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Association of faecal and urinary micro- and nanoplastics with markers of gut integrity and renal function

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ABSTRACT

Micro and nanoplastics (MNPs) constitute an emerging environmental concern due to their persistence and ubiquity, but evidence on health effects remains limited. We explored the association between MNPs exposure and markers of gut integrity and renal function in a cross-sectional study in Barcelona (Spain). We enrolled 50 healthy adults who self-collected spot urine and stool samples. MNPs (0.7-20 μm) were measured using high-performance liquid chromatography coupled to high-resolution mass spectrometry (HPLC-HRMS). Calprotectin, zonulin, lysozyme, β-defensin-2 and lactoferrin were measured in stool samples via ELISA. Urea, creatinine and albumin were measured in urine through kinetic and immunoturbidimetric methods. Generalized linear models adjusting for potential confounders were performed to evaluate the association between MNP presence in biological samples and the mentioned biomarkers. Six MNP polymers were detected across 52% of stool and 48% of urine samples. Polyamide was the most frequently detected polymer in stool (34%) and urine samples (16%). Polypropylene (PP) detection in stool was negatively associated with calprotectin (Ratio of means-ROM [95%CI] = 0.52 [0.31-0.94]). In urine, the presence of any MNP polymer was positively associated with the albumin-creatinine ratio (uACR) (ROM [95%CI] = 2.18 [1.17–4.01]). Negative associations between PP in stool and lactoferrin, and polybutene-1 (PB1) in urine and uACR were observed, although these must be cautiously interpreted due to substantial lactoferrin measurement variability (lactoferrin undetected in 32% of samples) and a small number of PB1 detected samples (N = 5). Findings of this exploratory study suggest oral exposure to MNPs may be associated with renal function and gut integrity.

1. Introduction

Plastic is a durable and versatile material that has allowed humanity to achieve significant advances in medicine, aerospace, construction, *etc.* (Landrigan et al., 2023). Since the start of plastic production in 1950, estimated 8300 million tonnes have been manufactured. Only 9% of plastic waste ever generated has been recycled, and 12% has been incinerated (Geyer et al., 2017). The remaining has been deposited in landfills or released into the environment, leading to plastic debris dispersion across the globe (Geyer et al., 2017). Microplastics (<5 mm) and nanoplastics (<1 μm) are generated by the weathering of plastic

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Abbreviations

micro- and nanoplastics (MNPs)
polyethylene (PE)
polypropylene (PP)
polyamide (PA)
polybutene-1 (PB1)
poly(methyl methacrylate) (PMMA)
polyoxymethylene (POM)
urine albumin-to-creatinine ratio (uACR)

objects, car tires and clothing, in addition to intentional use in personal care and cleaning products (Vethaak and Legler, 2021). Micro- and nanoplastics (MNPs) constitute ubiquitous emerging pollutants detected in air, water, soil and living organisms (Landrigan et al., 2023), raising concerns about potential effects on human health.

Ingestion appears to be a main exposure pathway to MNPs through food, beverages, drinking water (Wright and Kelly, 2017), and oropharyngeal deposition of inhaled particles (Huang et al., 2024). MNPs have been detected in seafood (EFSA CONTAM Panel, 2016), table salt (Zhang et al., 2020), honey (Diaz-Basantes et al., 2020), beer (Diaz-Basantes et al., 2020; Liebezeit and Liebezeit, 2014) and milk (Da Costa Filho et al., 2021) among other food products. MNPs have also been characterised in drinking water (Vega-Herrera et al., 2022, 2023), with plastic water pipes (Mohammadi et al., 2022) and plastic packaging (Oβmann et al., 2018) identified as significant sources. In addition, food and beverage packaging is able to release small plastic particles that migrate to food products and drinks (Zimmermann et al., 2025). This growing evidence of plastic contamination in daily consumption products highlights the need to understand potential health impacts in the gastrointestinal (GI) system.

Few but increasing number of studies have reported associations between MNP exposure and adverse effects. *In vivo* experimental studies with animal models have shown that oral MNP exposure results in MNP accumulation in the gut (Deng et al., 2017; Qiao et al., 2019) as well as translocation across the intestinal barrier to the bloodstream and to distal tissues (Browne et al., 2008; Schwarzfischer et al., 2022). *In vitro* and *in vivo* studies have reported cell damage, oxidative stress, and inflammation after MNP exposure in various organs and cell lines, including kidneys (Deng et al., 2017), intestine (Meng et al., 2022; Qiao et al., 2019), liver (Deng et al., 2017), placenta (Hu et al., 2022), and lungs (Woo et al., 2023). In human studies, MNPs have been detected in multiple organs and biological matrices, including the colon (Ibrahim et al., 2021), kidneys (Massardo et al., 2024), liver (Horvatits et al., 2022), lungs (Jenner et al., 2022), reproductive organs (Zhao et al., 2023), placenta (Ragusa et al., 2021), brain (Nihart et al., 2025), blood (Yang et al., 2024), urine (Massardo et al., 2024; Pironti et al., 2023), and stool (Calikanzaros et al., 2026; Schwabl et al., 2019; Zhang et al., 2021). Observational studies also suggest potential health implications. For example, higher MNP concentrations in stool were associated with inflammatory bowel disease (IBD) severity (Yan et al., 2022). Moreover, significantly higher MNP concentrations were found in tumoral colorectal tissue compared to non-tumoral tissue samples (Cetin et al., 2023).

Faecal calprotectin, lysozyme, β-defensin-2 and lactoferrin are molecules released by phagocytic immune cells that regulate intestinal inflammation, proposed as biomarkers for IBD diagnosis and management (Dai et al., 2018). Zonulin is a protein that regulates intestinal permeability by modulating intercellular tight junctions, and elevated levels in stool correlate with increased intestinal permeability (Malíčková et al., 2017). Urine urea, creatinine and albumin concentrations, as well as the urine albumin-to-creatinine ratio (uACR), are non-invasive markers used to evaluate the presence of excess protein levels in urine, a condition considered an early sign of impaired renal function in clinical settings (KDIGO CKD Work Group, 2024). Exploring the relationship between markers of gut inflammation and permeability and kidney function with MNP exposure will provide valuable insights into the potential role of these contaminants in the development of intestinal and kidney diseases. Therefore, we aimed to evaluate the presence of MNP polymers in urine and stool samples and explore the association with gut integrity and renal function markers among adult volunteers with no diagnosed renal or intestinal diseases.

