

Article

Concurrent Assessment of Micro- and Nanoplastics and Bisphenol A in Non-Dialysis Chronic Kidney Patients and Hemodialysis Patients: Results from a Cross-Sectional Human Biomonitoring Study Focusing on Sex-Related Patterns

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Abstract

Patients with chronic kidney disease (CKD), particularly those receiving hemodialysis (HD), may be more vulnerable to exposure to micro- and nanoplastics (MNPs) and bisphenol A (BPA) because of impaired renal clearance and frequent contact to plastic-based medical devices. This study aimed to assess and compare the levels of blood MNPs and urinary BPA in HD patients, non-dialysis CKD patients, and healthy subjects with a focus on sex-related differences and the potential role of hemodialysis as an additional source of exposure. A cross-sectional human biomonitoring study was conducted. Blood and urine samples were collected to quantify BPA using gas chromatography–mass spectrometry (GC–MS) and MNPs using scanning electron microscopy coupled with an Energy-Dispersive X-ray Detector (SEM-EDX). Descriptive statistics included medians, interquartile ranges (IQRs), 95% confidence intervals, frequencies, and percentages. Blood MNP levels were higher in HD [9889 (4525–14,939) p/mL] and non-dialysis CKD patients [9234 (3354–15,429) p/mL] than in healthy subjects [5755 (2488–9042) p/mL]. Urinary MNP levels were also higher in non-dialysis CKD patients [1231 (0–8281) p/mL] than in healthy subjects [411 (0–1636) p/mL]. Urinary MNPs were smaller in non-dialysis CKD patients than in healthy subjects. In contrast, urinary BPA levels were lower in non-dialysis CKD patients [0.16 (0.12–0.16)] than in healthy subjects [0.98 (0.60–2.36)]. Sex-specific patterns were observed. Females had higher blood MNP levels than males among HD patients (median 9907 vs. 9063 p/mL).



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and healthy subjects (6627 vs. 4131 p/mL), whereas the opposite pattern was observed in non-dialysis CKD patients (10,083 vs. 7340 p/mL). Overall, these findings suggest an increased systemic burden of MNPs and altered BPA elimination in non-dialysis CKD and HD patients. The observed sex-specific patterns suggest that biological sex may contribute to differences in internal exposure within this vulnerable population.

Keywords: micro- and nanoplastics; bisphenol A; chronic kidney disease; dialysis; environmental exposure

1. Introduction

Plastics are highly adaptable materials that have transformed numerous sectors, from the food industry to healthcare. Worldwide plastic production has increased dramatically, reaching 413.8 million metric tons in 2023, with a consequent rise in environmental contamination [1–3]. A substantial proportion of plastics is discarded after a single use and is frequently released into the environment. Most plastics are highly resistant to degradation and progressively fragment into micro- and nanoplastics (MNPs). MNPs are defined as plastic particles with a diameter of less than 5 mm [4] and are commonly classified as primary and secondary. Primary MNPs are intentionally manufactured for commercial applications (i.e., facial cleansers, toothpaste, exfoliating creams, and drug delivery vectors) [5,6]. In contrast, secondary MNPs originate from the breakdown of larger plastic materials and macroplastics through processes influenced by temperature, ultraviolet radiation, and biological activity [7,8].

MNPs accumulate in both aquatic and terrestrial environments. In marine ecosystems, large plastic debris, such as that found in the Great Pacific Garbage Patch [9], may act as a long-term source of MNPs through progressive fragmentation [10,11]. Similarly, in terrestrial environments, plastic residues can form persistent “scar” layers that may serve as stratigraphic indicators [12]. Numerous studies have also demonstrated the presence of MNPs in everyday consumables, including bottled water [13], tap water [14], fruit, vegetables [15], meat, and fish [16,17]. Human exposure to MNPs occurs predominantly through ingestion, inhalation and dermal contact [18]. Once internalized, MNPs may enter the bloodstream and accumulate in different tissues and organs, inducing cellular alterations through oxidative stress, metabolic disruption, and inflammation [19,20], as shown in both “in vitro” [21] and “in vivo” models [20,22]. In addition, MNPs may exert indirect toxic effects, by acting as carriers for microorganisms, viruses, and environmental pollutants [23]. Among the contaminants associated with MNPs, bisphenol A (BPA) represents a major concern due to its ability to adsorb onto MNPs and bioaccumulate in the human body [19]. BPA is a synthetic, heat-resistant xenobiotic with endocrine-disrupting properties, and is widely used in the production of polycarbonate plastics, water bottles, toys, cigarette filters, and medical devices [24]. It is one of the most extensively produced chemicals worldwide and is ubiquitous in the environment, contaminating air, water, soil, food, and beverages. [25]. Because of its documented adverse effects on human health, regulatory authorities have progressively introduced stricter measures, culminating in the recent European Union ban on BPA in food- and beverages-contact materials, established by Commission Regulation (EU) 2024/3190 [26–28]. This regulatory shift underscores the public health relevance of investigating internal BPA exposure, particularly in medically vulnerable populations such as patients with chronic kidney disease (CKD) and those undergoing hemodialysis (HD) who may experience impaired BPA elimination and additional exposure from medical devices. Despite growing evidence, previous studies have

rarely assessed BPA and MNPs co-exposure simultaneously, and none have systematically examined sex-specific differences in internal exposure [27,28]. This gap highlights the need for integrated sex-stratified analyses to clarify how dual contaminant burdens may differ across renal disease populations.

Human exposure to BPA occurs through pathways similar to those described for MNPs, including ingestion, inhalation, dermal absorption, and vertical transmission, with dietary intake considered the predominant source of exposure [29]. Because of its lipophilic nature, BPA can accumulate in multiple tissues and has been associated with an increased risk of cancer, hypertension, and cardiovascular disease [30], as well as with disruption of reproductive and neuroendocrine functions [31]. BPA exposure has also been proposed as a potential contributor to CKD progression, as the compound is primarily eliminated through urinary excretion [32]. Although the full impact of BPA on renal function remains under investigation, BPA-induced inflammation has been suggested as a possible mechanism contributing to CKD progression and impaired BPA clearance [33]. In patients undergoing HD, BPA exposure may be further exacerbated, as its removal during dialysis is limited and may depend on both the composition of medical devices and the dialysis modality used [34]. Given that HD accounts for approximately 89% of all dialysis treatments worldwide [35], it may also represent an additional source of MNPs exposure due to the extensive use of plastic-based medical devices [36,37]; however, this hypothesis requires confirmation in longitudinal studies specifically designed to evaluate exposure before and after dialysis initiation.

CKD represents a major global public health issue and is currently the ninth leading cause of death worldwide [38], affecting more than 10% of the global population, corresponding to over 800 million individuals, particularly among vulnerable populations [39]. CKD is defined by the presence of kidney damage or a glomerular filtration rate $< 60 \text{ mL/min/1.73 m}^2$ for 3 months [40], and may progress to kidney failure, cardiovascular diseases, and premature mortality [41]. Its clinical manifestations are heterogeneous, resulting in complex management strategies ranging from conservative treatment [42] to renal replacement therapies, such as HD [43]. Recent experimental studies using human embryonic kidney cells [44] and mice [45] have suggested that MNPs may induce renal alterations. CKD progression is influenced by multiple factors, with immune dysregulation and chronic inflammation playing a central role as both pathogenic drivers and therapeutic targets [46]. Given the strong link between inflammation and lifestyle and environmental factors [47], reducing exposure to environmental contaminants such as MNPs, alongside appropriate lifestyle modifications, may contribute to mitigating disease progression. The issue of plastic exposure in nephropathic patients has long been recognized by the nephrology community [48,49]. Recent studies have investigated the presence and excretion pathway of MNPs, confirming their detection in human urine, blood, kidneys, and other tissues, and underscoring their potential health relevance [50].

Given the growing evidence linking MNPs and BPA exposure to renal health, particularly in patients with CKD, this study aimed to quantify blood MNPs and urinary BPA levels in HD patients, non-dialysis CKD patients, and healthy subjects, with particular attention to sex-related differences in exposure levels. We also explored whether HD treatment may represent an additional source of exposure, thereby addressing an important knowledge gap regarding the burden of environmental contaminants in this vulnerable population.

2. Materials and Methods

2.1. Study Design and Participants

A cross-sectional human biomonitoring study with groups defined by disease status was conducted, including three predefined groups of participants: patients undergoing HD (group 1), patients with CKD not receiving dialysis (group 2) and healthy subjects (group 3). Participants were enrolled, between January 2022 and November 2024, at the Policlinic “G. Rodolico-San Marco” Hospital, Department of Internal Medicine, section of Nephrology, University of Catania, and “Azzurra” Nephrology and Dialysis Techniques Clinic (Catania, Italy). Inclusion criteria for HD patients included age > 18 years, stage 5 CKD, and at least six months of ongoing HD treatment. Inclusion criteria for non-dialysis CKD patients comprised a diagnosis of stage 3–4 CKD with no indication for renal replacement therapy. Healthy subjects were selected among hospital patients with no history of CKD. For each participant, blood and urine samples were collected to quantify MNPs and BPA; for HD patients, only blood samples were collected, as they did not produce urine due to their clinical condition.

