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IMPACT OF MICROPLASTICS AND NANOPLASTICS EXPOSURE ON CARDIOVASCULAR AND CEREBROVASCULAR OUTCOMES: A SYSTEMATIC REVIEW

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ABSTRACT

Background: Micro- and nanoplastics (MNPs) accumulate in human tissues, inducing oxidative stress, chronic inflammation, and endothelial dysfunction, which drive vascular remodeling and atherothrombotic events.
Aim: This systematic review evaluated the association between micro- and nanoplastic (MNP) exposure and human cardiovascular and cerebrovascular outcomes, summarizing deposition patterns, vascular manifestations, and inflammatory markers.
Methods: Following PRISMA guidelines, a systematic search of PubMed and Scopus identified 26 observational human studies on adult cohorts. Extracted variables included biological matrices, particle analytics, MNP sizes/concentrations, clinical endpoints, and pro-inflammatory biomarkers.
Results: MNPs were widely detected in human blood, vascular walls, atherosclerotic plaques, thrombi, and cerebrospinal fluid, with polyethylene, polystyrene, and PVC being most prevalent. Plaques contained up to 80-fold higher MNP concentrations than healthy tissue or blood. Higher MNP burdens strongly correlated with advanced atherosclerosis, acute coronary syndromes, stroke, and major adverse cardiovascular events (MACE). Particle accumulation was consistently linked to oxidative stress and elevated pro-inflammatory markers (IL-6, TNF-α, IL-1, IL-18) driving vascular inflammation and plaque instability.
Conclusion: MNPs accumulate throughout the cardiovascular and cerebrovascular systems, with higher exposure linked to advanced vascular disease and adverse outcomes. Key mechanisms include chronic inflammation, endothelial injury, and oxidative stress. However, observational designs, analytical heterogeneity, and potential contamination risks prevent proving causality, highlighting the need for standardized, large-scale prospective studies.

KEYWORDS
Microplastics, Nanoplastics, Cardiovascular Diseases, Atherosclerosis

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1. Introduction

Plastic pollution has become one of the most significant environmental and public health challenges of the twenty-first century. The continuous increase in global plastic production, combined with inadequate waste management and the remarkable durability of synthetic polymers, has resulted in the widespread accumulation of plastic debris throughout the environment. Progressive degradation of larger plastic materials through ultraviolet radiation, mechanical abrasion, and chemical weathering generates microplastics (MPs), defined as particles smaller than 5 mm, and nanoplastics (NPs), generally measuring less than 1 μm. Today, these particles are ubiquitous contaminants detected in marine and freshwater ecosystems, agricultural soils, atmospheric air, drinking water, and numerous food products, making chronic human exposure virtually unavoidable [1,2].

The primary routes of human exposure include ingestion and inhalation, although dermal absorption may also contribute under certain conditions. Recent analytical advances have confirmed the presence of MPs and NPs in multiple human tissues and biological fluids, including blood, lungs, placenta, liver, breast milk, and other organs, indicating that these particles can cross biological barriers and enter the systemic circulation [3]. Compared with larger plastic fragments, nanoplastics possess unique physicochemical characteristics that facilitate cellular uptake and tissue penetration. Their extremely small size, large surface area to volume ratio, and high chemical reactivity enable interactions with cellular membranes and intracellular organelles. Furthermore, plastic particles readily adsorb environmental contaminants such as heavy metals, persistent organic pollutants, and endocrine-disrupting chemicals, potentially acting as carriers that amplify their toxicological effects [2–4].

Although the environmental presence of MPs and NPs has been recognized for many years, their impact on human health has only recently become a major focus of biomedical research. Experimental studies have demonstrated that plastic particles induce oxidative stress, mitochondrial dysfunction, chronic inflammation, apoptosis, and immune dysregulation while disrupting cellular metabolism and intercellular signaling pathways. These biological responses closely resemble mechanisms involved in the development of numerous chronic diseases, particularly those affecting the cardiovascular system. Persistent oxidative stress and inflammation contribute to endothelial dysfunction, impair vascular homeostasis, and promote structural changes within the arterial wall, thereby creating favorable conditions for atherosclerosis and thrombotic complications [2,5].

