

Original Article

Impact of Microplastic Particle Exposure on Endothelial Dysfunction and Early Atherosclerotic Changes in Urban Adults

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ABSTRACT

Background: Microplastics are widespread environmental contaminants, but their relationship with endothelial dysfunction and subclinical arterial changes in humans remains uncertain. **Objective:** To examine the association of microplastic concentration with serum sICAM-1, ADMA, and mean cIMT among urban adults. **Methods:** This analytical cross-sectional study included 88 adults aged 30–50 years from the industrial and urban core of Punjab, Pakistan, between August 2025 and January 2026. Blood microplastics were quantified using micro-Raman spectroscopy combined with pyrolysis–gas chromatography–mass spectrometry. Serum sICAM-1 and ADMA were measured using ELISA, and cat was assessed using B-mode ultrasonography. Median-defined groups were compared using Welch's t-tests, and continuous associations were evaluated using Pearson correlations. **Results:** Microplastics were detected in all participants, with a mean concentration of 2.45 ± 0.81 particles/mL. Compared with the lower-concentration group, the higher-concentration group had greater sICAM-1 (mean difference: 74.9 ng/mL; 95% CI: 58.7–91.1), ADMA (0.21 μ mol/L; 95% CI: 0.16–0.26), and mean cIMT (0.13 mm; 95% CI: 0.10–0.16); all $p < 0.001$. Microplastic concentration correlated positively with sICAM-1 ($r = 0.684$), ADMA ($r = 0.592$), and cIMT ($r = 0.615$); all $p < 0.001$. **Conclusion:** Higher blood microplastic concentrations were associated with endothelial dysfunction markers and greater cIMT. Longitudinal studies are required to establish temporality and clinical significance. **Keywords:** ADMA; atherosclerosis; carotid intima-media thickness; endothelial dysfunction; microplastics; sICAM-1.

INTRODUCTION

The rapid growth of plastic production and consumption has resulted in widespread environmental accumulation of plastic waste. Through mechanical abrasion, ultraviolet degradation, and biological processes, larger plastic

materials fragment into microplastics, conventionally defined as plastic particles smaller than 5 mm. These contaminants have been identified in air, water, food, and terrestrial and aquatic ecosystems, creating multiple opportunities for human exposure through inhalation and ingestion. Microplastics and associated chemical additives may subsequently interact with biological systems, although the extent of their absorption, persistence, and health consequences in humans remains uncertain (1–4).

The cardiovascular system may be particularly relevant to the investigation of circulating microplastics because the vascular endothelium regulates vascular tone, inflammatory activity, coagulation, and leukocyte adhesion. Experimental evidence suggests that micro- and nanoplastics may promote oxidative stress, inflammatory signalling, mitochondrial disturbance, and impaired nitric oxide bioavailability. These processes could contribute to endothelial activation and, over time, structural vascular abnormalities. Reviews of microplastics in vascular disease and the broader cardiovascular effects of environmental pollution support the biological plausibility of such associations; however, direct human evidence remains limited and does not yet establish causality (5,6).

Endothelial dysfunction precedes clinically apparent atherosclerotic disease and can be evaluated using circulating and structural markers. Soluble intercellular adhesion molecule-1 (sICAM-1) reflects endothelial activation and leukocyte-adhesion signaling, whereas asymmetric dimethylarginine (ADMA) is an endogenous inhibitor of nitric oxide synthase associated with impaired endothelial function. Carotid intima-media thickness (cIMT) provides a non-invasive surrogate measure of subclinical arterial-wall change. Laboratory and animal studies have linked microplastic exposure with oxidative and inflammatory cellular responses, but the translation of these findings to human vascular health remains uncertain (7). Emerging clinical research has reported associations between microplastic burden and indicators of atherosclerotic risk in patients with acute coronary syndrome; nevertheless, findings from patients with established cardiovascular disease cannot be directly generalized to adults without diagnosed cardiovascular disease or other major clinical conditions (8).

Urban populations may encounter microplastics from traffic-related emissions, industrial activity, degraded waste, contaminated water, and plastic food-contact materials. These exposures may coexist with other environmental determinants of cardiovascular health, making carefully controlled human biomonitoring studies important (9). Existing experimental evidence across different organ systems provides a rationale for concern but also emphasizes uncertainty regarding particle size, polymer composition, exposure duration, internal dose, and clinically relevant outcomes (10). In particular, limited human evidence has simultaneously examined measured blood microplastic concentrations, circulating markers of endothelial dysfunction, and cIMT in urban adults without established cardiovascular disease, diabetes, chronic kidney disease, or active smoking.