Whole blood samples were collected from non-fasting participants into two pierceable K₂EDTA tubes at the time of the clinical visit. In non-dialysis CKD patients, blood samples were obtained from a peripheral vein, whereas in HD patients, blood was drawn from the vascular access (central venous catheter or arteriovenous fistula) before connection to the hemodialysis circuit. Urine samples from non-dialysis CKD patients were collected concurrently with blood sampling in pre-cleaned, sterilized glass containers to minimize the risk of external contamination. All samples were immediately stored at −20 °C without agitation until analysis.

2.2. Data Collection

A structured interview was administered to each participant by trained physicians to laboratory testing. The interview collected information on socio-demographic and clinical characteristics, habits and lifestyles, potential sources of MNPs and BPA exposure, and clinical data.

2.3. Laboratory Tests

2.3.1. Determination of Bisphenol A (BPA)

The analytical method has already been described elsewhere [51,52]. Briefly, urine samples were thawed at room temperature and homogenized by vortex mixing. A 5 mL aliquot of each sample was transferred into a conical-bottom glass tube (VWR International, Radnor, PA, USA) and spiked with 100 µL of the internal standard (BPA-d16+; LGC Standards, Teddington, UK). Subsequently, 40 µL of β-glucuronidase solution (Sigma-Aldrich, St. Louis, MO, USA) (20,000 U/mL in 1 M ammonium acetate buffer pH 5.0) was added. Samples were incubated overnight at 37 °C in a heat bath (Unitronic Orbital, J.P. Selecta, Barcelona, Spain) to ensure complete enzymatic hydrolysis of conjugated BPA. After incubation, 1325 µL of acetonitrile (MeCN; Sigma-Aldrich, St. Louis, MO, USA), 85 µL of tetrachloroethylene (T4CE; Sigma-Aldrich, St. Louis, MO, USA), and 125 µL of ammonium acetate (Sigma-Aldrich, St. Louis, MO, USA) were added. The mixture was vortexed (Vortex-Genie 2; Scientific Industries, Bohemia, NY, USA) and centrifuged at 2500 rpm for 20 min at 4 °C using a refrigerated centrifuge (Bioprocen 22R, Ortoalresa, Madrid, Spain). Finally, 70 µL of the lower organic phase was transferred into a 100 µL insert and stored at −80 °C until further analysis.

2.3.2. GC–MS Analysis

Gas chromatography–mass spectrometry (GC–MS) analyses were performed using an HP6890 gas chromatograph (Agilent Technologies, Santa Clara, CA, USA) coupled to an Agilent 5973 single quadrupole mass selective detector (Agilent Technologies, Santa Clara, CA, USA) operated in electron ionization (EI) mode. Separation was achieved using an HP-5MS capillary column (30 m × 0.25 mm × 0.25 µm) (Agilent J&W, Folsom, CA, USA) with helium employed as the carrier gas at a constant flow rate of 1.1 mL/min. Samples were injected in splitless mode at an injector temperature of 280 °C, with a purge-off time of 60 s. The oven temperature program started at 100 °C (held for 1 min), increased to 280 °C at a rate of 30 °C/min, and was held for 8 min, followed by a ramp to 310 °C at 40 °C/min, which was maintained for 1 min, resulting in a total run time of 16.75 min. The transfer line temperature was set at 280 °C, while the ion source and quadrupole temperatures were maintained at 230 °C and 150 °C, respectively. The mass spectrometer was operated in selected ion monitoring (SIM) mode, acquiring at least three characteristic ions per analyte. Data acquisition and processing were carried out using Agilent ChemStation software (version D.0200SP1, Agilent Technologies, Santa Clara, CA, USA).

2.3.3. Quality Assurance, Quality Control Measures, and Method Performance Parameters

All glassware and materials were pre-cleaned by sonication twice for 30 min in methanol (Sigma-Aldrich, St. Louis, MO, USA), followed by 30 min of sonication in Milli-Q water (Elix Reference Water Purification Systems, Merck Millipore, Burlington, MA, USA) and a final rinse with ethanol (Sigma-Aldrich, St. Louis, MO, USA). Sonication was performed using an ultrasonic bath (Ultrasons HD, J.P. Selecta, Barcelona, Spain). Procedural blanks prepared with Milli-Q water and representing 10% of the total number of samples were included throughout the extraction process. Samples were randomized during both processing and instrumental analysis, and quality control samples (pooled urine or calibration standards) were analyzed every ten injections to monitor instrumental performance and signal stability. Matrix-matched calibration curves were prepared with spiking standards containing bisphenol analogs (LGC Standards, Teddington, UK) at levels ranging from 0.2 to 50 µg/L. Internal standard (BPA-d16) was added to all standards and samples at a final concentration of 27 µg/L. Linear regression analysis yielded coefficients of determination (R^2) > 0.995 for all analytes. Method limits of detection (LODs) and quantification (LOQs) were determined as the levels corresponding to signal-to-noise ratios (S/N) of 3 and 10, respectively. Matrix effect was evaluated by comparing the slopes of the calibration curves prepared in matrix and solvent, and coefficient of variation (CV) was also calculated. Recovery was assessed by comparing the analytical response of analytes in samples spiked before and after the extraction step at two concentration levels (2.5 µg/L y 10 µg/L) to evaluate extraction efficiency. Further details are provided in Supplementary Materials (Table S1).

2.3.4. Creatinine Determination

Urinary bisphenol levels were normalized to creatinine levels to account for urine dilution. Creatinine was quantified using the Jaffé method with a commercial creatinine assay kit (Química Clínica Aplicada, Tarragona, Spain) and measured in a COBAS Mira biochemical analyzer (Roche Diagnostics, Rotkreuz, Switzerland).

2.3.5. MNPs in Blood and Urine

Sample Preparation

The blood MNPs extraction method employed in this study is protected by both national and international patents (PCT/IB2019/051838 of 7 March 2019) and was applied

according to the methodology described by Montano et al. [53]. The method has been previously validated, showing a mean recovery of 93% and a limit of detection (LOD) of 0.1 μm [13,54]. Several precautions were implemented to minimize environmental contamination during sample handling, including the use of nitrile gloves (Kimberly-Clark, Irving, TX, USA), cleaned laminar flow hoods, and filtration of reagent through glass fiber filter (Whatman GF/A; Cytiva, Marlborough, MA, USA) to reduce airborne dust and synthetic textile debris. Throughout all experimental phases (sampling, storage, sample pre-treatment, sample extraction, and analysis) only glass and aluminum equipment and containers were employed. All materials employed for samples processing were thoroughly washed with UPLC-MS grade water (Merk, Darmstadt, Germany). A 100 μL aliquot of whole blood from each sample was transferred into glass tubes (VWR International, Radnor, PA, USA), which were subsequently sealed. Samples underwent chemical treatment (Merk, Darmstadt, Germany) and were digested using a graphite digestion block system (digiPREP LS, QuantAnalitica, Trezzano sul Naviglio, Italy). Reagent blanks were processed in parallel for each batch of samples throughout the analytical procedure.

Following digestion, and liquid–liquid recovery of MNPs, the extracts were centrifuged (Eppendorf 5427R, Hamburg, Germany). The resulting extracts were then deposited on to aluminum stubs for scanning electron microscopy (SEM) analysis (Oxford Instruments, High Wycombe, UK), using 25 mm diameter SEM Specimen Stubs (Agar Scientific, Stansted, UK), previously cleaned with 100% high-purity dichloromethane (Carlo Erba Reagents, Milan, Italy).

SEM-EDX Analysis

The stubs were metallized with pure gold using a Cressington Sputter Coater 108 Auto (Cressington Scientific Instruments Ltd., Watford, UK). After that, the stubs were analyzed using a ZEISS LEO 1530 scanning electron microscope (SEM) coupled with an energy-dispersive X-ray detector (SEM–EDX) Oxford Instruments, High Wycombe, UK, operated with AZTEC software (version 5.1).

Blood and urine samples were spiked with certified standard MNPs (3 μm red polystyrene particles; Merck–Sigma Aldrich, Darmstadt, Germany), yielding mean recoveries ranging from a minimum of 83% to a maximum of 103% for blood and urine samples, respectively. This methodology was selected because the elimination of the sample filtration step prevents the substantial loss of micro- and nanoplastics with sizes smaller than the pore diameter of conventional filters. Such filtration steps are commonly employed in current MNP analytical protocols and may lead to underestimation of micro- and nanoplastic levels in both blood and urine samples.