2. Materials and methods

2.1. Study design and population

We enrolled 50 healthy adults in a cross-sectional study in Barcelona, Spain, from June 2022 to January 2023. The sample size was determined based on budget constraints. Formal sample size estimation was not feasible due to the lack of previous evidence, and this group was defined for a preliminary assessment. Participants were part of GCAT project (Obón-Santacana et al., 2018), as well as additional volunteers recruited through open advertisements distributed at ISGlobal headquarters, public buildings and social media. Interested individuals were requested to answer an online questionnaire to assess eligibility based on residential postal code, age, sex, dietary habits, and health status. Selected participants were Barcelona residents with no self-reported intestinal or kidney disease, approximately 50% men-women. Eligible volunteers were contacted with a formal invitation to participate, including detailed information about the study objective and procedures. Volunteers who agreed to participate signed an informed consent prior participation. Consent form, questionnaires, and procedures were reviewed and approved by the Parc de Salut MAR Clinical Research Ethics Committee (2022/10366/I), complying with relevant ethical guidelines.

2.2. Data and biological samples collection

Questionnaire. Selected participants were requested to complete an online questionnaire to collect socio-demographic and biometric information including country of origin, ethnicity, working situation, level of studies, height, weight and health status. Likewise, a food frequency questionnaire was used to gather data on dietary habits.

Biological samples. Participants self-collected a spot stool sample following instructions and materials provided. For MNPs analysis, a plastic garbage bag was used to completely cover the toilet, and a piece of aluminum foil was placed on top of it to intercept the sample and prevent contact with the bag. The sample (≈50 g) was collected using a disposable wooden spoon, wrapped in another aluminum foil and subsequently placed in a 120 mL container for storage and transportation. Stool samples to analyse gut integrity markers were collected using the Stool Sample Application System (SAS) from Immundiagnostik AG (Bensheim, Germany), following the manufacturer's instructions. This procedure required the collection of 15 mg of stool with a dipstick, which was subsequently diluted in 1.5 mL of IDK Extract® buffer (1:100 dilution factor). Urine samples were also self-collected by the participants in 150 mL polyethylene terephthalate (PET) containers (bisphenol A-free) with screw aluminum cap. Participants provided two urine samples: one from the last void of the day and one from the first morning void. All samples were labeled with the volunteer's code, time, and date of collection. Afterwards, they were promptly placed in hermetic sealed bags in the refrigerator until shipment to the research institute, which took place on the same day of sample collection, upon a telephone call to study personnel to arrange a courier to collect and transport samples. Urine samples were stored at −20 °C and stool samples were stored at −80 °C. After fieldwork completion, urine samples were thawed, and night and day samples from the same participant were pooled and homogenized using a Vortex Mixer for 5 s under a vacuum hood. Samples

were shipped to different laboratories for their respective analyses. Gut integrity biomarkers were analysed at the Immune Response and Biomarker Core Facility at ISGlobal. Renal function biomarkers were analysed at the Reference Laboratory of Catalonia, in Barcelona. MNP analysis in stool and urine samples was performed at the IDAEA-CSIC laboratories, Barcelona.

2.3. MNP analysis

MNPs extraction procedures. Prior to the characterization and quantification of MNP polymers in biological samples, plastic particles were extracted according to the Fenton digestion (Yan et al., 2020). In particular, for stool samples, 100 mL of 30% H_2O_2 and 40 mL of an iron catalyst solution were mixed with 0.5 g of lyophilized sample in 400 mL glass vials. The mixture was digested at 40 °C for 5 h. Afterwards, the sample was centrifuged at 3500 rpm for 15 min and was filtered using 0.7 μm glass fibre filters. 50 mL of 65% HNO_3 were added to the filters for digestion at 50 °C for 30 min, followed by 10 min at 70 °C. After digestion, the mixture was eluted from the filters, and the filters were washed with 50 mL of HPLC-grade water twice. A final wash with 50 mL of ethanol was applied. The filters were dried overnight in an oven at 40 °C and subsequently extracted with 10 mL of toluene in an ultrasonic bath for 15 min. This extraction step was repeated twice, and the resulting toluene extracts were combined and evaporated under a nitrogen stream until approximately 1 mL remained. The concentrated extract was transferred to a liquid chromatography (LC) vial and evaporated to a final volume of 1 mL.

Regarding urine samples, 0.5 mL of urine were extracted with 0.5 mL of toluene in an ultrasonic bath for 10 min. The supernatant was collected and transferred to an LC vial. This extraction process was repeated twice, and the resulting supernatants were combined and evaporated under a nitrogen stream until the sample volume was reduced to 0.5 mL.

MNPs determination procedures. The type and concentration of MNP polymers in biological samples was determined through size exclusion liquid chromatography coupled to high-resolution mass spectrometry (LC-HRMS) employing a previously validated method for complex samples, by using a QExactive-hybrid quadrupole−Orbitrap mass analyser (Llorca et al., 2021; Vega-Herrera et al., 2022, 2024). The separation process was done in an Acquity HPLC system from Waters Corp (Milford, MA, United States), equipped with an advanced polymer column (Acquity APC XT45 1.7 μm particle size) that facilitates the separation by size exclusion, using toluene as an isocratic mobile phase and at a flow rate of 0.5 mL/min for 10 μL of sample injected. The chromatographic system was coupled to an HRMS Q-Exactive hybrid quadrupole-Orbitrap mass spectrometer from Thermo Fisher Scientific (San José, CA, United States), equipped with an atmospheric pressure photo ionization (APPI) source which was operating in negative and positive ionization conditions. Acquisition data was in full scan mode from 500 to 3000 Da and at a resolution of 30,000 full widths at half maximum (FWHM). Data acquisition and processing was performed with Xcalibur v4.2 software (Thermo Fisher Scientific). The suspect screening approach for MNP polymers identification was according to the procedure previously developed and applied by the IDAEA-CSIC Environmental and Water Chemistry for Human Health group (Vega-Herrera et al., 2022, 2023). In summary, the identification and confirmation (level 2 of confidence) of polymers type was by means of Kendrick Mass Defect (KMD) analysis followed by unequivocal confirmation and quantification (level 1 of confidence) with the pure standards that are available in the laboratory. An external calibration curve of polymer standards was performed to quantify polymers based on equivalent concentrations.