Increasing evidence from experimental models further suggests that MPs and NPs adversely affect multiple components of the cardiovascular system. Exposure has been associated with impaired endothelial function, increased production of reactive oxygen species, activation of inflammatory cytokines, platelet activation, altered lipid metabolism, and accelerated vascular remodeling. Nanoplastics appear to exhibit greater biological activity than larger microplastic particles because their dimensions facilitate intracellular internalization and translocation across endothelial barriers. Similar pathophysiological mechanisms have also been proposed within the cerebral circulation, where endothelial injury, disruption of the blood brain barrier, microvascular dysfunction, and neuroinflammation may contribute to cerebrovascular pathology, including ischemic stroke [5–7].

Despite rapidly growing scientific interest, the available evidence regarding the cardiovascular and cerebrovascular consequences of MP and NP exposure remains fragmented. Most current knowledge originates from experimental studies, whereas human clinical investigations are relatively few and differ considerably with respect to study design, exposure assessment, analytical methods used for particle identification, and evaluated clinical outcomes. Consequently, the extent to which environmental exposure to MPs and NPs contributes to cardiovascular and cerebrovascular disease development remains uncertain, and the underlying biological mechanisms have yet to be fully elucidated.

Therefore, a comprehensive synthesis of currently available clinical evidence is warranted. The aim of this systematic review was to evaluate the association between exposure to microplastics and nanoplastics and cardiovascular as well as cerebrovascular outcomes in humans. Additionally, we sought to summarize the current evidence regarding tissue deposition of plastic particles, characterize reported cardiovascular and cerebrovascular manifestations, and discuss the biological mechanisms that may explain these associations while identifying important gaps for future research.

2. Materials and Methods

To investigate the Impact of Microplastics and Nanoplastics Exposure on Cardiovascular and Cerebrovascular Outcomes, a systematic literature search was conducted on July, 31st, 2026, in the PubMed electronic database.

For the PubMed database, the following search query was applied:

1. ("Microplastics"[Mesh] OR microplastic*[tiab] OR nanoplastic*[tiab] OR micro-plastic*[tiab] OR nano-plastic*[tiab] OR micro-nanoplastic*[tiab]) AND ("Cardiovascular Diseases"[Mesh] OR "Cerebrovascular Disorders"[Mesh] OR "Atherosclerosis"[Mesh] OR "Stroke"[Mesh] OR "Myocardial Infarction"[Mesh] OR cardiovascul*[tiab] OR cerebrovascul*[tiab] OR vascular[tiab] OR atherosclero*[tiab] OR "atherosclerotic plaque*"[tiab] OR carotid[tiab] OR stroke*[tiab] OR "myocardial infarction"[tiab] OR MACE[tiab] OR "major adverse cardiovascular event*"[tiab] OR endarterectom*[tiab]) AND ("Humans"[Mesh] OR human*[tiab] OR patient*[tiab] OR participant*[tiab]) NOT (animals[mh] NOT humans[mh]) NOT ("Review"[Publication Type] OR "Meta-Analysis"[Publication Type] OR "Case Reports"[Publication Type] OR editorial[pt] OR comment[pt]).

For the Scopus database, the search strategy was as follows:

1. (TITLE-ABS-KEY (microplastic* OR nanoplastic* OR micro-plastic* OR nano-plastic* OR micro-nanoplastic*) AND TITLE-ABS-KEY (cardiovascul* OR cerebrovascul* OR vascular OR atherosclero* OR "atherosclerotic plaque*" OR carotid OR stroke* OR "myocardial infarction" OR MACE OR "major adverse cardiovascular event*" OR endarterectom*) AND NOT DOCTYPE (re OR ed OR no OR le))

As a result of applying the above strategies, a total of 397 records were identified, 135 of which came from PubMed and 262 from Scopus. The results were exported to the Rayyan platform, dedicated to literature review management. Using this platform, 21 duplicates were identified and removed after manual verification by the research team. This process allowed for the identification of 376 unique articles, which were qualified for the next selection stage.

