) remains the leading cause of death worldwide.

Aim. This narrative review summarises current evidence regarding the presence of MNPs in the human cardiovascular system, their proposed mechanisms of toxicity, and their possible association with adverse cardiovascular outcomes.

Material and methods. A non-systematic search of PubMed, Scopus, and Web of Science was conducted for publications from 2018–2025 using terms including "microplastics," "nanoplastics," "cardiovascular disease," "atherosclerosis," "oxidative stress," and "endocrine disruption." Relevant original studies, clinical trials, and reviews were selected based on relevance and methodological quality.

Results. MNPs have been identified in atherosclerotic plaques, where their presence was associated with a significantly increased risk of major adverse cardiovascular events (MACE),

including myocardial infarction, stroke, and all-cause mortality (HR 4.53; 95% CI 2.00–10.27; P < 0.001). Proposed mechanisms include oxidative stress, chronic inflammation, endocrine disruption, and direct cytotoxicity. Experimental studies support these pathways, although causal evidence from human studies remains limited.

Conclusions. MNPs may represent an emerging cardiovascular risk factor. Further longitudinal studies, standardised detection methods, and public health policies reducing plastic pollution are urgently required.
Key words: microplastics; nanoplastics; cardiovascular disease; atherosclerosis; oxidative stress; endocrine disruption; narrative review.

1. Introduction

Plastic pollution constitutes one of the defining environmental challenges of the twenty-first century. Since mass production of synthetic polymers began in the mid-twentieth century, global plastic output has grown exponentially — from approximately 2 million metric tonnes in 1950 to over 450 million metric tonnes annually by 2024 [1]. Only a small fraction of this material is effectively recycled or incinerated; the vast majority is deposited in landfills or dispersed into terrestrial and aquatic ecosystems, where it undergoes physical, chemical, and biological fragmentation into progressively smaller particles [2]. Microplastics are defined as particles ranging in size from 0.1 to 5,000 μm, while nanoparticles range from 0.001 to 0.1 μm (1–100 nm) [3]. Primary MNPs are deliberately manufactured at micro- or nanoscale for use in cosmetics, abrasives, and industrial processes; secondary MNPs arise from the environmental degradation of larger plastic objects. Both fractions are now detectable in the Arctic ice, deep ocean sediments, drinking water, and agricultural soils, illustrating the global scale of contamination [4]. Crucially, MNPs have been identified in numerous human biological samples, including blood, urine, breast milk, lung tissue, the placenta, and hepatic parenchyma [5,6]. A landmark 2025 study published in Nature Medicine confirmed the bioaccumulation of MNPs in human brain, kidney, and liver tissue using pyrolysis gas chromatography–mass spectrometry, finding higher proportions of polyethylene in brain tissue relative to other organs. The data indicate that microplastic concentrations in the brains of deceased individuals were 7 to 30 times higher than those in the kidneys or liver. Even higher concentrations of microplastics were found in people with dementia [7]. These findings indicate systemic exposure and raise

the possibility of organ-specific toxicity. Cardiovascular disease (CVD) remains the foremost cause of morbidity and mortality worldwide, responsible for approximately 30% of all deaths [8, 9]. The classical risk factors established by the Framingham Heart Study — hypertension, dyslipidaemia, cigarette smoking, diabetes mellitus, and physical inactivity — do not fully account for the total attributable burden of CVD [10, 11]. Emerging evidence suggests that environmental pollutants, including fine particulate matter (PM2.5) and, more recently, MNPs, may act as additional, potentially modifiable risk factors [12].

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This narrative review synthesises the current state of knowledge on MNPs in the context of cardiovascular health, covering their detection in vascular tissue, the main proposed toxicological mechanisms, key clinical findings, limitations of existing research, and future directions.

2. Sources of Human Exposure and Pathways of Entry

Human exposure to MNPs occurs predominantly through three routes: ingestion, inhalation, and dermal contact [13]. Dietary intake represents the principal pathway, with MNPs detected

in seafood, table salt, bottled water, honey, and a broad array of packaged foods. Mason et al. (2018) identified synthetic polymer particles in commercially available bottled water from 11 widely distributed brands [14]. Similarly, Cox et al. (2019) estimated that an average adult in the United States may ingest between 39,000 and 52,000 MNP particles per year through diet alone, rising to over 74,000 when inhalation is included [15].