2.4. Quality control

Sample preparation was performed in a dedicated microplastics laboratory (2 m × 2 m) specifically designed to minimize airborne and procedural contamination. The room is equipped with a certified laminar-flow cabinet, exclusively metal work surfaces, and no plastic materials. All manipulations were conducted using glassware and metal instruments. Only cotton laboratory coats, cotton-based consumables, and latex gloves are allowed to enter this controlled environment, thereby reducing the risk of introducing exogenous polymeric particles. Multiple types of procedural blanks, including field blanks, laboratory blanks, and solvent blanks, were processed under identical conditions to the samples. These blanks were systematically subtracted from the results. In addition, the possible cross contamination coming from the instrumental analysis was monitored by injecting solvent blanks of toluene (for polymers analysis) and water-methanol 9:1 (for plastic additives analysis) between each sample injection.

2.5. Biomarker analysis

Gut integrity biomarkers. Calprotectin, zonulin, lactoferrin, β-defensin-2, and lysozyme in stool extracts were determined by Enzyme-linked immunosorbent assay (ELISA) using commercially available kits from Immundiagnostik AG (Bensheim, Germany), according to the manufacturer's instructions. All samples and standards were analysed in duplicate. For calprotectin, samples were further diluted 1:25 in sample buffer, resulting in a final dilution factor of 1:2500. For lactoferrin, β-defensin-2, and lysozyme, samples were diluted 1:1 in sample buffer, achieving a final dilution factor of 1:200. Zonulin was tested in undiluted samples. ELISA readings (absorbance at 450 nm) were performed using a BioTek Epoch 2 microplate reader (Agilent Technologies, California, United States), and biomarker concentrations were calculated using CurveExpert 1.4 software for Windows (Hyams Development).

Kidney function biomarkers. Urea, creatinine and albumin in urine were determined using commercial assay kits: Tina-quant Albumin Gen. 2, Creatinine Jaffé Gen. 2, and Urea/BUN from Roche Diagnostics. Concentrations of urea and creatinine were quantified through kinetic tests, while albumin concentrations were determined through immunoturbidimetry. The readings were performed using the Cobas c 701 photometric analyser from Roche Diagnostics. Once the concentrations for these markers had been obtained, the urine albumin-creatinine ratio (uACR) was calculated.

2.6. Statistical analysis

Categorical and continuous covariates were treated as dichotomous variables. Body Mass Index (BMI) was calculated as Kg/m^2 using participants' height and weight and was dichotomised at 25 (overweight threshold). Age was dichotomised at the median (52 years). Exposure variables (specific polymers and any polymer) were dichotomised in non-detected and detected. Outcome variables (gut barrier integrity and kidney function biomarkers) below the limit of quantification (LOQ) were assigned half the minimum quantified value when <30% of samples were below LOQ. This approach was applied to zonulin, lysozyme, β-defensin-2, and albumin, with, respectively, N = 2 (4%), N = 1 (2%), N = 1 (2%), N = 6 (12%) samples below LOQ. For biomarkers with ≥30% samples below the LOQ, imputed values between zero and the lowest quantified value were randomly generated according to a uniform distribution. This method was applied to lactoferrin, as N = 16 (32%) of the measurements were below the LOQ. Outcome values over the 99th percentile and below the 1st percentile were not considered in the statistical analyses in order to reduce the influence of extreme values.

Bivariate analyses were conducted between outcomes and exposures, using the Mann-Whitney *U* test to assess the statistical significance of the difference in the outcomes between exposure categories. Polymers detected in less than 5 samples were excluded from the statistical analyses due to limited statistical power. In total, 25 multiple comparisons

were conducted for gut markers and 20 for renal markers vs. MNP detection in stool and urine samples, respectively. Bivariate analyses were also performed between covariates and exposures, and covariates and outcomes to identify potential confounders, selected based on existing literature. We considered demographic (sex, age, country of origin, ethnicity, working situation, level of education), anthropometric (body mass index) and behavioural (type of diet, alcohol consumption, current smoking status) variables that could influence the outcomes of interest and/or MNP exposure. Chi square and Fisher's exact test (when expected counts <5) were used to assess the significance between these variables. Furthermore, we generated an alternative exposure variable indicating the type of sample in which MNP polymers were detected (none, only in urine, only in stool, both in stool and urine), to compare outcome levels depending on the matrices in which MNP were detected. Kruskal-Willis and pairwise Mann-Whitney U tests were performed to assess differences among and between groups.

Multivariate models for gut integrity markers were run for each MNP variable, comprising 25 multiple comparisons. Generalized linear models (GLM) following a Gamma distribution with a log link function were fit to estimate the ratio of means (ROM) between biomarkers and MNP detection as well as adjusting for covariables. These models were constructed using backward elimination for the covariables that were significantly related to the outcome and/or the exposure. Age and sex were included in all models independently of their statistical significance, as they are factors that have been reported to influence many biological processes, including gut inflammation (Goepp et al., 2025; Xu et al., 2022) and renal function (Clotet-Freixas et al., 2024; Glassock et al., 2017). The same covariate structure was applied within each marker category (gut integrity and renal function markers) in order to ensure comparability across models and avoid overfitting. Models for gut integrity markers were adjusted for sex, age, and working situation, except in the case of lactoferrin, which was only adjusted for sex and age due to non-convergence of the proposed model. Renal function marker models were adjusted for sex, age, BMI and type of diet. Age and BMI were tested as continuous variables in alternative models with no substantial changes observed. We computed false discovery rate (FDR)-corrected p-values to account for multiple comparison. Data management and statistical analyses were done with R studio software (version 24.12.0 + 467).

3. Results

Characteristics of the study participants are presented In Table 1. Participants included were 48% females, had a median age of 52 years and interquartile range (IQR) of 39-58, and median BMI of 24.2 IQR of 22.2-25.7. Most participants were of European ancestry (68%), born in Spain (84%), employed (78%), and had a university degree (72%). The 36% of participants reported following a vegan or vegetarian diet.

In total, 52% of stool samples and 48% of urine samples contained MNP polymers, with 6 different polymers detected. Polyethylene (PE), polypropylene (PP) and polyamide (PA) were detected in both stool and urine samples, while polybutene (PB1) and polymethyl methacrylate (PMMA) were only observed in urine, and polyoxymethylene (POM) only in stool. PA was the most frequent polymer detected in stool samples (34% of the samples). In urine, PA and PP were the most frequently detected polymers (16% of samples). These results are displayed in Table 2. MNP detection rates by categories of socio-demographic and lifestyle characteristics are shown in Supplementary Tables 1 and 2.