EDX analysis allowed discrimination between plastic and non-plastic micro- and nanoparticles, in accordance with previously published studies [15,55]. Quantification of MNPs was performed over a total analyzed area of 1 mm^2 per stub (Al-234 copper stub coated with pure gold), corresponding to the analysis of 228 fields at 1500 $\times$ magnification. Results were expressed as the number of particles per milliliter (p/mL) of sample.

Three reagent blanks were processed with each batch of samples, and no measurable MNPs were detected, indicating the absence of detectable contamination arising from sample handling and analytical procedures.

2.3.6. MNPs Chemical Composition Typization

The chemical composition of the polymer particles was assessed using pyrolysis–gas chromatography/mass spectrometry (Py GC/MS). Each analysis was carried out on approximately 0.1–0.2 mg of the sample.

The low level plastics calibration standard (MPCS L) dispersed in a SiO₂ powder matrix was supplied by Frontier Laboratories Ltd. (Koriyama, Japan). This standard mixture contained the following polymers: polystyrene (PS), polycarbonate (PC), polymethyl methacrylate (PMMA), polyvinyl chloride (PVC), polypropylene (PP), polyethylene (PE), nylon 6.6 (N-66) and polyethylene terephthalate (PET).

A multi shot pyrolyzer (EGA/PY 3030D, Frontier Laboratories Ltd., Koriyama, Japan) was used and coupled to a GC–MS system (GCMS TQ8040 NX, Shimadzu Corporation, Kyoto, Japan). Samples were analyzed in single shot mode at a pyrolysis temperature of 600 °C with a holding time of 0.2 min. The Py GC/MS interface temperature was maintained at 320 °C. The pyrolyzer was directly connected to the GC/MS split/splitless (SSL) injector (Shimadzu Corporation, Kyoto, Japan), which operated in split mode with a split ratio of 50:1 and an injector temperature of 232 °C.

Separation of the pyrolysis products was achieved using an Ultra Alloy[®] 5 capillary column (30 m × 0.25 mm i.d. 0.25 µm film thickness) provided by Frontier Laboratories. Ultra-high purity helium served as the carrier gas at a constant flow rate of 1.87 mL min^{−1}. The GC oven temperature program consisted of an initial temperature of 40 °C held for 3 min. followed by a ramp of 30 °C min^{−1} to 100 °C with a 5 min hold, and a final ramp of 10 °C min^{−1} to 300 °C. maintained for 10 min. The total run time was 40 min.

The mass spectrometer operated in electron ionization (EI) mode at 70 eV, with the ion source temperature set to 230 °C and the interface temperature held at 320 °C. Data were acquired in full scan mode over an *m/z* range of 29–600.

Quality assurance and quality control (QA/QC) procedures were implemented throughout the analytical workflow. Blank samples were included in each analytical batch to monitor potential laboratory contamination, and repeatability analyses were performed to assess the reproducibility of polymer identification by Py-GC/MS. Polymer concentrations below the analytical limit of detection (LOD) were reported as <LOD. It should be noted that the analytical sensitivity of Py-GC/MS depends on several factors, including the biological matrix, sample volume, and instrumental performance; therefore, the absence of detectable concentrations should not be interpreted as the complete absence of a given polymer. Previous release experiments performed on the same polymeric materials used in hemodialysis systems have shown that detectable amounts of PVC, PET, and PE require the extraction of approximately 2 L of dialysate, a sampling approach that is not feasible for biological specimens [56]. Consequently, with the exception of polypropylene (PP), polymers not exceeding the analytical LOD were reported as <LOD. Chemical structures were identified by comparing the experimental mass spectra with those in the MS libraries (NIST11.Lib. NIST11s.Lib. NIST14.Lib. NIST14s.Lib. and WILEY8.Lib). Identification of pyrolysis products was further validated by comparing retention times and mass spectra with those obtained from the microplastics calibration standards (MPs SiO₂ L).

2.3.7. Statistical Data Analysis

Data were summarized using medians, interquartile ranges (IQRs), and 95% confidence intervals for quantitative variables, and frequencies and percentages for qualitative variables. In accordance with the American Statistical Association recommendations on statistical inference, the present study adopted a descriptive analytical approach and did not rely on null hypothesis significance testing or *p*-values. Accordingly, the analyses were intended to characterize exposure distributions across the study groups rather than to establish statistical associations or causal relationships. Consistent with the exploratory aim of this human biomonitoring study, no multivariable adjustment was undertaken, and potential confounding factors, including age, smoking history, occupational exposure, and other lifestyle characteristics, were considered when interpreting the findings. Therefore, the

observed exposure patterns should be interpreted as descriptive and hypothesis-generating rather than as evidence of independent associations [57–59]. All statistical analyses were performed using SPSS for Windows, version 31.0 (IBM, Armonk, NY, USA).

3. Results

3.1. General Characteristics of the Participants

We enrolled a total of 92 individuals (31 HD patients, 31 non-dialysis CKD patients, 30 healthy subjects). Table 1 shows the general characteristics of the participants comparing HD, non-dialysis CKD patients, and healthy subjects. Age, sex, marital status, education levels, and smoking habits were different between the compared groups. Most of the HD and non-dialysis CKD patients were retired and had worked in public administration or in education, health, and other social services.

Table 1. Characteristics of the study population.

Variables	HD Patients (n = 31) n (%)	Non-Dialysis CKD Patients (n = 31) n (%)	Healthy Subjects (n = 30) n (%)
Median age, y (IQR)	75.0 (63.0–80.0)	70.0 (55.0–80.0)	64.5 (52.0–72.8)
95% CI	(69.0–80.0)	(56.0–75.0)	(58.0–69.0)
Sex			
Female	14 (45.2)	15 (48.4)	15 (50.0)
Male	17 (54.8)	16 (51.6)	15 (50.0)
Marital status			
Unmarried *	12 (38.7)	14 (45.2)	8 (26.7)
Married/unmarried partner	19 (61.3)	17 (54.8)	22 (73.3)
Education level			
None and Primary school	16 (51.6)	6 (19.4)	4 (13.3)
Middle school	5 (16.1)	10 (32.3)	12 (40.0)
High school	8 (25.8)	8 (25.8)	11 (36.7)
Degree	2 (6.5)	7 (22.6)	3 (10.0)
Median BMI (IQR)	25.6 (21.9–27.5)	26.2 (22.1–28.4)	24.6 (21.9–28.9)
95% CI	22.8–27.3	23.0–27.6	22.8–28.1
Median KIDMED (IQR)	5 (5–8)	6 (5–8)	7 (5–9)
95% CI	5–7	5–8	6–8
Smoking habits			
No smoker	14 (45.2)	10 (32.3)	18 (60.0)
Smoker	2 (6.5)	10 (32.3)	5 (16.7)
Former smoker	15 (48.4)	11 (35.5)	7 (23.3)
Hypertension	3 (9.7)	0 (0)	14 (46.7)

* Separated/divorced, single, widowed; HD: Hemodialysis; CKD: Chronic kidney disease; IQR: Interquartile range (25th–75th); 95% CI: Confidence interval; BMI: Body mass index; KIDMED: Mediterranean Diet Quality Index.

3.2. Distribution of MNPs and BPA in Blood and Urine

Distribution of MNPs particle size, number per mL (p/mL), and BPA levels in blood and urine are reported in Table 2. The median blood plasma level of MNPs was higher in HD compared to non-dialysis CKD patients and healthy subjects. In contrast, no difference was found for blood MNPs diameter. CKD patients had higher median MNPs urinary levels than healthy subjects. The median diameter of urine MNPs was 5.9 (3.7–8.3) μm in the healthy subjects and 4.0 (2.7–4.7) μm in non-dialysis CKD patients. Median urinary BPA levels were lower in non-dialysis CKD patients than healthy subjects.

Table 2. Blood and urinary micro- and nanoplastics (MNPs) and urinary bisphenol A (BPA) distribution according to study groups.

Variables	HD Patients Median (IQR) 95% CI	Non-Dialysis CKD Patients Median (IQR) 95% CI	Healthy Subjects Median (IQR) 95% CI
Blood			
MNPs levels (p/mL) *	9889 (4525–14,939)	9234 (3354–15,429)	5755 (2488–9042)
	6620–13,396	4052–14,434	3270–6627
MNPs diameter (μm)	4.1 (3.3–4.7)	4.5 (3.8–5.1)	3.9 (3.5–4.5)
	3.8–4.4	4.0–4.9	3.7–4.4
Urine			
MNPs levels (p/mL) *	na	1231 (0–8281)	411 (0–1636)
		0–8199	0–2459
MNPs diameter (μm)	na	4.0 (2.7–4.7)	5.9 (3.7–8.3)
		2.7–4.7	2.9–8.3
BPA ($\mu\text{g/L}$)	na	0.16 (0.12–0.16)	0.98 (0.60–2.36)
		0.11–0.18	0.54–2.52
BPA ($\mu\text{g/g}$ creatinine)	na	0.15 (0.12–0.15)	0.93 (0.57–2.24)
		0.11–0.17	0.52–2.39

* p/mL: particles per milliliters; HD: Hemodialysis; CKD: Chronic kidney disease; IQR: Interquartile range (25th–75th); 95% CI: Confidence interval; MNPs: Micro- and nanoplastics; BPA: Bisphenol A; na: not applicable because dialysis subjects do not produce urine.