Inhalation constitutes a clinically significant, though often underappreciated, route of exposure, particularly in occupational settings and indoor environments where airborne fibre counts can be substantially elevated. Amato-Lourenco et al. (2020) documented the potential respiratory effects of inhaled MNPs and classified them as an emerging class of air pollutants [16]. The dermal route is considered less important in quantitative terms, though injection and surgical procedures may represent a direct intravascular pathway, bypassing epithelial barriers [17].

Once inside the body, the fate of MNPs depends critically on particle size. Particles exceeding 150 μm are generally not absorbed across gastrointestinal mucosa, whereas those smaller than 1.5 μm may achieve systemic bioavailability [3, 18]. Approximately 0.3% of particles measuring between 1 and 10 μm are estimated to be absorbed in the intestinal tract; smaller nanoscale particles may be internalised by immune cells, including macrophages and dendritic cells, which can then distribute them throughout the body [17, 19].

Food packaging and preparation processes further amplify dietary exposure. Heating food in plastic containers, repeated reuse of single-use packaging, and mechanical abrasion of polymer surfaces during food processing have all been shown to increase the release of micro- and nanoscale fragments into food matrices [13, 18]. Tea bags manufactured from nylon or polyethylene terephthalate release billions of sub-micron particles per cup when steeped in near-boiling water, illustrating that even routine domestic practices can constitute a non-trivial exposure source [18]. Household dust represents an additional, frequently overlooked reservoir, with synthetic textiles, furnishings, and degrading plastic products continuously shedding fibres into indoor air that are subsequently inhaled or ingested, particularly by young children through hand-to-mouth behaviour [16, 19].

3. Microplastics and Nanoplastics in the Cardiovascular System

3.1 Detection in Atherosclerotic Plaque

The most consequential human study to date linking MNPs directly to cardiovascular pathology was published by Marfella et al. in The New England Journal of Medicine in March 2024 [20]. This prospective, multicentre, observational study enrolled 304 patients undergoing carotid endarterectomy for asymptomatic carotid artery disease at centres across Italy. Excised atherosclerotic plaque specimens were analysed for MNP content using pyrolysis–gas chromatography–mass spectrometry, stable isotope analysis, and electron microscopy.

Polyethylene was detected in carotid artery plaque specimens from 150 patients (58.4%), with a mean concentration of 21.7 ± 24.5 μg per milligram of plaque. Polyvinyl chloride was additionally identified in 31 patients (12.1%), with a mean level of 5.2 ± 2.4 μg per milligram. Electron microscopy revealed jagged-edged foreign particles dispersed among plaque macrophages and within the external debris of atheromatous lesions. Concentrations of inflammatory biomarkers, including interleukin-18 and matrix metalloproteinase-9, were significantly elevated in patients with MNP-positive plaque compared to those without detectable particles [20].

3.2 Association with Major Adverse Cardiovascular Events

Following a mean follow-up period of 34.0 months, patients with MNPs detected within the atheroma demonstrated a markedly higher incidence of the composite primary endpoint — encompassing myocardial infarction, stroke, and death from any cause — compared to those with MNP-negative plaque (hazard ratio 4.53; 95% confidence interval 2.00–10.27; $P < 0.001$) [20]. This association persisted after adjustment for established cardiovascular risk factors, including diabetes mellitus, arterial hypertension, and dyslipidaemia.

As noted by O'Dowling (2025) in an editorial published in JACC Advances, this finding invites a comparison with the historical identification of cardiovascular risk factors through the Framingham Heart Study and raises the question of whether MNPs should now be formally considered a novel cardiovascular risk factor [1]. The authors of the NEJM study themselves cautioned that causality has not been established and that unmeasured confounding variables could have contributed to the observed associations [20].

The publication of these findings generated substantial interest across the cardiology and toxicology communities, prompting a wave of commentaries, correspondence, and secondary analyses. Several authors have called for replication of the Marfella et al. cohort in geographically and ethnically diverse populations, noting that the original study was conducted exclusively at Italian centres and that dietary, occupational, and environmental exposure patterns vary considerably across regions [1, 12]. Others have emphasised that carotid plaque, while a reasonable surrogate for systemic atherosclerotic burden, may not fully generalise to coronary or cerebrovascular disease, and have called for parallel investigations using coronary artery specimens obtained at autopsy or during cardiac surgery [12].