Regarding the distribution of gut integrity and renal function biomarker concentrations (Fig. 1), most of the markers showed a right-skewed distribution, except for lysozyme, which followed a slightly right-skewed distribution. Lactoferrin, albumin concentrations and uACR were highly disperse variables, as their respective standard deviations surpassed their means. Concentrations of gut integrity and kidney function markers across categories of socio-demographic and lifestyle variables are described in Supplementary Figs. 1 and 2.

Table 1
Socio-demographic characteristics of the study participants (N = 50).

	N	%	Median [IQR]
Sex			
Female	24	48	
Male	26	52	
Age (years)			
≤52	26	52	52 [39-58]
>52	24	48	
Country of origin			
Spain	42	84	
Other	8	16	
Ethnicity			
European	34	68	
Other	16	32	
Level of studies			
University	36	72	
Other	14	28	
Working status			
Working	39	78	
Other	11	22	
Body mass index (kg/m2)			
<25	33	66	24.2 [22.2-25.7]
≥25	17	34	
Diet			
Vegetarian/Vegan	18	36	
Omnivorous	32	64	
Alcohol consumption			
≥1-2 times/week	33	66	
Never	17	34	
Smoking status			
Smoker	5	10	
Non-smoker	45	90	

Table 2
Absolute (N) and relative frequencies (%) of urine and stool samples with detectable micro-and nanoplastic polymers in the study participants (N = 50).

	LOD (μg/kg)	>LOD N	>LOD %
STOOL			
Polyamide (PA)	0.5	17	34
Polyethylene (PE)	0.03	13	26
Polypropylene (PP)	0.33	11	22
Polyoxymethylene (POM)	0.33	5	10
Any polymer		26	52
URINE	(μg/L)		
Polyamide (PA)	0.2	8	16
Polyethylene (PE)	0.02	7	14
Polypropylene (PP)	0.05	8	16
Polybutene-1 (PB1)	84.7	5	10
Poly(methyl methacrylate) (PMMA)[a]	1.1	3	6
Any polymer		24	48

[a] Excluded from the bivariate analysis (N < 5).

Figs. 2 and 3 present the bivariate analyses between gut integrity and renal function markers by MNP polymer detection in stool and urine samples, respectively. All FDR-corrected p-values became non-significant, and given the exploratory nature of the present study, unadjusted p-values are presented. Higher calprotectin concentrations (median [IQR]) were observed in samples free of PP (37 μg/g [24-52]), compared to those with detected PP (19 μg/g [16-36]). Samples with detected POM showed higher β-defensin-2 levels (83 ng/g [79-89]) compared to those with non-detected POM (25 ng/g [8-47]). Participants with any detected MNP polymer in urine exhibited a significantly higher uACR, with a median [IQR] of 2.25 mg/g [1.25–4.55], compared to 0.65 mg/g [0.38–3.38] in those without MNP polymers. Similar results were observed for albumin, as individuals with any MNP polymer in urine showed higher albumin concentrations, median [IQR] 1.75 mg/L [0.65 - 4.97] compared to non-exposed individuals (0.5 mg/L [0.3-1.6]). Accordingly, statistically significantly higher uACR and albumin concentrations were found in participants with MNP polymer detected