Table 3 shows the distribution of the number and size of blood MNPs according to the year of HD onset and the type of HD technique used. Patients who started HD in 2017 or earlier had a median MNPs level of 10,701 MNPs/mL (IQR: 7385–14,636), with a median MP diameter of 4.3 μm (IQR: 3.7–4.5). Those who began HD between 2018 and 2020 showed a similar MNPs level, with a median size of 4.0 μm (IQR: 3.1–4.8). Patients who initiated HD from 2021 onward exhibited a lower median MNPs level and slightly smaller MNPs.

Regarding HD techniques, patients undergoing hemodiafiltration (HDF) had a higher median MP concentration compared to those treated with acetate-free biofiltration with potassium profiling (AFB-K), whose median MP concentration was 8664 p/mL (IQR: 3784–13,327). The median MP size was similar between the two groups. Finally, stratified analyses by hemodialysis technique were performed for exploratory purposes and should be interpreted with caution given the limited sample size of the subgroups.

Table 3. Blood micro- and nanoplastics (MNPs) number and size according to year of hemodialysis onset and type of hemodialysis technique.

Variables	HD Patients n (%)	MNPs (p/mL) Median (IQR) 95% CI	MNPs Diameter (µm) Median (IQR) 95%CI
Year of HD onset			
≤2017	11 (35.5)	10,701 (7385–14,636)	4.3 (3.7–4.5)
		6620–15,846	3.2–4.7
2018–2020	15 (48.4)	10,646 (2495–19,777)	4.0 (3.1–4.8)
		2495–19,777	3.1–4.8
2021+	5 (16.1)	6272 (853–12,330)	3.8 (2.9–4.3)
		0–13,396	2.7–4.4
Type of HD technique			
HDF	6 (19.4)	15,004 (5569–27,483)	4.1 (3.6–4.6)
		2485–46,193	3.0–5.6
AFB-K	25 (80.6)	8664 (3784–13,327)	4.2 (3.2–4.7)
		4896–13,119	3.3–4.7

HD: Hemodialysis; MNPs: Micro- and nanoplastics; IQR: Interquartile range (25th–75th); 95% CI: Confidence interval; HDF: Hemodiafiltration; AFB-K: Acetate-free biofiltration with potassium profile.

3.3. Blood and Urine MNPs and BPA Distribution in HD, Non-Dialysis CKD, and Healthy Subjects by Sex

Table 4 shows blood and urine MNPs and BPA distribution in HD, CKD and healthy subjects stratified by sex.

Table 4. Blood and urine micro- and nanoplastics (MNPs) and bisphenol A levels by sex according to study group.

Variables	HD Patients		Non-Dialysis CKD Patients		Healthy Subjects	
	Median (IQR) 95% CI		Median (IQR) 95% CI		Median (IQR) 95% CI	
	Female	Male	Female	Male	Female	Male
Blood						
MNPs levels (p/mL) *	9907 (3382–13,499)	9063 (5322–18,405)	7340 (2495–15,429)	10,083 (4249–118,064)	6627 (3270–9944)	4131 (1665–6556)
	3290–14,637	4896–19,257	2495–15,429	4017–19,130	3270–9944	1665–6556
MNPs diameter (µm)	4.4 (3.8–4.8)	3.9 (3.1–4.3)	4.3 (3.6–5.4)	4.6 (3.9–5.0)	3.9 (3.3–4.6)	3.9 (3.7–4.4)
	3.3–4.9	3.1–4.3	3.6–5.4	3.9–5.0	3.3–4.6	3.7–4.4
Urine						

Table 4. Cont.

Variables	HD Patients Median (IQR) 95% CI		Non-Dialysis CKD Patients Median (IQR) 95% CI		Healthy Subjects Median (IQR) 95% CI	
	Female	Male	Female	Male	Female	Male
MNPs levels (p/mL) *	na	na	820 (0–6578)	1629 (0–8322)	812 (0–1636)	411 (103–1947)
			0–10,825	0–8363	0–8284	0–2459
MNPs diameter (µm)	na	na	4.0 (2.8–4.6)	4.1 (2.5–4.7)	4.0 (2.9–6.1)	6.0 (5.9–7.1)
			2.5–4.7	2.4–4.9	2.9–8.3	5.8–8.3
BPA (µg/L)	na	na	0.16 (0.11–0.18)	0.16 (0.13–0.16)	0.98 (0.65–2.52)	0.97 (0.54–1.40)
			0.04–0.32	0.12–0.16	0.11–4.44	0.54–1.39
BPA (µg/g creatinine)	na	na	0.15 (0.11–0.17)	0.15 (0.12–0.15)	0.93 (0.62–2.39)	0.92 (0.52–1.32)
			0.04–0.30	0.12–0.15	0.11–4.22	0.52–1.32

* p/mL: particles per milliliter; IQR: Interquartile range (25th–75th); 95% CI: Confidence interval; MNPs: Micro- and nanoplastics; BPA: Bisphenol A; na = not applicable because dialysis patients do not produce urine.

Stratified analysis by sex showed higher blood MNPs levels in females than in males in the healthy subjects group and HD group. Conversely, males in the non-dialysis CKD group had higher MNPs levels than females.

Urinary MNPs levels were also higher in females than in males in the healthy subjects group; conversely, males in the CKD group had higher MNPs levels than females. The MNPs in the urine of females in the healthy subjects group had a smaller diameter than the males, while those of the non-dialysis CKD group had values comparable between females and males. Both females and males in the healthy subjects had higher urinary BPA levels than non-dialysis CKD patients. Finally, stratified analyses by sex were performed for exploratory purposes and should be interpreted with caution given the limited sample size of the subgroups.

3.4. MNPs Chemical Composition Typization

Table 5 shows the chemical characterization of micro- and nanoplastics (MNPs) in blood and urine samples from healthy subjects, hemodialysis (HD), and non-dialysis CKD patients. Polyethylene (PE) was the predominant polymer in all groups, with the highest levels observed in the blood of dialysis patients and non-dialysis CKD patients. Polypropylene (PP) also showed higher values in the blood of dialysis patients compared to the other groups. The levels of polystyrene, polymethyl methacrylate, polyvinylchloride, polyethylene terephthalate, and N-66: nylon-6.6 were below their respective limits of detection (LODs) in all analyzed samples.

Table 5. Blood and urine micro- and nanoplastics (MNPs) chemical composition typization according to study group (µg/mL).

	LOD	Healthy Subject Blood	Dialysis Blood	Non-Dialysis CKD Blood	Non-Dialysis CKD Urine
PE	4.24	21.32	26.85	25.66	22.78
PP	1.07	1.80	3.34	<LOD	<LOD

PE: Polyethylene; PP: Polypropylene; LOD: Limit of detection.

4. Discussion

This study highlights a concerning accumulation of MNPs and altered BPA excretion patterns in non-dialysis CKD patients and HD patients, compared with healthy subjects. The higher internal burden observed among non-dialysis CKD and HD patients may reflect several non-mutually exclusive mechanisms, including impaired renal elimination, exposure associated with dialysis procedures, differences in environmental exposure patterns, or residual confounding related to demographic and lifestyle characteristics. However, owing to the cross-sectional design of the present study, these mechanisms cannot be distinguished.

These findings are consistent with previous human biomonitoring evidence indicating that MNPs can be detected in blood and may persist in the circulation, particularly in populations with compromised physiological clearance mechanisms [60–64].

Non-dialysis CKD patients also exhibited higher urinary MNP levels than healthy subjects, suggesting that some degree of urinary elimination is preserved despite reduced glomerular filtration rate. However, because exposure and disease status were assessed simultaneously, this observation should not be interpreted as evidence of a causal mechanism or enhanced detoxification. Rather, it may reflect pathological alterations of the glomerulo-tubular barrier, including loss of size and charge selectivity and impaired tubular handling, which may allow abnormal passage of particulate matter into the urine [65–67].

Recent mechanistic studies have demonstrated that micro- and nanoplastics can directly disrupt podocyte structure, alter slit diaphragm integrity, and increase glomerular permeability, supporting the hypothesis that MNPs may exacerbate glomerular barrier dysfunction in CKD [68]. Experimental models also show renal accumulation of MNPs associated with oxidative stress, tubular inflammation, and early fibrotic changes, further linking MNP exposure to impaired renal filtration mechanisms [69].