2.1. Inclusion Criteria

Research inclusion criteria were structured based on the PICOS framework (Population, Intervention, Comparison, Outcome, Study design), in accordance with PRISMA guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses). The selected articles aimed to answer the following research question: "What is the association between the presence, concentration or level of exposure to microplastics and nanoplastics and cardiovascular or cerebrovascular events, features of vascular disease and related biological markers in adults?"

The main inclusion criteria were: (1) primary human analytical observational studies, including prospective or retrospective cohort studies, case-control studies, cross-sectional studies, and ecological studies (analyzed separately); (2) a population of adult participants, irrespective of whether they have established cardiovascular disease, including patients undergoing cardiovascular or vascular procedures, individuals from the general population, or cohorts at increased cardiovascular risk; (3) documented presence, concentration, or level of exposure to microplastics and/or nanoplastics, evaluated in biological samples (e.g., blood, tissues, vascular specimens, or atherosclerotic plaques) or determined through individual-level environmental or occupational exposure assessments; (4) presence of a comparison group (absence of detectable microplastics or nanoplastics, lower concentration levels, or comparisons across exposure categories), as well as dose-response or continuous variable exposure analyses; (5) evaluation of relevant cardiovascular outcomes, including major cardiovascular and cerebrovascular events (myocardial infarction, stroke, transient ischemic attack, major adverse cardiovascular events [MACE], revascularization, cardiovascular hospitalization, and cardiovascular death), subclinical, imaging, or histopathological vascular features (plaque burden or instability,

carotid intima-media thickness, vascular calcification, thrombosis, endothelial dysfunction, and vascular injury), or circulating biomarkers of inflammation, oxidative stress, coagulation, or endothelial dysfunction (e.g., hs-CRP, IL-6, and TNF-α).

Exclusion criteria included: (1) literature reviews, systematic reviews, meta-analyses, editorials, commentaries, study protocols, and individual case reports; (2) preclinical in vitro experiments, animal studies, or research conducted exclusively in pediatric populations; (3) studies evaluating general or other environmental pollutants without a distinct, standalone assessment of microplastics or nanoplastics; (4) purely descriptive studies reporting the detection or quantification of microplastics or nanoplastics without analyzing their direct association with clinical, vascular, histopathological, or biological outcomes.

2.2. Study Selection

The literature selection process was carried out in multiple stages, strictly following PRISMA guidelines. Initially, a five-person team of independent reviewers assessed the titles and abstracts of 376 articles, examining their compliance with the main objective of the study. To ensure maximum objectivity and minimize systematic error, this stage was conducted independently on the Rayyan platform. Works failing to meet the basic assumptions of the PICOS protocol were excluded. The next step involved an independent, full-text assessment of the remaining publications by all five authors, based on detailed inclusion and exclusion criteria. Potential disagreements and conflicting assessments were subjected to joint analysis and discussed until a final consensus was reached.