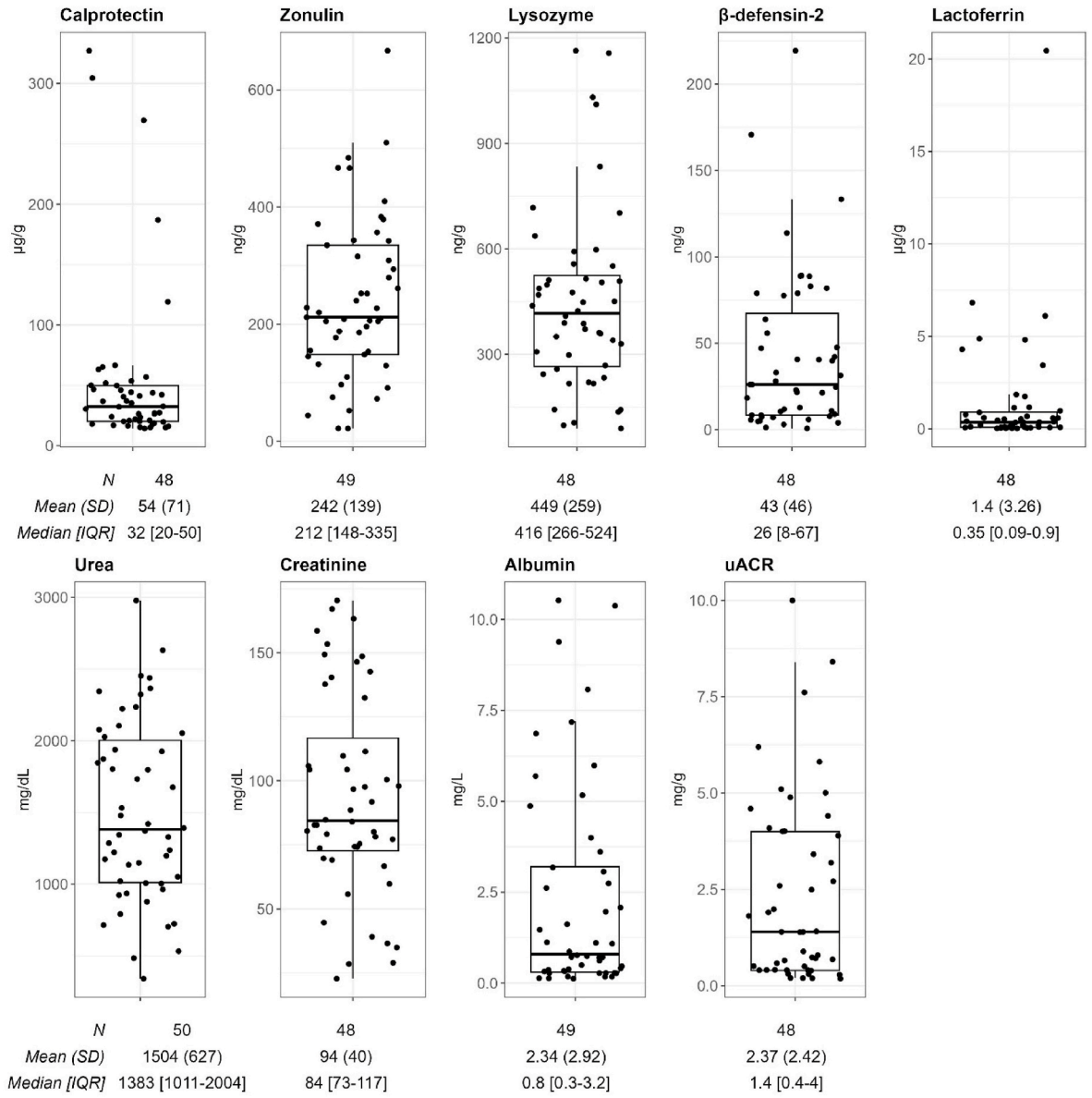

Fig. 1. Distribution of gut integrity and renal function biomarkers concentrations among samples. The dots represent each participant's individual biomarker concentration. The central horizontal line is the median, whereas the other two horizontal lines represent the interquartile range. This figure displays the biomarker concentration distribution after outlier removal.

both in stool and urine compared to those with non-detectable MNPs (Supplementary Fig. 3). Other differences in kidney markers between detected vs. non-detected MNP polymers were not statistically significant.

Among 25 multiple comparisons between stool MNP detection and gut markers in multivariate models, a negative association was observed for calprotectin and lactoferrin concentration in PP exposed groups in the multivariate analysis (Fig. 4). Specifically, PP detection was associated with a 47% decrease in the average calprotectin concentration (ROM [CI95%] = 0.53 [0.31-0.94]) and 79% reduction in the mean lactoferrin levels (ROM = 0.21 [0.09-0.63]). Increased β-defensin-2 concentrations in individuals exposed to POM were observed in the unadjusted models (Supplementary Fig. 4), although this trend remained in the adjusted models, it became no longer statistically significant. In the multivariate analysis for renal function markers a total of 20 multiple comparisons were performed (Fig. 5). Urine samples with any MNP polymer detected presented a mean increase of 116% in the uACR (ROM [95%CI] = 2.16 [1.14 – 4.10]) and 115% in albumin concentrations (ROM [95%CI] = 2.15 [1.08 – 4.28]). In contrast, we observed a 70% decrease in the mean uACR in the PB1 exposed group (ROM [95%CI] = 0.3 [0.13 – 0.83]). This trend was also observed between albumin and PB1, although it was not statistically significant. A 123% increase in the average concentrations of albumin in PP exposed individuals was also reported (ROM [95%CI] = 2.23 [1.01 – 5.68]). ROMs for unadjusted renal function biomarker models are displayed on Supplementary Fig. 5.

4. Discussion

Our exploratory study assessed the relationship between MNPs exposure and markers of gut integrity and renal function in 50 healthy adults from Barcelona (Spain). MNP polymers were detected in 52% of stool and 48% of urine samples, comprising 6 distinct polymers across both types of samples. The bivariate analyses indicated positive associations between POM and β-defensin-2 in stool, and any MNP polymer and uACR and albumin in urine; and negative associations between PP

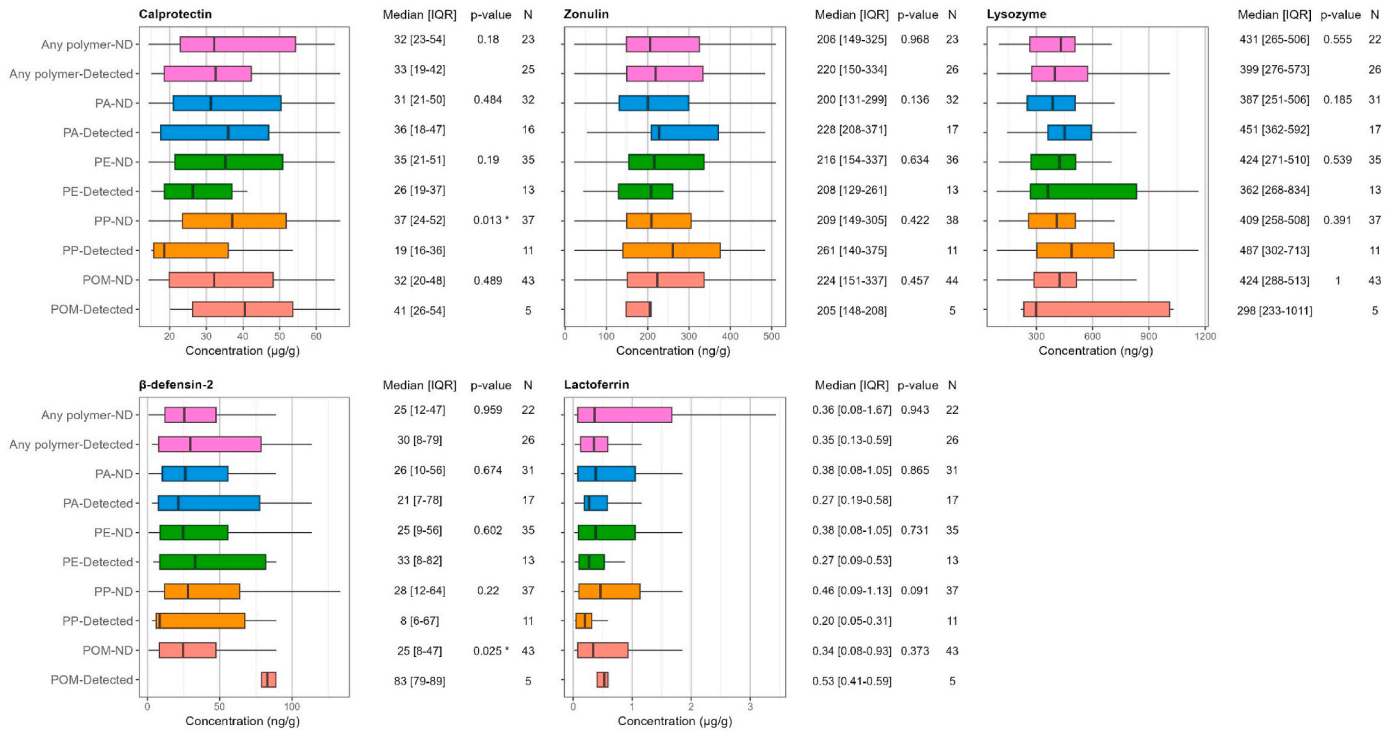

Fig. 2. Comparison of gut integrity markers in stool samples with detected vs non-detected (ND) MNP polymers. Comparison between groups was performed using Mann-Whitney *U* test. The central lines are the medians, whereas the box represents the interquartile range. The displayed p-values are not corrected for multiple comparisons.