The smaller median diameter of urinary MNPs observed in non-dialysis CKD patients compared with healthy subjects further is compatible with selective retention of larger particles within the circulation or renal tissue, as previously suggested by experimental and human evidence on renal microplastic deposition [27,61,70].

This interpretation aligns with recent evidence showing that MNPs can be trapped within the renal microvasculature and peritubular capillaries, contributing to endothelial dysfunction, impaired vasodilation, and increased vascular oxidative stress, mechanisms known to accelerate CKD progression [71].

Experimental evidence suggests that MNPs exposure may promote inflammatory responses; however, the present study was not designed to investigate these mechanisms, which require confirmation in longitudinal and mechanistic studies [49].

Recent studies indicate that MNPs can activate renal inflammatory pathways, including NF- κ B and NLRP3 inflammasome signaling, promoting tubular injury and interstitial fibrosis, key processes in CKD progression [20,72].

Elevated MNP levels in non-dialysis CKD patients may also result from increased intestinal absorption secondary to disruption of the intestinal epithelial barrier, a common feature of the uremic state, which could enhance the translocation of luminal MNPs into the circulation [73,74].

Moreover, in relation to HD patients, the higher blood MNP levels compared with both non-dialysis CKD patients and healthy subjects may suggest that iatrogenic exposure cannot be excluded. In addition, patients undergoing hemodialysis for longer periods showed higher MNP levels than those treated for shorter periods. Likewise, differences according to dialysis technique may reflect variations in exposure to polymer-based medical devices and extracorporeal circuits. But these subgroup findings should be interpreted with caution because of the limited sample size within each dialysis subgroup.

The extensive use of plastic-based materials in HD procedures, including catheters, tubing, dialyzers, water treatment systems, and concentrate bags, may contribute to the observed MNP burden, in addition to the mechanisms hypothesized for non-dialysis CKD patients. These results are in line with the literature suggesting that exposure to MNPs during HD might play a role in the development of complications in this population, although dedicated longitudinal studies will be necessary to clarify the contribution of dialysis-related exposure [37,75,76].

The analysis of particle size distribution showed that healthy subjects excreted MNPs of greater size compared with non-dialysis CKD patients, who instead exhibited a higher presence of particles of increased diameter in the bloodstream. In contrast, HD patients had circulating particles of comparable size to those observed in healthy subjects, which may be compatible with a possible influence of the dialysis process on particle clearance according to diameter.

In terms of BPA exposure, CKD patients showed lower urinary BPA levels than healthy subjects, likely reflecting impaired renal elimination rather than reduced exposure, since BPA is primarily excreted through the kidneys and reduced renal function is associated with decreased clearance and systemic accumulation [77–79]. Indeed, previous studies have reported associations between elevated BPA levels, CKD progression, and increased cardiovascular risk [67,78,80,81]. Additional mechanisms potentially contributing to reduced urinary BPA include altered metabolism or increased intestinal reabsorption, possibly influenced by CKD-associated gut dysbiosis [82,83]. Nonetheless, further investigations, including serum BPA measurements, are needed to confirm systemic accumulation and to clarify its toxicological implications.

The sex-specific differences observed in the present study may reflect underlying biological differences in endocrine regulation, xenobiotic metabolism and immune responses. BPA is a well-established endocrine-disrupting chemical capable of interacting with estrogen and androgen signaling pathways [84,85], whereas growing evidence indicates that MNPs and their associated chemicals may also interfere with endocrine homeostasis by disrupting hypothalamic–pituitary endocrine axes, inducing oxidative stress and promoting chronic inflammation [86]. Consequently, sex-related differences in hormonal regulation may not only influence BPA toxicodynamics but also modify the biological responses to co-occurring MNPs exposure, potentially contributing to the sex-specific patterns observed in our study. In addition, chronic kidney disease is characterized by profound alterations of the gut microbiome and persistent systemic inflammation, while experimental studies indicate that MNP exposure may impair intestinal barrier integrity and alter gut microbial composition [36,87]. Although these mechanisms were not directly investigated in the present study, the higher circulating levels of BPA and MNPs observed in non-dialysis CKD patients and those undergoing hemodialysis are biologically consistent with the hypothesis that alterations in the gut–kidney axis may contribute to the internal burden of these contaminants. However, these mechanistic pathways remain speculative in the context of our findings and should be clarified by future studies integrating environmental exposure assessment with endocrine, inflammatory and gut microbiome biomarkers [28,36,84,86,87].

Emerging evidence shows that MNPs can aggravate gut dysbiosis, increase intestinal permeability, and enhance translocation of luminal particles and pro-inflammatory mediators into the bloodstream, thereby amplifying gut–kidney axis dysfunction and potentially contributing to renal injury [71,88].

In addition to differences between disease groups, this study identified exploratory sex-specific differences in blood MNP levels among non-dialysis CKD, HD patients and healthy subjects. Given the limited sample size within each subgroup, these observations should be interpreted cautiously and regarded as hypothesis-generating rather than confirmatory.

Experimental evidence suggests that biological sex may influence the toxicokinetic and biological effects of MNPs through differences in endocrine regulations, oxidative stress responses, and immune pathways; however, the available evidence derives predominantly from experimental models and remains insufficient to explain the heterogeneous patterns observed in the present study [68,69]. Therefore, the observed sex-specific differences should be considered preliminary and require confirmation in larger human biomonitoring studies specifically designed to investigate sex-related heterogeneity in internal exposure before any biological mechanisms can be inferred.

Overall, the present observations highlight the importance of considering biological sex in future environmental nephrology research.

Beyond their toxicological relevance, these observations may also have important implications for environmental health and public health. Patients with non-dialysis CKD and those undergoing hemodialysis may represent particularly vulnerable populations because of chronic disease, repeated exposure to polymer-based medical devices and the potential accumulation of environmental contaminants.

Recent studies further suggest that individuals with CKD exhibit heightened susceptibility to MNP-induced endothelial dysfunction and vascular inflammation, which may compound their already elevated cardiovascular and renal risk profiles [89,90].

Although the present study does not support specific clinical recommendations, it highlights the importance of precautionary strategies aimed at minimizing avoidable exposure to BPA and MNPs whenever feasible, including the optimization of polymer-based healthcare materials, the adoption of safer medical devices when available, and the implementation of environmental exposure awareness programs for healthcare professionals and patients. From a public health perspective, our findings support the inclusion of vulnerable renal populations in future biomonitoring programs and environmental risk assessment frameworks, thereby contributing to the identification of high-risk groups and to the development of evidence-based strategies for reducing exposure to emerging environmental contaminants.

Strengths and Limits

This study has two main strengths. To our knowledge, it is among the first cross-sectional biomonitoring studies to directly compare MNPs and BPA exposure across HD patients, non-dialysis CKD patients, and healthy subjects, focusing on sex differences. In addition, the use of sensitive and validated analytical methods enabled an accurate assessment of exposure levels in biological matrices. However, several limitations should be acknowledged. The study has inherent methodological constraints. First, the single-center design may limit generalizability, as the population reflects the demographic and clinical characteristics of one nephrology unit. Second, although the sample size was adequate for this exploratory human biomonitoring study, it limits statistical power to detect subtle differences between groups, particularly in subgroup analyses, and may reduce the generalizability of the findings. To strengthen analytical reliability, we have included detailed quality-control information for Py-GC/MS, including blank control results and repeatability assessments supporting MNP identification. In some studies, such as Kara et al. [56], release tests performed on the same materials used in our dialysis system have shown that detectable amounts of PVC, PET, and PE require the extraction of approximately 2 L of dialysate, an approach that is not feasible in biological matrices. Consequently, aside from PP, which reflects components of our dialysis device, trace polymers must be reported as <LOD, as analytical chemistry does not allow for an absolute zero and detection sensitivity depends on the matrix, sample volume, and instrumentation. Differences in age, comorbidities, and lifestyle characteristics between HD patients, non-dialysis patients

and healthy subjects may have introduced residual confounding, potentially influencing exposure patterns and biomarker distributions. The absence of serum BPA measurements limits the assessment of internal BPA dose, as reduced urinary levels may reflect impaired elimination rather than systemic accumulation. In addition, although both MNPs and BPA were measured in the same participants, the present study was not designed to evaluate correlations between these contaminants or their combined toxicological effects.

Furthermore, no predefined thresholds for clinically meaningful differences were established; therefore, all findings should be interpreted as exploratory and hypothesis-generating.

The contribution of healthcare-related sources of exposure, including repeated contact with polymer-based medical devices, laboratory equipment, and disposable consumables, was not specifically evaluated.

An additional limitation is that, although MNP particle size was available, individual renal function indicators, such as estimated glomerular filtration rate, serum creatinine, blood urea nitrogen or CKD stage, were not collected.