Therefore, this analytical cross-sectional study aimed to examine the association of blood microplastic concentration with serum sICAM-1, serum ADMA, and mean cIMT among urban adults aged 30–50 years residing in the industrial and urban core of Punjab, Pakistan. The study evaluated continuous exposure–outcome relationships and compared vascular measures between median-defined lower and higher microplastic-concentration groups. It was hypothesized that higher blood microplastic concentrations would be positively associated with sICAM-1, ADMA, and mean cIMT.

MATERIALS AND METHODS

An analytical cross-sectional study was conducted to examine contemporaneous associations between blood microplastic concentration, biomarkers of endothelial dysfunction, and subclinical arterial changes among urban adults. Data were collected from August 2025 to January 2026 in the industrial and urban core of Punjab, Pakistan. This setting was selected because its population may encounter microplastic contamination from industrial activity, vehicular traffic, plastic use, and municipal waste. The target sample comprised urban residents aged 30–50 years who had lived continuously in the study area for at least five years.

Potential participants were screened against predefined eligibility criteria during the study period. Of the 95 adults screened, 88 met the eligibility criteria and completed the data-collection procedures. Adults with established cardiovascular disease, diabetes mellitus, chronic kidney disease, any history of smoking, or current use of lipid-lowering or anti-inflammatory medication were excluded to reduce confounding by major cardiovascular conditions and related treatments. The planned sample of 88 participants was based on previous pilot research examining environmental particulate exposure and vascular reactivity and was selected to balance the anticipated ability to

detect differences in endothelial measures with the requirements of specialized spectroscopic and laboratory processing.

Demographic and clinical information included age, sex, body mass index, systolic blood pressure, and total cholesterol. Following an overnight fast, peripheral blood samples were collected using glass syringes and stored in specialized glass vials to reduce external plastic contamination during specimen collection and handling. Blood microplastic concentration was expressed as particles per millilitre and served as the principal exposure variable. Microplastic particles were assessed using micro-Raman spectroscopy in combination with pyrolysis–gas chromatography–mass spectrometry. These procedures were used to characterize the polymer composition and quantify particles smaller than 5 µm, including polyethylene and polystyrene.

Endothelial function was evaluated using serum soluble intercellular adhesion molecule-1 (sICAM-1) and asymmetric dimethylarginine (ADMA). Concentrations of both biomarkers were measured using enzyme-linked immunosorbent assays and expressed as ng/mL and µmol/L, respectively. Early structural arterial change was evaluated through carotid intima-media thickness (cIMT). High-resolution B-mode ultrasonography was used to examine the right and left common carotid arteries, and the bilateral measurements were used to determine mean cIMT in millimetres. All ultrasound examinations were performed by a single blinded sonographer to promote consistency and reduce measurement-related observer bias.

Blood microplastic concentration was analysed as a continuous exposure variable when examining correlations with vascular outcomes. For secondary group comparisons, participants were divided at the sample median into a lower-concentration group of less than 2.38 particles/mL and a higher-concentration group of at least 2.38 particles/mL, with 44 participants in each group. The outcomes were serum sICAM-1 concentration, serum ADMA concentration, and mean cIMT. The use of restrictive eligibility criteria, glass blood-collection equipment, standardized laboratory assessments, and a single blinded sonographer constituted the principal measures used to reduce clinical confounding, external plastic contamination, and outcome-assessment variability.

The distributions of continuous variables were evaluated using the Shapiro–Wilk test. Normally distributed continuous data were summarized as mean and standard deviation, whereas categorical variables were summarized as frequency and percentage. Independent-samples t-tests were used to compare sICAM-1, ADMA, and mean cIMT between the lower- and higher-concentration groups. Pearson correlation coefficients were calculated to assess the direction and magnitude of associations between continuous blood microplastic concentration and each vascular outcome. Multiple linear regression models were constructed to evaluate whether blood microplastic concentration remained associated with sICAM-1, ADMA, and mean cIMT after accounting for measured demographic variation. Statistical tests were two-sided, and a p-value below 0.05 was considered statistically significant. Analyses included the 88 participants who met the eligibility criteria and completed the data-collection procedures.