2.3. Data Extraction

The following data were extracted from the qualified publications: DOI number, first author's name, year of publication, study type, number of participants in the study and control groups,control group type and matching, type of control, mean age and gender of the study group, patient characteristics, study duration, clinical diagnosis of patients, comorbidities, tested biological material, plastic detection method, types of plastics detected, particle concentration, quantity and size, hard endpoints, risk estimates, inflammatory markers and plaque features

3. Results

Among the 26 articles included in the analysis, the vast majority comprised prospective observational studies, including cross-sectional, cohort, and case-control designs. The study populations primarily consisted of patients with multivessel atherosclerotic cardiovascular disease, with several studies specifically focusing on outcomes in individuals post-myocardial infarction [12, 13, 18, 20, 22, 32], ischemic stroke, or deep vein thrombosis [13, 15, 33] and atrial fibrillation (AF), or valvular heart disease [28]. The analyzed specimens were predominantly whole blood and vascular biopsies, as well as thrombi [12, 13, 25], left atrial appendage tissue [17, 28], cerebrospinal fluid (CSF) [23], skeletal system tissues [29], corpus cavernosum tissue [30], and umbilical cord tissue [31]. Qualitative and quantitative analysis of micro- and nanoplastics (MNPs) was performed using pyrolysis-gas chromatography-mass spectrometry (Py-GC/MS), scanning electron microscopy (SEM/EM), laser direct infrared (LDIR) spectroscopy, Raman spectroscopy, and micro-Fourier transform infrared spectroscopy (μFTIR). Inflammatory biomarkers were measured using enzyme-linked immunosorbent assay (ELISA) and immunohistochemical techniques.

3.1. Detected types of plastic materials

Among the plastic materials analyzed in the studies, polyethylene, which belongs to the group of olefin polymers proved to be the most prevalent [9–11, 14–16, 18, 19, 21, 24, 25, 27, 28, 30, 31, 33], likely driven by its ubiquitous global production, extensive application in single-use food packaging, and high environmental persistence. Polystyrene was detected second; it most often appears as microplastic due to the degrading effect of UV radiation on the molecule’s brittle benzene ring [8, 12, 17], a photo-oxidative process that promotes structural embrittlement, micro-fragmentation, and subsequent biological uptake via inhalation or ingestion. Less frequently detected components included polyamide-66 (PA-66) from synthetic textiles,

alkyd resins derived from commercial surface coatings, polypropylene (PP), polyvinyl chloride (PVC), and ethylene-vinyl acetate copolymer (EVA).

The human materials used for analysis included whole venous blood [8, 11, 14, 15, 19, 24, 32], arterial blood [12, 18, 26] atherosclerotic plaques from arteries [9, 27], blood clots [13, 25, 33], vascular tissues [10, 16, 17, 21, 28] and cerebrospinal fluid [23] . The identification of plastic particles in venous blood confirms their systemic circulation throughout the vascular network, while their presence within arterial atherosclerotic plaques and intravascular clots points to potential pathophysiological involvement in localized vascular inflammation and cardiovascular disease. Notably, the detection of micro- and nanoplastic fractions in human cerebrospinal fluid indicates that these microscopic contaminants are capable of crossing the protective blood-brain barrier, raising critical concerns regarding neurotoxicity and central nervous system health.

3.2. Concentration and size of the detected plastic particles

Most included studies reported variable concentrations and sizes of micro- and nanoplastics (MNPs) across analyzed tissues and fluids, indicating preferential accumulation in diseased tissues compared to healthy controls. Cui et al. demonstrated that mean total MNP concentrations in carotid atherosclerotic plaques (432.9 μg/g) were nearly six times higher than in paired peripheral blood samples (75.2 μg/g). Similarly, Massie et al. observed an 80-times higher accumulation in femoral artery plaques (3234 ± 3629 μg/g) compared to age-matched healthy vessels (40.68 ± 23.23 μg/g). Furthermore, Marfella et al. reported plaque concentrations of 21.7 ± 24.5 μg/mg for polyethylene (PE) and 5.2 ± 2.4 μg/mg for polyvinyl chloride (PVC).

Polymer concentrations significantly correlated with cardiovascular disease severity. Yu et al. observed elevated blood MNP levels in patients with extracranial artery stenosis (174.89 ± 24.95 μg/g vs. 79.82 ± 31.73 μg/g in controls). Similarly, Yang et al. reported blood MNP concentrations averaging 150.08 μg/g (up to 413.99 μg/g) in acute coronary syndrome patients, with the highest levels among post-myocardial infarction cases. In cardiac tissue, Pan et al. identified higher MNP burden in the left atrial appendage of patients with severe myocardial fibrosis compared to those with mild fibrosis (171.74 vs. 119.33 μg/g).