Although information on smoking habits, occupational history and lifestyle characteristics was collected, the present exploratory study was not designed to perform multivariable adjustment for potential confounders. Therefore, differences in internal MNPs burden should not be interpreted as being exclusively attributable to CKD or hemodialysis. Residual confounding remains a plausible explanation and should be addressed in larger prospective studies.

Finally, an important limitation concerns the assessment of BPA exposure. Although urinary BPA is widely recognized as a reliable biomarker of recent exposure because BPA is rapidly metabolized and predominantly excreted in urine, it mainly reflects short-term exposure and may not fully characterize systemic distribution or the internal burden of BPA. Since circulating MNPs were quantified in blood, the lack of concurrent serum BPA measurements limited our ability to directly compare the two contaminants within the same biological compartment and to better explore their potential interactions.

5. Conclusions

This study provides novel human biomonitoring evidence indicating that patients with CKD, particularly those undergoing hemodialysis, exhibit an increased systemic burden of MNPs alongside altered urinary elimination of BPA.

Importantly, sex-specific patterns emerged across study groups, highlighting the need to consider sex not merely as a confounder but as a potential effect modifier in environmental nephrology research.

Overall, the results underscore the relevance of environmental and healthcare-related plastic exposures in vulnerable populations with compromised renal function. Further longitudinal and multicenter studies are warranted to clarify exposure sources, underlying mechanisms, and potential long-term health implications, as well as to inform preventive strategies and risk assessment frameworks tailored to patients with chronic kidney disease.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/microplastics5030159/s1>, Table S1: Bisphenol A method performance parameters.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of University Hospital Policlinico “G. Rodolico—San Marco” (protocol code 30937-02.07.2021) on 2 July 2021.

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The datasets generated and analyzed during the current study are not publicly available due to privacy and ethical restrictions associated with human biological samples. Data are available from the corresponding author upon reasonable request.

Conflicts of Interest: The authors declare no conflicts of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript; or in the decision to publish the results. The method employed in this study is both nationally and internationally protected. The industrial patent No. 102018000003337, granted on 7 March 2018, is titled “Method for the extraction and determination of microplastics in samples with organic and inorganic matrices.” European Patent No. 3788344 was approved on 20 July 2022.

Abbreviations

The following abbreviations are used in this manuscript:

CKD	Chronic kidney disease
HD	Hemodialysis
MNPs	Micro- and nanoplastics
BPA	Bisphenol A
IQR	Interquartile range
SEM	Scanning electron microscopy

References

1. Plastics Europe. *Plastics—The Fast Facts 2024*; Plastics Europe: Bruxelles, Belgium, 2025.
2. Ritchie, H.; Samborska, V.; Roser, M. Plastic Pollution. Our World in Data. 2023. Available online: <https://ourworldindata.org/plastic-pollution> (accessed on 7 January 2026).
3. Walker, T.R.; Fequet, L. Current Trends of Unsustainable Plastic Production and Micro(Nano)Plastic Pollution. *TrAC Trends Anal. Chem.* **2023**, *160*, 116984. [[CrossRef](#)]
4. Wang, H.; Wang, Q.; Lv, M.; Li, J.; Zhao, X.; Song, Z.; Wang, X.; You, J.; Wang, Y.; Chen, L. Marine Micro(Nano)Plastics Determination and Its Environmental Toxicity Evaluation. *TrAC Trends Anal. Chem.* **2023**, *168*, 117332. [[CrossRef](#)]
5. Patel, M.M.; Goyal, B.R.; Bhadada, S.V.; Bhatt, J.S.; Amin, A.F. Getting into the Brain: Approaches to Enhance Brain Drug Delivery. *CNS Drugs* **2009**, *23*, 35–58. [[CrossRef](#)] [[PubMed](#)]
6. Tagg, A.S.; Ivar Do Sul, J.A. Is This Your Glitter? An Overlooked but Potentially Environmentally-Valuable Microplastic. *Mar. Pollut. Bull.* **2019**, *146*, 50–53. [[CrossRef](#)] [[PubMed](#)]
7. Dube, E.; Okuthe, G.E. Plastics and Micro/Nano-Plastics (MNPs) in the Environment: Occurrence, Impact, and Toxicity. *Int. J. Environ. Res. Public Health* **2023**, *20*, 6667. [[CrossRef](#)] [[PubMed](#)]
8. El Hadri, H.; Gigault, J.; Maxit, B.; Grassl, B.; Reynaud, S. Nanoplastic from Mechanically Degraded Primary and Secondary Microplastics for Environmental Assessments. *NanoImpact* **2020**, *17*, 100206. [[CrossRef](#)]
9. Amador, M.F.G.; Sansón, L.Z. Surface Dispersion of Coastal Discharges in North America towards the Great Pacific Garbage Patch. *Mar. Pollut. Bull.* **2023**, *191*, 114961. [[CrossRef](#)] [[PubMed](#)]
10. Vaseashta, A.; Klavins, M.; Stabnikova, O. *Micro and Nanoplastics in Aquatic Environment*, 1st ed.; CRC Press: Boca Raton, FL, USA, 2025; ISBN 978-1-003-38946-0.
11. Kumar, M.; Mrunalini, K.; Zhou, Z.; Modi, R.U.; Bolan, S.; Rao, C.S.; Braun, M.; Wrigley, O.; Li, Y.; Wang, L.; et al. Sources, Distribution and Migration of Micro-Nano-Plastics from Terrestrial to Aquatic Environments and Its Environmental and Health Impacts: A Systematic Review. *J. Soils Sediments* **2025**, *25*, 1767–1801. [[CrossRef](#)]