Institutional review board approval was obtained before the study commenced. Participant information was handled confidentially throughout data collection, laboratory assessment, analysis, and reporting.

RESULTS

Of the 95 adults screened, 88 met the eligibility criteria and completed the study procedures, corresponding to 92.6% of those screened. The participants had a mean age of 39.4 ± 5.8 years, and 46 (52.3%) were men. The median-based lower- and higher-concentration groups each included 44 participants. Age, sex, BMI, systolic blood pressure, and total cholesterol did not differ appreciably between the groups, with all p-values exceeding 0.30 (Table 1).

Microplastic particles were detected in the blood samples of all 88 participants. The mean blood microplastic concentration was 2.45 ± 0.81 particles/mL, with polyethylene and polystyrene identified as the predominant polymer types. Mean concentrations were 1.78 ± 0.34 particles/mL in the lower-concentration group and 3.12 ± 0.52 particles/mL in the higher-concentration group.

Table 1. Demographic and clinical characteristics according to blood microplastic concentration (N = 88)

Variable	Total (N = 88)	Lower concentration (n = 44)	Higher concentration (n = 44)	Mean difference (95% CI)	p-value
Age, years	39.4 ± 5.8	38.9 ± 5.6	39.9 ± 6.0	1.0 (−1.5 to 3.5)	0.421
Male sex, n (%)	46 (52.3)	22 (50.0)	24 (54.5)	—	0.831
BMI, kg/m ²	24.1 ± 2.4	23.9 ± 2.2	24.3 ± 2.6	0.4 (−0.6 to 1.4)	0.438
Systolic blood pressure, mmHg	118.4 ± 7.2	117.9 ± 6.8	118.9 ± 7.6	1.0 (−2.1 to 4.1)	0.517
Total cholesterol, mg/dL	182.3 ± 15.4	180.6 ± 14.2	184.0 ± 16.5	3.4 (−3.1 to 9.9)	0.303
Blood microplastic concentration, particles/mL	2.45 ± 0.81	1.78 ± 0.34	3.12 ± 0.52	—	—

Values are mean ± SD unless otherwise indicated. Mean differences are higher minus lower concentration group. Continuous variables were compared using Welch's independent-samples t-test; sex was compared using Fisher's exact test. Groups were defined by the median blood microplastic concentration: lower, <2.38 particles/mL; higher, ≥2.38 particles/mL. No inferential comparison was performed for blood microplastic concentration because this variable defined the groups. BMI, body mass index; CI, confidence interval; SD, standard deviation.

Table 2. Endothelial and arterial outcomes according to blood microplastic concentration

Outcome	Lower concentration (n = 44), mean ± SD	Higher concentration (n = 44), mean ± SD	Mean difference (95% CI)	t-statistic	Hedges' g (95% CI)	p-value
sICAM-1, ng/mL	210.5 ± 34.2	285.4 ± 41.8	74.9 (58.7 to 91.1)	9.20	1.94 (1.44 to 2.45)	<0.001
ADMA, μmol/L	0.48 ± 0.09	0.69 ± 0.12	0.21 (0.16 to 0.26)	9.29	1.96 (1.46 to 2.47)	<0.001
Mean cIMT, mm	0.58 ± 0.06	0.71 ± 0.08	0.13 (0.10 to 0.16)	8.62	1.82 (1.33 to 2.32)	<0.001

Mean differences are higher minus lower concentration group. Estimates were calculated from the reported group means, SDs, and sample sizes using Welch's independent-samples t-test. Hedges' g represents the standardized mean difference with small-sample correction. ADMA, asymmetric dimethylarginine; CI, confidence interval; cIMT, carotid intima-media thickness; SD, standard deviation; sICAM-1, soluble intercellular adhesion molecule-1.

Table 3. Pearson correlations among blood microplastic concentration and vascular outcomes (N = 88)

Variable pair	Pearson r	95% CI	t-statistic	p-value
Microplastic concentration–sICAM-1	0.684	0.554 to 0.781	8.70	<0.001
Microplastic concentration–ADMA	0.592	0.437 to 0.713	6.81	<0.001
Microplastic concentration–mean cIMT	0.615	0.466 to 0.730	7.23	<0.001
sICAM-1–ADMA	0.514	0.341 to 0.653	5.56	<0.001
sICAM-1–mean cIMT	0.553	0.389 to 0.683	6.16	<0.001
ADMA–mean cIMT	0.488	0.310 to 0.633	5.18	<0.001

Confidence intervals were calculated using Fisher's z transformation. Tests used 86 degrees of freedom. ADMA, asymmetric dimethylarginine; CI, confidence interval; cIMT, carotid intima-media thickness; sICAM-1, soluble intercellular adhesion molecule-1.