Particle size analysis revealed marked heterogeneity, demonstrating biological barrier penetration by both micro- and nanometric fractions. While several studies documented micrometric particles ranging from 2 to 500 μm, Leonard et al. and Rotchell et al. identified surprisingly large fragments in blood and venous tissue, with mean lengths of 127.99 ± 293.26 μm (ranging up to 3000 μm) and 122.8 μm, respectively. On the contrary, studies by Zhang et al. and Paolisso et al. confirmed a substantial presence of submicrometric and nanometric fractions (< 1 μm and < 0.5 μm) capable of systemic translocation.

3.3. Cardiovascular and cerebrovascular risk factors

In the study by Lian et al. [10], MNPs were detected in 76% of analyzed arterial wall specimens obtained from patients undergoing vascular interventions for atherosclerotic cardiovascular disease, exhibiting the highest degree of accumulation within the carotid artery. MNP concentrations were shown to be approximately six times higher in atherosclerotic plaques compared to whole blood [14] and up to 80-fold higher than in healthy arterial walls, independent of patient age [16, 19]. Furthermore, MNPs were present in saphenous vein walls [20, 22, 32], peripheral blood [21, 24], and thrombi [25, 33]. MNPs were also identified in CSF, where their presence was associated with an increased risk of intracranial aneurysm [23]. MNP accumulation was elevated in patients presenting with acute ischemic stroke at admission or during thrombolytic therapy compared to healthy controls [15].

Vascular tissue and blood MNP accumulation was associated with an increased risk of cardiovascular events across three observational studies [8, 9, 12]. Additionally, Yu et al. [26] demonstrated a positive correlation between blood MNP concentrations and the severity of extracranial artery stenosis, while Goldsworth et al. [27] in modelling study using secondary data highlighted their impact on mortality, morbidity, and healthcare costs in vascular surgery.

Studies also emphasized an association between higher blood MNP concentrations and acute coronary syndrome [11], ST-segment elevation myocardial infarction (STEMI) [18], thromboembolic disease [13], myocardial fibrosis [17], pregnancy-induced hypertension [31], and AF [28]. In the latter analysis, the authors reported that MNPs were present in all analyzed left atrial appendage biopsy specimens.

The majority of reviewed studies suggested a potential role for vascular tissue MNP accumulation in the pathogenesis of atherosclerotic plaque.

3.4. Inflammatory markers associated with exposure to plastic materials

Increasing evidence suggests that exposure to microplastics (MPs) and nanoplastics (NPs) is associated with enhanced systemic and local inflammatory responses. Across the included studies, the most consistently reported findings were elevated concentrations of pro-inflammatory cytokines, particularly interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), and interleukin-18 (IL-18), accompanied by increased immune cell infiltration within vascular tissues and features of plaque vulnerability.

IL-6 was the most frequently reported inflammatory biomarker. Several studies demonstrated a significant positive association between MP burden and circulating or tissue IL-6 concentrations. Higher levels of MPs were associated with elevated serum IL-6 together with increased oxidative stress and endothelial injury markers [8]. Similarly, increased MP concentrations in peripheral blood correlated with significantly higher IL-6 and IL-12p70 levels, accompanied by increased numbers of B lymphocytes and natural killer (NK) cells, indicating activation of both innate and adaptive immune responses [11]. Comparable findings were reported in coronary blood samples, where the presence of MPs was positively correlated with IL-6 concentrations [19]. Likewise, vascular tissue analyses demonstrated a significant relationship between total MP burden and local IL-6 expression, supporting persistent vascular inflammation associated with plastic particle deposition [22].