4. Proposed Pathophysiological Mechanisms

4.1 Oxidative Stress and Inflammation

The most extensively characterised mechanism through which MNPs exert toxicity is the induction of oxidative stress. In vitro studies utilising human endothelial and vascular smooth muscle cell lines have demonstrated that exposure to polystyrene microplastics significantly increases the intracellular generation of reactive oxygen species (ROS), depletes antioxidant enzymes, and activates the nuclear factor-κB (NF-κB) signalling pathway, culminating in upregulation of pro-inflammatory cytokines including tumour necrosis factor-α, interleukin-6, and interleukin-1β [2,4].

Chronic low-grade vascular inflammation is a central driver of atherosclerotic plaque progression and vulnerability to rupture. By sustaining this inflammatory milieu, MNPs could plausibly accelerate the development and destabilisation of coronary and carotid plaques, thereby increasing the risk of acute ischaemic events. Animal models consistently support this hypothesis: polystyrene MNP-exposed rodents demonstrate elevated circulating markers of systemic inflammation and accelerated endothelial dysfunction [21, 22].

A further mechanistic layer involves activation of the NLRP3 inflammasome, a multiprotein complex that senses cellular danger signals, including particulate matter, and triggers caspase-1-mediated cleavage of pro-interleukin-1β into its active, highly pro-atherogenic form. Particulate activation of NLRP3 has already been implicated in the pathogenesis of atherosclerosis in the context of cholesterol crystals and silica particles, and

several in vitro studies suggest that polystyrene and polyethylene fragments can trigger an analogous response in macrophages [2, 4]. Mitochondrial dysfunction appears to sit upstream of much of this inflammatory signalling: internalised MNPs have been shown to impair mitochondrial membrane potential and electron transport chain efficiency in endothelial cells, generating a feed-forward cycle in which mitochondrial ROS further amplifies NF-κB activation and cytokine release [2].

4.2 Endocrine Disruption

Many plastic polymers contain or adsorb endocrine-disrupting chemicals (EDCs), including bisphenol A (BPA), phthalates, and polychlorinated biphenyls (PCBs), which may leach into biological fluids following MNP uptake. These compounds can interfere with oestrogen and androgen receptor signalling, disrupt thyroid hormone metabolism, and alter the hypothalamic-pituitary-adrenal axis. Experimental exposure to polystyrene nanoplastics has been shown to downregulate the expression of genes involved in spermatogenesis and to alter steroidogenic pathways in rodent models [21, 22, 23]. Endocrine disruption has established links to dyslipidaemia, insulin resistance, and hypertension — all established cardiovascular risk factors [4].

4.3 Direct Cellular Toxicity and DNA Damage

At sufficiently high concentrations, MNPs exert direct cytotoxicity characterised by mitochondrial dysfunction, lysosomal membrane destabilisation, and induction of apoptosis. Genotoxic effects, including DNA strand breaks and micronucleus formation, have been documented in human cell lines following exposure to micro- and nanoscale polystyrene particles [2]. Furthermore, MNPs may serve as vectors for environmental carcinogens and heavy metals, amplifying their inherent toxicity through synergistic interactions [4].

4.4 Perturbation of the Gut Microbiota

An emerging body of evidence suggests that chronic exposure to MNPs may dysregulate the intestinal microbiome, reducing the abundance of beneficial taxa such as Akkermansia muciniphila and Lactobacillus species while promoting the growth of pro-inflammatory bacteria. Gut dysbiosis has been mechanistically linked to systemic inflammation, altered bile

acid metabolism, and impaired intestinal barrier integrity — all of which may potentiate CVD risk through both direct and immune-mediated pathways [24].

5. Current State of Clinical Evidence and Limitations

Beyond the landmark study by Marfella et al. [20], clinical evidence directly linking MNP exposure to human cardiovascular outcomes remains sparse. The majority of mechanistic data derives from in vitro cell culture experiments and animal models, which, while biologically informative, do not necessarily replicate the complexity of human exposure or the multifactorial aetiology of atherosclerotic CVD [4].

Several important methodological challenges limit progress in this field. First, there is currently no internationally standardised protocol for the detection and quantification of MNPs in human biological matrices. Variability in sample preparation, analytical techniques, and reporting metrics makes cross-study comparisons unreliable [3,17]. Second, as highlighted by critics of the Marfella et al. study, the risk of environmental contamination of surgical specimens with MNPs — given their ubiquitous presence in operating room environments — cannot be entirely excluded without rigorous anticontamination protocols [20].