Participants in the higher-concentration group had a mean sICAM-1 concentration of 285.4 ± 41.8 ng/mL compared with 210.5 ± 34.2 ng/mL in the lower-concentration group. The mean difference was 74.9 ng/mL (95% CI, 58.7–91.1; p<0.001), with a standardized mean difference of 1.94 (95% CI, 1.44–2.45). Mean ADMA concentration was also greater in the higher-concentration group, with a between-group difference of 0.21 μmol/L (95% CI, 0.16–0.26; p<0.001; Hedges' g = 1.96). Mean cIMT was 0.71 ± 0.08 mm in the higher-concentration group and 0.58 ± 0.06 mm in the lower-concentration group, corresponding to a mean difference of 0.13 mm (95% CI, 0.10–0.16; p<0.001; Hedges' g = 1.82) (Table 2). These standardized differences were numerically large, although their interpretation is limited to the cross-sectional group comparison.

Blood microplastic concentration was positively correlated with all three vascular outcomes. The strongest correlation was observed with sICAM-1 (r = 0.684; 95% CI, 0.554–0.781; p<0.001), followed by mean cIMT (r = 0.615; 95% CI, 0.466–0.730; p<0.001) and ADMA (r = 0.592; 95% CI, 0.437–0.713; p<0.001). Positive correlations were also identified among sICAM-1, ADMA, and mean cIMT, with coefficients ranging from 0.488 to 0.553 (all p<0.001) (Table 3).

The manuscript's claimed adjusted regression findings cannot yet be presented because no regression coefficients, confidence intervals, covariate list, model fit statistics, or statistical output were supplied. All

statements describing microplastic concentration as an “independent predictor” or “independent risk factor” should remain excluded until those results are provided.

DISCUSSION

This analytical cross-sectional study identified positive associations between blood microplastic concentration and three indicators of vascular health among urban adults aged 30–50 years. Participants in the higher-concentration group had greater serum sICAM-1 and ADMA concentrations and greater mean cIMT than those in the lower-concentration group. The corresponding continuous correlations indicated that the findings were not limited to the median-based exposure classification. Collectively, these results suggest that a greater measured blood microplastic burden is associated with endothelial activation, impaired nitric oxide-related regulation, and subclinical arterial-wall alteration. However, the cross-sectional design does not establish whether microplastic exposure preceded or caused these vascular findings.

The observed association between blood microplastic concentration and cIMT is broadly consistent with emerging clinical evidence linking microplastics with vascular pathology. Yang et al. reported that microplastics were associated with greater atherosclerotic risk and vascular complexity among patients with acute coronary syndrome (8). The present study examined a younger population without established cardiovascular disease, diabetes, chronic kidney disease, or smoking history and used cIMT as a surrogate measure of subclinical arterial change. Although these differences in population and outcome assessment limit direct comparison, findings from both studies support further investigation of microplastics in relation to human vascular health. Reviews of micro- and nanoplastics in vascular disease similarly describe potential relationships with endothelial injury, inflammation, thrombosis, and atherosclerotic processes, while emphasizing that the available human evidence remains limited (5).

Several biological mechanisms could plausibly explain the observed associations, although none was directly tested in this study. Experimental research suggests that micro- and nanoplastics may promote reactive oxygen species production, mitochondrial dysfunction, inflammatory signalling, and disruption of endothelial-cell homeostasis. Plastic particles may also transport adsorbed metals, plasticizers, and other environmental contaminants that could contribute to biological effects after internalization (3,4,7,10). Increased sICAM-1 may reflect endothelial activation and enhanced leukocyte-adhesion signalling, whereas increased ADMA may indicate reduced nitric oxide synthase activity and impaired nitric oxide bioavailability. Both processes are biologically relevant to endothelial dysfunction and the early development of vascular disease (11). Nevertheless, the simultaneous measurement of exposure and outcomes means that these mechanisms remain explanatory hypotheses rather than demonstrated pathways in the present participants.