12. Zalasiewicz, J.; Waters, C.N.; Ivar Do Sul, J.A.; Corcoran, P.L.; Barnosky, A.D.; Cearreta, A.; Edgeworth, M.; Gałuszka, A.; Jeandel, C.; Leinfelder, R.; et al. The Geological Cycle of Plastics and Their Use as a Stratigraphic Indicator of the Anthropocene. *Anthropocene* **2016**, *13*, 4–17. [CrossRef]
13. Zuccarello, P.; Ferrante, M.; Cristaldi, A.; Copat, C.; Grasso, A.; Sangregorio, D.; Fiore, M.; Oliveri Conti, G. Exposure to Microplastics (<10 Mm) Associated to Plastic Bottles Mineral Water Consumption: The First Quantitative Study. *Water Res.* **2019**, *157*, 365–371. [CrossRef] [PubMed]
14. Vega-Herrera, A.; Llorca, M.; Borrell-Diaz, X.; Redondo-Hasselerharm, P.E.; Abad, E.; Villanueva, C.M.; Farré, M. Polymers of Micro(Nano) Plastic in Household Tap Water of the Barcelona Metropolitan Area. *Water Res.* **2022**, *220*, 118645. [CrossRef] [PubMed]
15. Oliveri Conti, G.; Ferrante, M.; Banni, M.; Favara, C.; Nicolosi, I.; Cristaldi, A.; Fiore, M.; Zuccarello, P. Micro- and Nano-Plastics in Edible Fruit and Vegetables. The First Diet Risks Assessment for the General Population. *Environ. Res.* **2020**, *187*, 109677. [CrossRef] [PubMed]
16. Garrido Gamarro, E. *Microplastics in Food Commodities*; FAO: Rome, Italy, 2022; ISBN 978-92-5-136982-1.
17. Habib, R.Z.; Kindi, R.A.; Salem, F.A.; Kittaneh, W.F.; Poulouse, V.; Iftikhar, S.H.; Mourad, A.-H.I.; Thiemann, T. Microplastic Contamination of Chicken Meat and Fish through Plastic Cutting Boards. *Int. J. Environ. Res. Public Health* **2022**, *19*, 13442. [CrossRef] [PubMed]
18. Borriello, L.; Scivico, M.; Cacciola, N.A.; Esposito, F.; Severino, L.; Cirillo, T. Microplastics, a Global Issue: Human Exposure through Environmental and Dietary Sources. *Foods* **2023**, *12*, 3396. [CrossRef] [PubMed]
19. Ali, N.; Katsouli, J.; Marczylo, E.L.; Gant, T.W.; Wright, S.; Bernardino de la Serna, J. The Potential Impacts of Micro-and-Nano Plastics on Various Organ Systems in Humans. *EBioMedicine* **2024**, *99*, 104901. [CrossRef] [PubMed]
20. Pulvirenti, E.; Ferrante, M.; Barbera, N.; Favara, C.; Aquilia, E.; Palella, M.; Cristaldi, A.; Conti, G.O.; Fiore, M. Effects of Nano and Microplastics on the Inflammatory Process: In Vitro and In Vivo Studies Systematic Review. *Front. Biosci. (Landmark Ed.)* **2022**, *27*, 287. [CrossRef] [PubMed]
21. Bishop, B.; Webber, W.S.; Atif, S.M.; Ley, A.; Pankratz, K.A.; Kostelecky, R.; Colgan, S.P.; Dinarello, C.A.; Zhang, W.; Li, S. Micro- and Nano-Plastics Induce Inflammation and Cell Death in Human Cells. *Front. Immunol.* **2025**, *16*, 1528502. [CrossRef] [PubMed]
22. Mahmud, F.; Sarker, D.B.; Jocelyn, J.A.; Sang, Q.-X.A. Molecular and Cellular Effects of Microplastics and Nanoplastics: Focus on Inflammation and Senescence. *Cells* **2024**, *13*, 1788. [CrossRef] [PubMed]
23. Zhang, Q.; He, Y.; Cheng, R.; Li, Q.; Qian, Z.; Lin, X. Recent Advances in Toxicological Research and Potential Health Impact of Microplastics and Nanoplastics In Vivo. *Environ. Sci. Pollut. Res.* **2022**, *29*, 40415–40448. [CrossRef] [PubMed]
24. Abraham, A.; Chakraborty, P. A Review on Sources and Health Impacts of Bisphenol A. *Rev. Environ. Health* **2020**, *35*, 201–210. [CrossRef] [PubMed]
25. Czarny-Krzyżnińska, K.; Krawczyk, B.; Szczukocki, D. Bisphenol A and Its Substitutes in the Aquatic Environment: Occurrence and Toxicity Assessment. *Chemosphere* **2023**, *315*, 137763. [CrossRef] [PubMed]
26. Commission Adopts Ban of Bisphenol A in Food Contact Materials—European Commission. Available online: https://food.ec.europa.eu/food-safety-news-0/commission-adopts-ban-bisphenol-food-contact-materials-2024-12-19_en (accessed on 26 June 2025).
27. Augey, L.; Berdough-Tritz, J.; Mbarek, A.; Jadoul, M.; Massy, Z.; Deray, G.; Liabeuf, S. Effects of Microplastics and Nanoplastics on the Kidneys. *Nephrol. Dial. Transplant.* **2026**, *41*, 1412–1420. [CrossRef] [PubMed]
28. Lombardi, M.; Bergesio, F. Micro- and Nanoplastics and the Kidney: Human Evidence, Clinical Vulnerabilities, Dialysis-Related Exposure, and CKD. *G. Clin. Nefrol. Dial.* **2026**, *38*, 82–88. [CrossRef]
29. Chen, D.; Kannan, K.; Tan, H.; Zheng, Z.; Feng, Y.-L.; Wu, Y.; Widelka, M. Bisphenol Analogues Other Than BPA: Environmental Occurrence, Human Exposure, and Toxicity—A Review. *Environ. Sci. Technol.* **2016**, *50*, 5438–5453. [CrossRef] [PubMed]
30. Nomiri, S.; Hoshyar, R.; Ambrosino, C.; Tyler, C.R.; Mansouri, B. A Mini Review of Bisphenol A (BPA) Effects on Cancer-Related Cellular Signaling Pathways. *Environ. Sci. Pollut. Res.* **2019**, *26*, 8459–8467. [CrossRef] [PubMed]
31. Qi, T.; Jing, D.; Zhang, K.; Shi, J.; Qiu, H.; Kan, C.; Han, F.; Wu, C.; Sun, X. Environmental Toxicology of Bisphenol A: Mechanistic Insights and Clinical Implications on the Neuroendocrine System. *Behav. Brain Res.* **2024**, *460*, 114840. [CrossRef] [PubMed]
32. Nie, H.; Wang, F.; Zhang, Y.; Zhang, S.; Han, X.; Zhang, X.; Guo, H.; He, M. Associations of Serum Bisphenol A Levels with Incident Chronic Kidney Disease Risk. *Sci. Total Environ.* **2021**, *771*, 145401. [CrossRef] [PubMed]
33. Lin, Y.-J.; Chang, J.-W.; Ponnusamy, V.K.; Huang, H.-B.; Chen, H.-C.; Huang, P.-C. Urinary Bisphenol A and Its Substitutes Exposure Increased the Risk of Renal Tubular Injury (N-Acetyl-β-d-Glucosaminidase) in the General Taiwanese Population. *Front. Public Health* **2025**, *13*, 1505578. [CrossRef] [PubMed]
34. Cambien, G.; Dupuis, A.; Belmouaz, M.; Bauwens, M.; Bacle, A.; Ragot, S.; Migeot, V.; Albouy, M.; Ayraud-Thevenot, S. Bisphenol A and Chlorinated Derivatives of Bisphenol A Assessment in End Stage Renal Disease Patients: Impact of Dialysis Therapy. *Ecotoxicol. Environ. Saf.* **2024**, *270*, 115880. [CrossRef] [PubMed]

35. Bello, A.K.; Okpechi, I.G.; Osman, M.A.; Cho, Y.; Htay, H.; Jha, V.; Wainstein, M.; Johnson, D.W. Epidemiology of Haemodialysis Outcomes. *Nat. Rev. Nephrol.* **2022**, *18*, 378–395. [CrossRef] [PubMed]
36. de Oliveira, R.B.; Pelepenko, L.E.; Masaro, D.A.; Lustosa, G.M.M.M.; de Oliveira, M.C.; Roza, N.A.V.; Marciano, M.A.; Dos Reis, L.M.; Kamel, S.; Louvet, L.; et al. Effects of Microplastics on the Kidneys: A Narrative Review. *Kidney Int.* **2024**, *106*, 400–407. [CrossRef] [PubMed]
37. Passos, R.S.; Davenport, A.; Busquets, R.; Selden, C.; Silva, L.B.; Baptista, J.S.; Barceló, D.; Campos, L.C. Microplastics and Nanoplastics in Haemodialysis Waters: Emerging Threats to Be in Our Radar. *Environ. Toxicol. Pharmacol.* **2023**, *102*, 104253. [CrossRef] [PubMed]
38. The Top 10 Causes of Death. Available online: <https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death> (accessed on 26 June 2025).
39. Kovesdy, C.P. Epidemiology of Chronic Kidney Disease: An Update 2022. *Kidney Int. Suppl.* **2022**, *12*, 7–11. [CrossRef] [PubMed]
40. Stevens, P.E.; Ahmed, S.B.; Carrero, J.J.; Foster, B.; Francis, A.; Hall, R.K.; Herrington, W.G.; Hill, G.; Inker, L.A.; Kazancioğlu, R.; et al. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. *Kidney Int.* **2024**, *105*, S117–S314. [CrossRef] [PubMed]
41. Lo Cicero, L.; Lentini, P.; Sessa, C.; Castellino, N.; D’Anca, A.; Torrisi, I.; Marcantoni, C.; Castellino, P.; Santoro, D.; Zanolì, L. Inflammation and Arterial Stiffness as Drivers of Cardiovascular Risk in Kidney Disease. *Cardiorenal Med.* **2025**, *15*, 29–40. [CrossRef] [PubMed]
42. Elendu, C.; Elendu, R.C.; Enyong, J.M.; Ibhiedu, J.O.; Ishola, I.V.; Egbunu, E.O.; Meribole, E.S.; Lawal, S.O.; Okenwa, C.J.; Okafor, G.C.; et al. Comprehensive Review of Current Management Guidelines of Chronic Kidney Disease. *Medicine* **2023**, *102*, e33984. [CrossRef] [PubMed]
43. Vaidya, S.R.; Aeddula, N.R. Chronic Kidney Disease. In *StatPearls*; StatPearls Publishing: Treasure Island, FL, USA, 2025.
44. Goodman, K.E.; Hua, T.; Sang, Q.-X.A. Effects of Polystyrene Microplastics on Human Kidney and Liver Cell Morphology, Cellular Proliferation, and Metabolism. *ACS Omega* **2022**, *7*, 34136–34153. [CrossRef] [PubMed]
45. Xiong, X.; Gao, L.; Chen, C.; Zhu, K.; Luo, P.; Li, L. The Microplastics Exposure Induce the Kidney Injury in Mice Revealed by RNA-Seq. *Ecotoxicol. Environ. Saf.* **2023**, *256*, 114821. [CrossRef] [PubMed]
46. Sato, Y.; Yanagita, M. Immune Cells and Inflammation in AKI to CKD Progression. *Am. J. Physiol. Ren. Physiol.* **2018**, *315*, F1501–F1512. [CrossRef] [PubMed]
47. Bektas, A.; Schurman, S.H.; Sen, R.; Ferrucci, L. Aging, Inflammation and the Environment. *Exp. Gerontol.* **2018**, *105*, 10–18. [CrossRef] [PubMed]
48. Bommer, J.; Gerns, D.; Waldherr, R.; Kessler, J.; Ritz, E. Plastic Filing from Dialysis Tubing Induces Prostanoid Release from Macrophages. *Kidney Int.* **1984**, *26*, 331–337. [CrossRef] [PubMed]
49. Tan, R.-Y.; She, Q.-Y.; Ma, Y.-C.; Liu, M.-H.; Li, L.-J.; Huang, L.-L.; Zhong, Y.-W.; Bi, H.-X. The Threat of Microplastics to Human Kidney Health: Mechanisms of Nephrotoxicity and Future Research Directions. *Environ. Res.* **2025**, *283*, 122124. [CrossRef] [PubMed]
50. Xu, J.-L.; Lin, X.; Wang, J.J.; Gowen, A.A. A Review of Potential Human Health Impacts of Micro- and Nanoplastics Exposure. *Sci. Total Environ.* **2022**, *851*, 158111. [CrossRef] [PubMed]
51. Cunha, S.C.; Fernandes, J.O. Quantification of Free and Total Bisphenol A and Bisphenol B in Human Urine by Dispersive Liquid–Liquid Microextraction (DLLME) and Heart-Cutting Multidimensional Gas Chromatography–Mass Spectrometry (MD–GC/MS). *Talanta* **2010**, *83*, 117–125. [CrossRef] [PubMed]
52. González, N.; Cunha, S.C.; Monteiro, C.; Fernandes, J.O.; Marquès, M.; Domingo, J.L.; Nadal, M. Quantification of Eight Bisphenol Analogues in Blood and Urine Samples of Workers in a Hazardous Waste Incinerator. *Environ. Res.* **2019**, *176*, 108576. [CrossRef] [PubMed]
53. Montano, L.; Raimondo, S.; Piscopo, M.; Ricciardi, M.; Guglielmino, A.; Chamayou, S.; Gentile, R.; Gentile, M.; Rapisarda, P.; Oliveri Conti, G.; et al. First Evidence of Microplastics in Human Ovarian Follicular Fluid: An Emerging Threat to Female Fertility. *Ecotoxicol. Environ. Saf.* **2025**, *291*, 117868. [CrossRef] [PubMed]
54. Ferrante, M.; Oliveri Conti, G.; Zuccarello, P. Patent Method for the Extraction and Determination of Micro- and Nano-Plastics in Organic and Inorganic Matrix Samples: An Application on Vegetals. *MethodsX* **2020**, *7*, 100989. [CrossRef] [PubMed]
55. Ferrante, M.; Pietro, Z.; Allegui, C.; Maria, F.; Antonio, C.; Pulvirenti, E.; Favara, C.; Chiara, C.; Grasso, A.; Omayma, M.; et al. Microplastics in Fillets of Mediterranean Seafood. A Risk Assessment Study. *Environ. Res.* **2022**, *204*, 112247. [CrossRef] [PubMed]
56. Kara, E.; Konur, K.; Koca, Y.Ş.; Aytan, Ü. Unveiling Hidden Contaminants: A Systematic Quantification and Characterization of Microplastics in Hemodialysis and Peritoneal Dialysis Fluids. *BMC Nephrol.* **2025**, *26*, 359. [CrossRef] [PubMed]
57. Ioannidis, J.P.A. The Importance of Predefined Rules and Prespecified Statistical Analyses: Do Not Abandon Significance. *JAMA* **2019**, *321*, 2067–2068. [CrossRef] [PubMed]
58. Onyije, F.M.; Olsson, A.; Baaken, D.; Erdmann, F.; Stanulla, M.; Wollschläger, D.; Schüz, J. Environmental Risk Factors for Childhood Acute Lymphoblastic Leukemia: An Umbrella Review. *Cancers* **2022**, *14*, 382. [CrossRef] [PubMed]