Third, epidemiological studies face the challenge of establishing dose-response relationships in populations where exposure assessment is inherently imprecise. No consensus currently exists on what constitutes a hazardous internal body burden of MNPs [6]. Fourth, long-term prospective cohort studies with validated biomarkers of MNP exposure and adjudicated cardiovascular outcomes are lacking; without these, causal inference remains speculative.

A comprehensive systematic review by Ririe et al. (2025), incorporating human-based evidence from PubMed, Scopus, Web of Science, and Google Scholar for publications from 2010 to 2025, concluded that while MNPs have been consistently detected in human cardiovascular and other tissues, direct causal evidence linking MNP exposure to adverse clinical outcomes in humans remains insufficient to make definitive public health recommendations [6].

Publication and selection bias represent additional concerns in this rapidly evolving literature. Studies reporting positive associations between MNP exposure and adverse outcomes may be preferentially submitted and accepted for publication relative to null findings, potentially inflating the perceived strength of the evidence base [3, 6]. Population heterogeneity

further complicates interpretation: existing studies have drawn on relatively small, geographically restricted cohorts, and exposure levels are likely to vary substantially with diet, occupation, place of residence, and local waste-management infrastructure. Confounding by co-exposure to other environmental toxicants, including heavy metals and persistent organic pollutants that frequently adsorb to plastic surfaces, cannot be fully excluded using currently available analytical techniques [4, 17]. Finally, reverse causality remains a theoretical possibility in cross-sectional and case-control designs: it is conceivable that diseased or inflamed vascular tissue accumulates particulate material more readily than healthy tissue, rather than MNP deposition being the primary causal event.

6. Future Research Directions and Public Health Implications

Given the pace of accumulating evidence and the global scale of MNP contamination, the research and regulatory communities face considerable urgency. Priority areas for future investigation include: the development and validation of standardised, reproducible methods for MNP detection in human blood, tissue, and urine; the establishment of large-scale longitudinal cohort studies with prospective MNP exposure assessment and hard cardiovascular endpoints; and the characterisation of dose-response relationships across relevant exposure ranges.

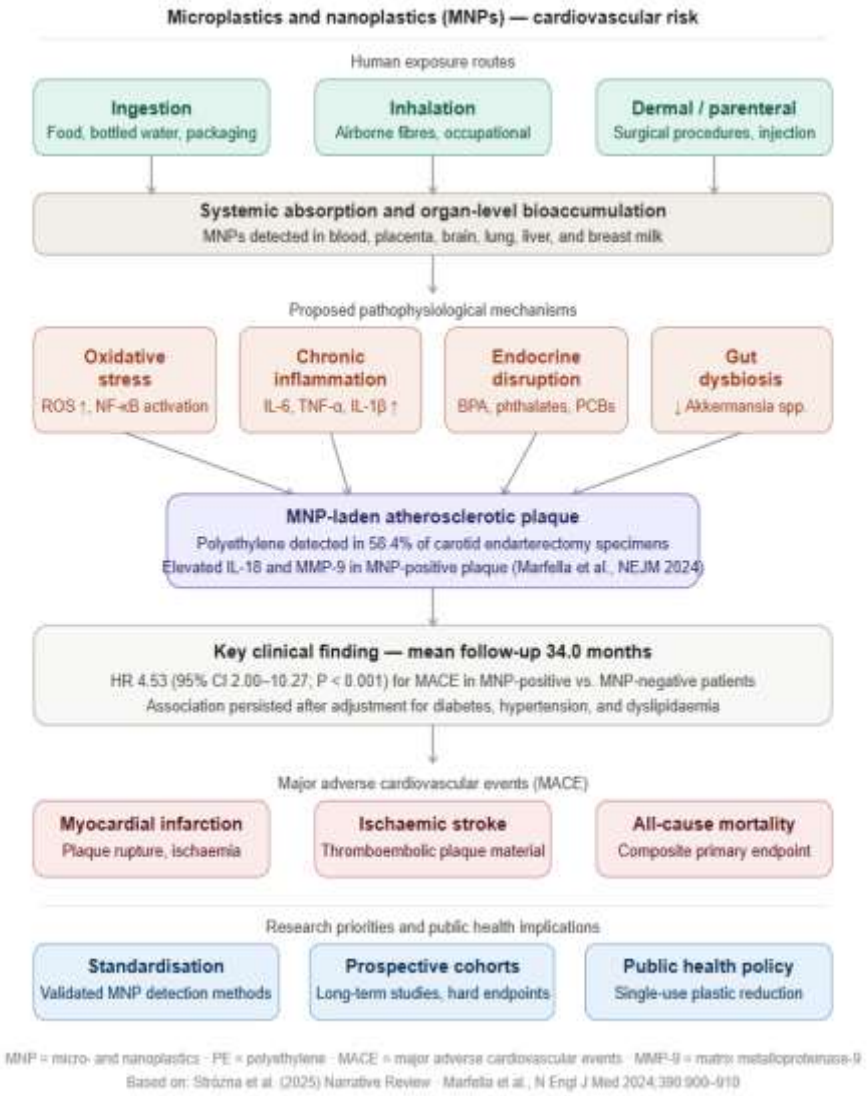
A review published in The Lancet Planetary Health (2025) proposed a comprehensive framework for assessing the disease burden attributable to MNP exposure across multiple organ systems, emphasising the need for interdisciplinary collaboration between toxicologists, epidemiologists, cardiologists, and environmental scientists [21]. The review highlighted that evidence for an association between MNP exposure and metabolic, respiratory, cardiovascular, and neuroendocrine disorders is accumulating rapidly, even if causality has not been definitively established for most conditions.