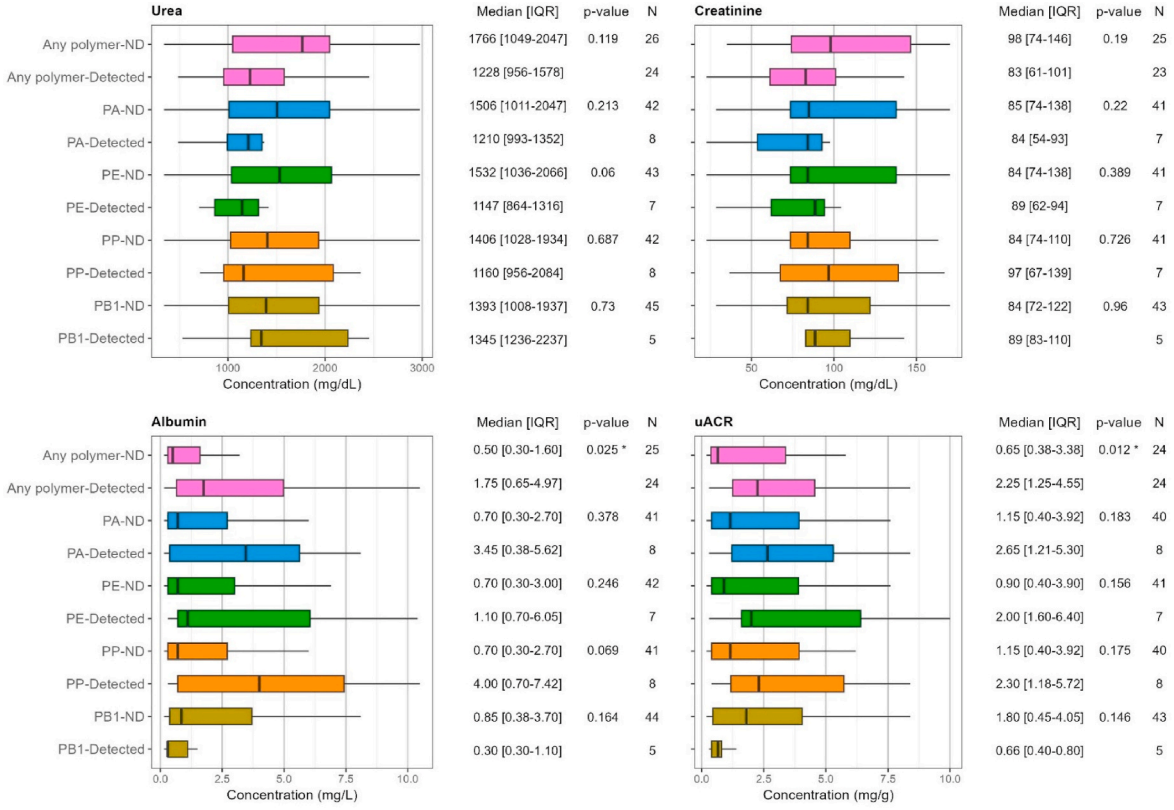

Fig. 3. Comparison of kidney function biomarkers in urine samples with detected vs non-detected (ND) MNP polymers. Comparison between groups was performed using Mann-Whitney *U* test. The central lines are the medians, whereas the box represents the interquartile range. The displayed p-values are not corrected for multiple comparisons.

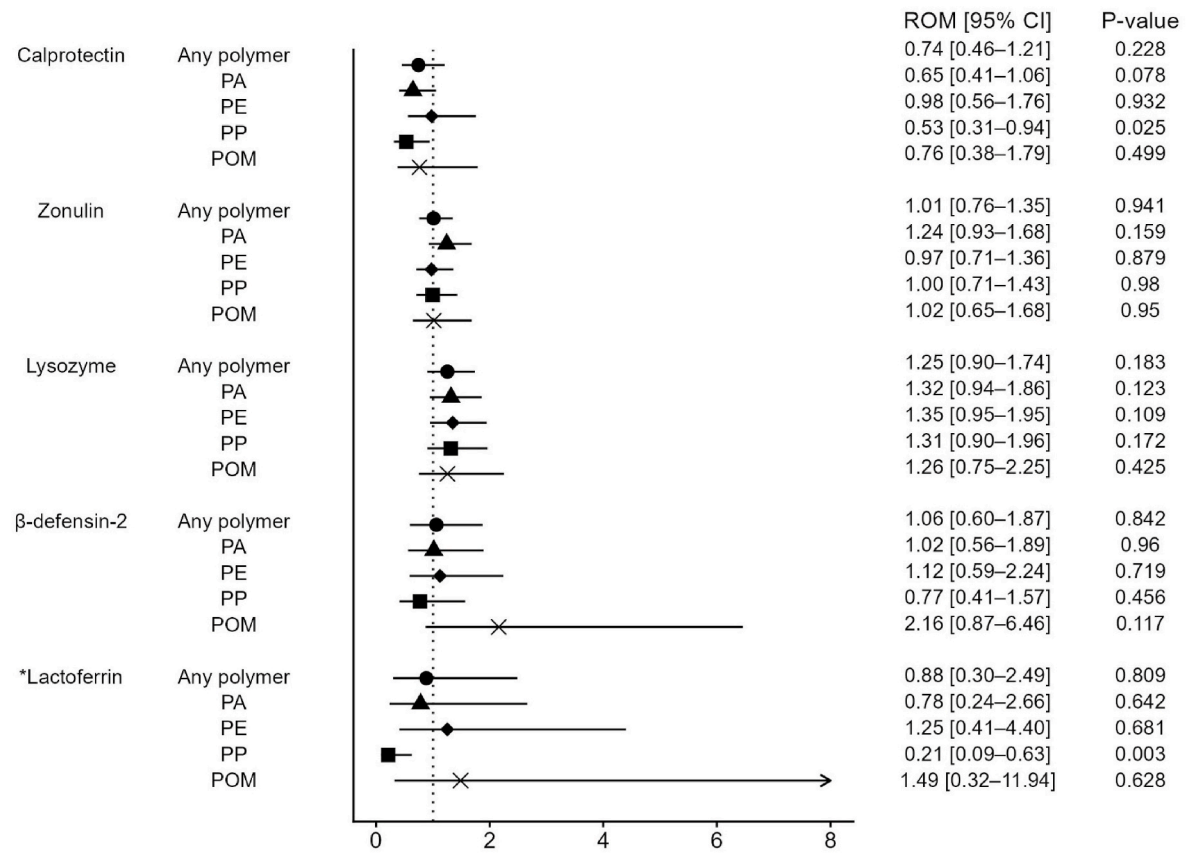

Fig. 4. Ratio of means (ROM) and 95% confidence intervals (CI) for gut integrity biomarker concentrations associated with MNP polymer detection vs. non-detection adjusted for sex, age, and working status. The figure compares the average mean for each biomarker between samples with detectable concentrations of MNP and samples without detectable levels of MNPs in terms of ROM and its associated 95% CI. The displayed p-values are not corrected for multiple comparisons. *Only adjusted for sex and age due to statistical constraints.