Tumor necrosis factor-α represented another consistently elevated inflammatory mediator. Increased TNF-α concentrations were observed both in experimental clinical cohorts and in vascular tissues containing MPs [8, 12, 19, 22]. Positive correlations between polyvinyl chloride (PVC) concentration and TNF-α expression were additionally accompanied by increased macrophage infiltration within coronary thrombi, suggesting enhanced inflammatory activation at sites of acute coronary occlusion [12].

Several studies also identified activation of the IL-1 signaling pathway. Higher concentrations of PVC within thrombotic material were associated with increased IL-1β and IL-18 expression [12]. Similarly, carotid atherosclerotic plaques containing MPs exhibited markedly increased expression of IL-1β and IL-18 together with abundant CD68-positive macrophages and CD3-positive T lymphocytes, indicating a highly inflammatory plaque phenotype characterized by reduced collagen content and greater structural instability [9].

Beyond individual cytokines, multiple investigations demonstrated evidence of broader immune activation. Increased infiltration of macrophages and lymphocytes was consistently observed in tissues containing MPs [9 ,12, 23]. Histological analyses revealed inflammatory infiltrates distributed throughout all layers of the vascular wall, including the intima, media, and adventitia, with plastic particles embedded within the extracellular matrix, suggesting chronic vascular exposure and persistent immune stimulation [23]. Additional transcriptomic analyses identified activation of several inflammation-related signaling pathways, including TNF/NF-κB, TGF-β, and MAPK, together with increased expression of transforming growth factor-β1 (TGF-β1), α-smooth muscle actin (α-SMA), and type I collagen, indicating concomitant inflammatory activation and vascular remodeling [18].

Several studies also highlighted oxidative stress as an important component of MP-induced vascular injury. Increased reactive oxygen species (ROS), mitochondrial dysfunction, endothelial to mesenchymal transition (EndMT), macrophage ferroptosis, and activation of the cGAS-STING pathway were reported in association with plastic particle accumulation, suggesting that oxidative stress and innate immune activation may jointly promote vascular fibrosis and plaque progression [31].

Not all inflammatory biomarkers were consistently altered. In one study evaluating patients with extracranial carotid artery stenosis, D-dimer concentrations were significantly increased whereas no differences were observed for C-reactive protein (CRP) or fibrinogen between groups [20]. Similarly, several investigations focused primarily on particle detection or lipid metabolism without assessing inflammatory mediators [10 ,13, 14, 15, 16].

Overall, the available evidence consistently indicates that MPs and NPs are associated with activation of proinflammatory cytokine networks, immune cell recruitment, oxidative stress, and signaling pathways involved in vascular inflammation and plaque remodeling. Although substantial heterogeneity exists across studies, IL-6 and TNF-α appear to be the most reproducible inflammatory biomarkers associated with plastic particle exposure.

4. Discussion

This systematic review summarizes the emerging clinical evidence on the presence of micro- and nanoplastics (MNPs) in the human cardiovascular system and their potential association with cardiovascular and cerebrovascular disease. MNPs have been detected in circulating blood, arterial walls, atherosclerotic plaques, vascular thrombi, saphenous vein tissue, left atrial appendage specimens, and cerebrospinal fluid [9–14, 16–26, 28, 32, 33]. Higher MNP burdens were reported in patients with advanced atherosclerosis, acute coronary syndromes, myocardial infarction, extracranial arterial stenosis, ischemic stroke, and thromboembolic disease [9, 11–13, 15, 16, 18, 23, 26, 33]. These findings, together with evidence of inflammatory and oxidative responses, support the biological plausibility of an association between MNP exposure and vascular pathology. However, the predominantly observational and cross-sectional nature of the evidence precludes causal inference.