The positive correlation between microplastic concentration and mean cIMT may indicate that circulating particle burden is related to early arterial-wall alteration. However, cIMT is a surrogate vascular measure and should not be interpreted as direct confirmation of microplastic-induced atherosclerosis. The association may also reflect environmental or behavioural factors correlated with microplastic exposure. Urban residents can experience concurrent exposure to ambient particulate matter, industrial emissions, metals, persistent organic pollutants, and other contaminants that have recognized cardiovascular effects (6,9). Diet, occupation, socioeconomic conditions, physical activity, passive smoking, drinking-water source, and the use of plastic food-contact materials may likewise influence either microplastic burden or vascular health. These factors were not comprehensively measured or adjusted for and could partly account for the observed relationships.

The findings have potential scientific and public-health relevance because microplastic exposure is widespread and potentially modifiable at the environmental level. However, the present results do not establish a safe or harmful blood concentration threshold and do not support cardiovascular screening or clinical treatment decisions based on microplastic measurements. Instead, they provide hypothesis-generating human data that support further standardization of microplastic biomonitoring and investigation of vascular outcomes. Broader evidence concerning microplastic exposure and non-communicable diseases also highlights the need to distinguish biological plausibility from demonstrated human disease risk (1,2).

The study has several strengths. Blood microplastic concentration, two complementary circulating endothelial markers, and a structural vascular measure were assessed within the same population. Exclusion of participants

with established cardiovascular disease, diabetes, chronic kidney disease, smoking history, or selected medication use reduced the influence of several important clinical factors. The use of glass blood-collection equipment was intended to limit external plastic contamination, and cIMT assessment by a single blinded sonographer may have reduced interobserver variability. Analysis of microplastic concentration as a continuous variable also provided more information than reliance on the median-based group comparison alone.

These findings should nevertheless be interpreted in view of important limitations. The cross-sectional design precluded assessment of temporality and introduced the possibility of reverse causation. A single blood measurement may not represent long-term exposure because the persistence, distribution, and within-person variability of circulating microplastics remain uncertain. The sample was relatively small, geographically restricted, and limited to adults aged 30–50 years, reducing generalizability to other populations. Participant recruitment and the sampling frame were not sufficiently characterized to exclude selection bias. Although several cardiovascular conditions were addressed through eligibility criteria, residual confounding from demographic, behavioural, dietary, occupational, and environmental factors remains possible. Median dichotomization produced a sample-specific threshold, reduced exposure information, and should therefore be regarded as a secondary analysis.

Additional limitations concern exposure and outcome measurement. Interpretation of universal microplastic detection depends on the analytical detection limits, procedural blanks, recovery experiments, and blank-correction procedures. Polymer-specific concentrations and particle-size distributions were not presented, and the integration of micro-Raman spectroscopy with pyrolysis–gas chromatography–mass spectrometry requires detailed methodological reporting. Likewise, measurement error in ELISA biomarkers or cIMT could have influenced the estimates. Finally, the reported regression analyses could not be evaluated because adjusted coefficients, confidence intervals, covariates, model fit, and diagnostic results were not presented. Consequently, the findings support associations but do not establish blood microplastic concentration as an independent cardiovascular risk factor.

Future investigations should use larger, multicentre longitudinal cohorts with repeated exposure measurements, standardized contamination-control procedures, and detailed reporting of polymer type, particle size, mass, and concentration. Comprehensive measurement of co-pollutants and conventional cardiovascular determinants will be necessary to assess residual confounding. Prespecified multivariable models should evaluate continuous exposure–outcome relationships and test whether associations persist after appropriate adjustment. Longitudinal follow-up would also help determine whether measured microplastic burden precedes changes in endothelial biomarkers, progression of cIMT, or clinically relevant cardiovascular outcomes.

CONCLUSION

Higher blood microplastic concentrations were associated with greater serum sICAM-1 and ADMA concentrations and increased mean cIMT among urban adults aged 30–50 years. These findings suggest a relationship between measured microplastic burden, endothelial dysfunction markers, and subclinical arterial-wall alteration, but they do not establish causality or cardiovascular risk. Larger longitudinal studies with standardized exposure assessment and comprehensive control of confounding are required to determine the temporal and clinical significance of these associations.

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