From a public health standpoint, precautionary action does not necessarily require proof of causality. The parallels with the history of tobacco-related cardiovascular research — in which observational associations preceded mechanistic understanding by decades — are instructive. Regulatory measures to reduce single-use plastics, improve waste management infrastructure, and mandate the disclosure of plastic additives could yield co-benefits for both environmental and cardiovascular health [1].

Clinicians should be aware of MNPs as a potential emerging risk factor and consider discussing plastic exposure reduction with high-risk patients, even in the absence of definitive guidance. The development of validated exposure biomarkers will be essential for integrating MNP assessment into clinical risk stratification frameworks.

Regulatory momentum in this area is beginning to build. Several jurisdictions, including the European Union, have introduced restrictions on intentionally added microplastics in cosmetic and household products, while international negotiations toward a binding global plastics treaty remain ongoing under the auspices of the United Nations Environment Programme. Whether such measures meaningfully reduce human internal exposure over clinically relevant timeframes remains to be demonstrated, underscoring the need for exposure-biomonitoring programmes that can track population-level trends alongside policy implementation [1, 21]. From a research infrastructure perspective, integrating MNP quantification into existing large-scale cardiovascular cohort studies and biobanks would allow investigators to leverage already-collected outcome data, substantially accelerating the pace at which causal questions can be addressed relative to establishing entirely new prospective cohorts.

Figure 1. Schematic overview of human MNP exposure routes, pathophysiological mechanisms, and associated cardiovascular outcomes. MNP, micro- and nanoplastics; MACE, major adverse cardiovascular events.



7.Conclusions

Microplastics and nanoplastics represent a novel and plausible class of cardiovascular risk factors. Their presence in atherosclerotic plaque, their association with elevated inflammatory biomarkers, and their link to a markedly increased incidence of major adverse cardiovascular events in a landmark prospective human study collectively constitute a compelling, if not yet conclusive, body of evidence. The proposed mechanisms — oxidative stress, chronic inflammation, endocrine disruption, direct cytotoxicity, and gut microbiota perturbation — are biologically coherent and supported by in vitro and animal data.

Significant scientific gaps remain, particularly regarding the establishment of causal pathways in humans, the identification of safe exposure thresholds, and the development of standardised analytical methods. Addressing these gaps will require coordinated international research efforts, regulatory engagement, and investment in longitudinal epidemiological infrastructure. In the interim, the precautionary principle warrants both continued scientific vigilance and early consideration of plastic-reduction policies as a public health priority.

Author's Contribution:

Conceptualization: MS, AN, MZ
Methodology: KS, MS, JC
Formal analysis: ŁK, JC, MZ
Investigation: AN, MZ, KS, ŁK, JC, MS
Supervision: MS, JC, KS
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