The detection of MNPs in both blood and cardiovascular tissues suggests that environmental plastic particles may enter the systemic circulation and reach anatomically distant sites. Polyethylene was the most frequently identified polymer, followed by polyvinyl chloride, polypropylene, polystyrene, and polyamide-66 [9–14, 16, 18–21, 24–26, 28, 31, 33]. Differences in polymer profiles and reported concentrations may reflect variation in exposure and tissue distribution, but also substantial heterogeneity in sample collection, contamination-control procedures, analytical techniques, particle-size thresholds, and reporting units.

A particularly relevant finding was the preferential accumulation of MNPs in diseased vascular tissues. Concentrations were higher in carotid atherosclerotic plaques than in paired blood samples [14, 19] and markedly higher in femoral plaques than in age-matched non-atherosclerotic arterial tissue [16]. MNPs were also detected within saphenous vein specimens obtained during coronary artery bypass grafting [20, 22, 32]. Endothelial dysfunction, increased vascular permeability, lipid deposition, and macrophage recruitment may promote particle retention within the arterial wall. Conversely, deposited particles may intensify inflammation and oxidative injury. Because these studies were predominantly cross-sectional, it remains uncertain whether MNPs contribute to lesion development or preferentially accumulate in previously damaged tissues.

The strongest longitudinal evidence was provided by Marfella et al. [9]. Among patients undergoing carotid endarterectomy, the presence of polyethylene or polyvinyl chloride in excised plaques was associated with an increased risk of myocardial infarction, ischemic stroke, or death from any cause during approximately 34 months of follow-up (adjusted HR 4.53, 95% CI 2.00–10.27). MNP-containing plaques also exhibited increased expression of IL-18, IL-1β, TNF-α, and inflammatory-cell markers [9]. Similarly, in patients with myocardial infarction, higher coronary-blood PVC concentrations were associated with subsequent major adverse cardiovascular events [12]. Nevertheless, residual confounding by smoking, diet, occupational exposure, air pollution, and socioeconomic factors cannot be excluded.

Inflammation and oxidative stress may represent key mechanisms linking MNP exposure to cardiovascular injury. Higher particle burdens were associated with increased IL-6, TNF-α, IL-1β, IL-18, and IL-12p70 concentrations, as well as macrophage and lymphocyte infiltration [8, 9, 11, 12, 18, 21, 22]. Activation of TNF/NF-κB, TGF-β, and MAPK signaling has also been reported in association with myocardial inflammation and fibrosis [17]. These processes may promote endothelial dysfunction, vascular remodeling, and plaque instability. However, biomarker findings were not fully consistent: patients with extracranial arterial stenosis demonstrated higher D-dimer concentrations, whereas CRP and fibrinogen did not differ significantly between groups [26].

MNPs may also be involved in thromboinflammatory processes. Plastic particles have been identified in coronary, cerebral, arterial, and venous thrombi [12, 13, 25, 33], and higher concentrations were associated with greater disease severity in some studies [13, 25]. Potential mechanisms include endothelial injury, platelet activation, coagulation-pathway activation, and interaction with the fibrin network. Nevertheless, particles may also become passively incorporated into an already-forming thrombus; therefore, their detection does not demonstrate that they initiated thrombosis.

Evidence concerning cerebrovascular outcomes remains limited. Patients with acute ischemic stroke had higher blood microplastic concentrations than healthy controls in a small pilot study [15], while MNP burden was associated with the presence and severity of extracranial arterial stenosis [26]. MNPs were also detected in cerebrospinal fluid, where higher concentrations of selected polymers were associated with intracranial aneurysm [23]. However, the small and heterogeneous populations, as well as the use of patients with leptomeningeal metastases as controls in the cerebrospinal-fluid study, limit interpretation. Detection in

cerebrospinal fluid suggests possible access to the central nervous system but does not establish direct passage across an intact blood-brain barrier.