detection and calprotectin in stool. These trends remained in the multivariate analysis, although without statistical significance for POM and β-defensin-2. In addition, we observed statistically significant negative associations between lactoferrin-PP and uACR-PB1 as well as a positive association between albumin and PP detection. Findings suggest that MNP exposure may be associated with renal function and gut integrity changes.

MNP polymers were detected in approximately half of the urine and stool samples, which contrasts with the detection frequencies reported in previous studies, where MNPs were found in almost every sample (O'Callaghan et al., 2025; Schwabl et al., 2019; Yan et al., 2022; Zhang et al., 2021). Several of these previous studies were conducted in non-European countries; therefore, differences in population exposure and methodological approaches, including quality-control procedures, may contribute to the lower polymer detection observed in our study. However, comparability across studies is limited due to the differences in methodologies, quantification limits and accuracy of the analytical methods employed to assess MNPs, which originates from the lack of standardized analytical techniques for MNP quantification. PA was the most frequently detected polymer in both stool and urine samples, followed by PE, PP and POM in stool and PP, PE, PB1, PMMA in urine. Except for PB1, the rest of the detected polymers were previously reported in these biological matrices (Krause et al., 2024; O'Callaghan et al., 2025).

In the bivariate analysis and the unadjusted GLM, the presence of POM in stool was associated with higher concentrations of β-defensin-2, an antimicrobial peptide produced by gut epithelial cells in response to inflammatory cytokines (Gacesa et al., 2021), suggesting increased gut inflammation. Nevertheless, this association was no longer statistically significant when adjusting for covariates. Although the small number of POM-detected samples hampered our ability to properly assess this relationship, this result is consistent with previous studies reporting associations between MNP exposure (including POM polymers) and increased gut inflammation in IBD patients (Yan et al., 2022). Similarly, an experimental study in mice with chronic colitis showed that polystyrene exposure aggravated inflammation and intestinal injuries (Ma et al., 2023). It is important to acknowledge that animal models are usually exposed to high doses of single polymer types, whereas realistic MNP exposure scenarios usually entail low doses of MNPs from several sources and exposure pathways. Thus, contrarily to rodent models, real MNP exposure levels in humans might not be high enough to trigger a detectable response in the GI tract of healthy individuals.

Significantly lower concentrations of calprotectin were observed in stool samples with detectable PP levels in both the bivariate analysis and multivariate model. We also observed significantly lower levels of lactoferrin in PP-detected samples in the multivariate analysis. Both molecules are well-established non-invasive biomarkers for IBD diagnosis and monitorisation (Lopez et al., 2017). Considering previous evidence showing a positive association between MNP detection in stool and IBD severity (Yan et al., 2022), a potential explanation to this unexpected finding could be reverse causation. For instance, sub-clinical gut inflammation levels may lead to increased intestinal permeability, which might lead to inflammatory molecules release in the gut lumen and MNP translocation (Macura et al., 2024). In addition, *in vitro* studies have shown that MNPs may compromise the intestinal barrier's integrity through the induction of oxidative stress and inflammatory responses, leading to mucosal tight junction disruption (Xu et al., 2021; Zeng et al., 2024) and allowing plastic polymers to enter the bloodstream.

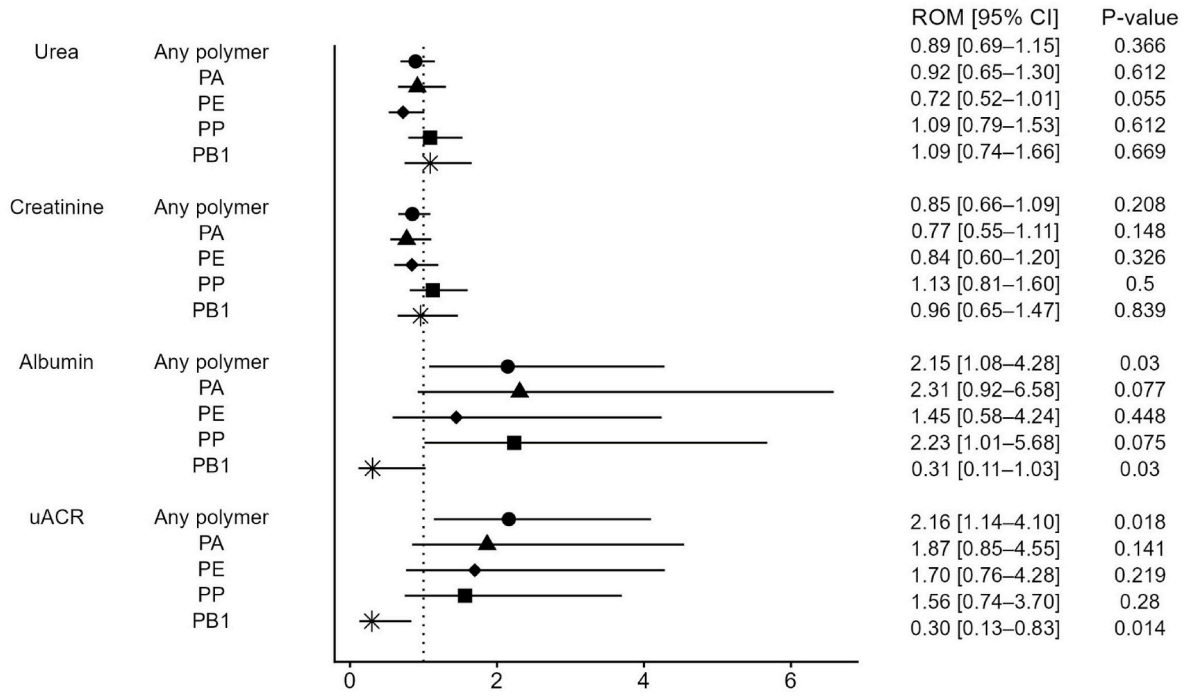

Fig. 5. Ratio of means (ROM) and 95% confidence intervals (CI) of renal function biomarker concentrations associated with MNP polymer detection vs. non-detection, adjusted for sex, age, body mass index, and diet. The figure compares the average mean concentration for each biomarker between samples with detectable levels of MNP and samples without detectable levels of MNPs in terms of ROM and its associated 95% CI. The displayed p-values are not corrected for multiple comparisons.

Therefore, increased concentrations of biomarkers of gut inflammation in stool samples might potentially be linked to systemic MNP uptake. However, these remain speculative explanations and findings require replication. Moreover, the high percentage of samples with undetected lactoferrin hinders the ability to draw reliable conclusions for this marker.

A positive association was found between any MNP polymer in urine and uACR, which is an established marker of renal function and early indicator of chronic kidney disease (CKD) (Chagnac and Friedman, 2024). This finding may suggest a mild increase in albumin excretion relative to creatinine associated with MNP exposure, implying a subtle impact of MNPs on renal filtration processes, as all study participants had uACR values within the clinically normal range, well below the kidney disfunction threshold (30 mg/g) defined in clinical guidelines (KDIGO CKD Work Group, 2024). Although there is no previous evidence assessing renal health effects of MNPs in humans, animal studies have reported increased renal inflammation (Wang et al., 2021), proteinuria and CKD related symptoms (Liang et al., 2024) in mice exposed to MNPs, supporting our results. On the other hand, it is important to bear in mind that the analytical method employed detects MNPs sized between 0.7 and 20 μm, which is larger than the presumed glomerular filtration threshold size (<10 nm) (Du et al., 2017). However, animal studies have reported renal clearance of nanoparticles measuring up to 0.5 μm with high aspect ratios (ranging from 100:1 to 500:1) (Rotchell et al., 2024; Ruggiero et al., 2010). Therefore, this evidence underpins the biological plausibility of our results, suggesting that MNP exposure might be associated with kidney function. Furthermore, we found that higher uACR was specifically observed among participants with MNP in both their stool and urine samples compared to participants with no MNPs. A speculative explanation could be that MNPs detection in multiple biological matrices might suggest systemic exposure, which in turn may be associated with stronger changes in kidney function. Finally, the observed negative association obtained between uACR and PB1 needs to be cautiously interpreted, given the small sample size of the PB1 exposed group, chance may explain this specific finding.

We observed a significant positive association between albumin and any MNP polymer in both the bivariate analysis and the adjusted GLM, and albumin-PP in the adjusted GLM. Despite the widespread use of urine albumin as a renal function marker, direct comparisons between individual measurements might not be reliable due to the significant influence of urine dilution (KDIGO CKD Work Group, 2024). In other words, two individuals with a similar renal function could have substantially different urinary albumin concentrations due to urine dilution, thus, the comparison of these would lead to drawing false inferences. Consequently, the observed associations involving albumin are not likely to represent real differences of renal function between the exposed and non-exposed individuals. Nevertheless, albumin can be used to calculate the uACR, a renal function marker that corrects for urine concentration (Chagnac and Friedman, 2024), allowing us to reliably compare renal function between exposed and non-exposed groups.

To our knowledge, this is the first study to assess renal function and gut barrier integrity associated with MNP exposure in a healthy population (i.e., individuals without known kidney or gastrointestinal disease). Previous human studies reporting the presence of MNPs in stool and tissues explored the relationship with intestinal disease (Yan et al., 2022) and colorectal cancer (Ibrahim et al., 2021), but none evaluated renal function or gut integrity biomarkers. However, the limited sample size reduces the statistical power of the study, as reflected in the wide confidence intervals of the multivariate models' estimates (ROM). This hinders our ability to detect stable significant associations. The cross-sectional design prevents us from establishing temporality of events and reverse causation cannot be ruled out. Single-time measurements cannot account for within-person variability in biomarker levels due to circadian rhythms, diet, or other short-term factors, which may introduce measurement error. Similarly, time-point sampling to measure MNPs does not necessarily reflect long-term exposure, which constitutes a limitation. When combined with the small sample size, this variability may increase the risk of obtaining spurious associations in the bivariate and multivariate analyses. We acknowledge that chance could partly explain the detected associations, as after applying multiple

comparison corrections no statistically significant associations remained. We computed FDR-corrected p-values but did not include them in the results because of the hypothesis generating nature of the work. Residual confounding by factors not measured in the present study (e.g. occupational exposures, dietary intake) may also influence the associations obtained. Moreover, a potential selection bias may have been induced due to the characteristics of the study population (volunteers who were predominantly middle-aged and highly educated) rendering the sample unrepresentative of the general population and thereby limiting the external validity of the findings (Blay et al., 2025). Nonetheless, the sample remains valid to provide valuable insights into the hypothesis under investigation.

5. Conclusions

Findings of this exploratory study suggest oral exposure to MNPs may be associated with renal function and gut integrity. However, the direction of the associations cannot be established with this cross-sectional design and findings remain hypotheses to be tested in future studies. Confirmatory longitudinal studies with larger and repeated biomarker measurements are warranted. If confirmed, these findings could have important implications for understanding the health effects of environmental microplastic pollution, particularly regarding intestinal and renal human health.

CRediT authorship contribution statement

Maria Miguela-Benavides: Writing – original draft, Visualization, Formal analysis. **Emma Calikanzaros:** Writing – review & editing, Supervision, Conceptualization. **Carolina Donat-Vargas:** Writing – review & editing, Conceptualization. **Ruth Aguilar:** Writing – review & editing, Investigation, Resources. **Felipe Raimondi:** Investigation, Resources. **Susana Iraola:** Resources, Investigation. **Carlota Dobaño:** Writing – review & editing, Resources. **Rafael de Cid:** Writing – review & editing, Resources, Investigation. **Marta Llorca:** Investigation, Resources. **Marinella Farré:** Writing – review & editing, Investigation, Resources. **Cristina M. Villanueva:** Writing – review & editing, Supervision, Project administration, Funding acquisition, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2026.124197.

Data availability

Data will be made available on request.

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