Several limitations should be considered. Most studies included small, highly selected clinical populations and lacked healthy or appropriately matched controls. Analytical heterogeneity across Py-GC/MS, μFTIR, Raman spectroscopy, LDIR, and electron microscopy substantially limits comparisons between studies. Contamination during sample collection and processing remains an important concern, despite the use of procedural blanks in several investigations [20, 22, 24, 25, 32, 33]. Furthermore, environmental exposure and major potential confounders were assessed inconsistently, and reverse causation cannot be excluded.

Overall, current human evidence indicates that MNPs are present throughout the cardiovascular system and may be associated with atherosclerosis, inflammation, vascular remodeling, thrombosis, and adverse cardiovascular outcomes. Nevertheless, the available evidence is insufficient to establish causality or support routine clinical measurement of MNP burden. Large prospective studies using standardized analytical and reporting protocols are required to establish temporality, dose-response relationships, and the independent contribution of MNP exposure to cardiovascular and cerebrovascular risk.

5. Conclusions

This systematic review demonstrates that micro- and nanoplastics (MNPs) are detectable in circulating blood and across multiple components of the human cardiovascular system, including arterial walls, atherosclerotic plaques, cardiac tissue, saphenous veins, and vascular thrombi. Their presence in cerebrospinal fluid further suggests that MNP distribution may extend beyond the systemic circulation to the central nervous system. Polyethylene and polyvinyl chloride were the most frequently identified polymers, although substantial differences in polymer profiles, concentrations, and particle sizes were reported across studies.

Current clinical evidence indicates that a higher MNP burden may be associated with advanced atherosclerosis, acute coronary syndromes, myocardial infarction, extracranial arterial stenosis, ischemic stroke, thromboembolic disease, and adverse cardiovascular outcomes. Preferential accumulation within diseased vascular tissues suggests a potentially relevant relationship between MNP deposition and vascular pathology. Inflammatory and oxidative mechanisms – including endothelial injury, immune-cell recruitment, activation of pro-inflammatory cytokine pathways, vascular remodeling, and thromboinflammatory responses – may contribute to these associations. Furthermore, the potential role of MNPs as vector carriers for adsorbed chemical toxicant additive compounds represents an additional layer of pathological complexity. Nevertheless, it remains unclear whether MNPs directly promote cardiovascular disease, serve as markers of broader environmental exposure, or preferentially accumulate in previously damaged tissues.

Interpretation of the available evidence is limited by the predominance of observational and cross-sectional designs, small and selected study populations, inconsistent adjustment for confounding factors, potential sample contamination, and considerable heterogeneity in analytical and reporting methods. In particular, the lack of standardized, plastic-free tissue harvesting and extraction protocols presents a major challenge for quantitative cross-study comparisons.

Therefore, the current findings do not establish causality or justify routine clinical measurement of MNP burden. Large prospective studies using standardized detection protocols, rigorous contamination control, and harmonized outcome definitions are required to clarify temporality, dose-response relationships, and the independent contribution of MNP exposure to cardiovascular and cerebrovascular risk. Ultimately, bridging these analytical and clinical gaps will be critical for determining whether MNP pollution should be formally integrated into cardiovascular risk stratification frameworks and targeted by environmental public health policies.

Author Contributions:

Conceptualization, W.Ż, K.Ś, M.L; methodology, K.Ś,, J.T, J.S, W.Ż and M.L; validation, K.Ś and W.Ż.; formal analysis, K.Ś, W.Ż, M.L ; resources, K.Ś, J.T, J.S, W.Ż and M.L.; data curation,K.Ś, J.T, J.S, W.Ż, M.L; writing—original draft preparation, K.Ś, J.T, J.S, W.Ż. and M.L.; writing—review and editing, K.Ś; visualization, K.Ś, J.T, J.S, W.Ż and M.L.; supervision, K.Ś and W.Ż.; project administration, K.Ś and W.Ż .; funding acquisition, K.Ś, W.Ż. , J.T., J.S, M.L. All authors have read and agreed to the published version of the manuscript.

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