59. Wasserstein, R.L.; Schirm, A.L.; Lazar, N.A. Moving to a World Beyond “ $p < 0.05$ ”. *Am. Stat.* **2019**, *73*, 1–19. [[CrossRef](#)]
60. Ibrahim, Y.S.; Tuan Anuar, S.; Azmi, A.A.; Wan Mohd Khalik, W.M.A.; Lehata, S.; Hamzah, S.R.; Ismail, D.; Ma, Z.F.; Dzulkarnaen, A.; Zakaria, Z.; et al. Detection of Microplastics in Human Colectomy Specimens. *JGH Open* **2021**, *5*, 116–121. [[CrossRef](#)] [[PubMed](#)]
61. La Porta, E.; Exacoustos, O.; Lugani, F.; Angeletti, A.; Chiarenza, D.S.; Bigatti, C.; Spinelli, S.; Kajana, X.; Garbarino, A.; Bruschi, M.; et al. Microplastics and Kidneys: An Update on the Evidence for Deposition of Plastic Microparticles in Human Organs, Tissues and Fluids and Renal Toxicity Concern. *Int. J. Mol. Sci.* **2023**, *24*, 14391. [[CrossRef](#)] [[PubMed](#)]
62. Leslie, H.A.; van Velzen, M.J.M.; Brandsma, S.H.; Vethaak, A.D.; Garcia-Vallejo, J.J.; Lamoree, M.H. Discovery and Quantification of Plastic Particle Pollution in Human Blood. *Environ. Int.* **2022**, *163*, 107199. [[CrossRef](#)] [[PubMed](#)]
63. Prata, J.C.; da Costa, J.P.; Lopes, I.; Duarte, A.C.; Rocha-Santos, T. Environmental Exposure to Microplastics: An Overview on Possible Human Health Effects. *Sci. Total Environ.* **2020**, *702*, 134455. [[CrossRef](#)] [[PubMed](#)]
64. Fiore, M.; Bivona, F.; Palella, M.; Fatuzzo, P.M.; Zanolì, L.M.; Timperanza, C.; Longo, V.M.; Lo Cicero, L.; Oliveri Conti, G.; Ferrante, M. Exposure to Microplastics via Hemodialysis: Differences between Women and Men. *Eur. J. Public Health* **2024**, *34*, ckae144.1415. [[CrossRef](#)]
65. Haraldsson, B.; Nyström, J.; Deen, W.M. Properties of the Glomerular Barrier and Mechanisms of Proteinuria. *Physiol. Rev.* **2008**, *88*, 451–487. [[CrossRef](#)] [[PubMed](#)]
66. Lee, I.; Kim, Y.; Lee, J.P.; Kim, Y.; Lee, G.; Park, H.; Park, N.-Y.; Jung, S.; Kho, Y.; Lee, J.; et al. Influence of Urinary Creatinine Concentration and Specific Gravity Correction in Exposure Assessment: Comparison between Chronic Kidney Disease Patients and General Population. *Environ. Res.* **2025**, *286*, 122689. [[CrossRef](#)] [[PubMed](#)]
67. Lee, S.E.; Park, Y.S. Environmental Pollution and Its Impact on Kidney Diseases: A Comprehensive Review of Current Evidence. *Life* **2026**, *16*, 291. [[CrossRef](#)] [[PubMed](#)]
68. Jiang, N.; Zheng, X.; Zhang, N.; Cao, Y. The Detrimental Effects of Microplastic Exposure on Kidney Function. *Front. Med.* **2025**, *12*, 1620733. [[CrossRef](#)] [[PubMed](#)]
69. Lee, S.E.; Kim, D.Y.; Jeong, T.S.; Park, Y.S. Micro- and Nano-Plastic-Induced Adverse Health Effects on Lungs and Kidneys Linked to Oxidative Stress and Inflammation. *Life* **2025**, *15*, 392. [[CrossRef](#)] [[PubMed](#)]
70. Eknoyan, G.; Lameire, N.; Barsoum, R.; Eckardt, K.-U.; Levin, A.; Levin, N.; Locatelli, F.; MacLeod, A.; Vanholder, R.; Walker, R.; et al. The Burden of Kidney Disease: Improving Global Outcomes. *Kidney Int.* **2004**, *66*, 1310–1314. [[CrossRef](#)] [[PubMed](#)]
71. Huang, N.; Xu, M.; Sheng, L.; Sun, R. Polystyrene Microplastics Induce Nephrotoxicity via Oxidative Stress, Inflammation, Autophagy, and Ferroptosis. *J. Appl. Toxicol.* **2026**, 1–10. [[CrossRef](#)] [[PubMed](#)]
72. Chulkov, V.; Gasanov, M.; Isakov, V.; Denisenko, A.; Nwosu, C.; Rodkin, S. Molecular and Cellular Effects of Microplastics and Nanoplastics in the Pathogenesis of Cardiovascular, Nervous, Urinary, Digestive, and Reproductive System Diseases: A Global Systematic Review. *Int. J. Mol. Sci.* **2025**, *26*, 11194. [[CrossRef](#)] [[PubMed](#)]
73. Rysz, J.; Franczyk, B.; Ławiński, J.; Olszewski, R.; Ciałkowska-Rysz, A.; Gluba-Brzózka, A. The Impact of CKD on Uremic Toxins and Gut Microbiota. *Toxins* **2021**, *13*, 252. [[CrossRef](#)] [[PubMed](#)]
74. Dennis, J.; Arulraj, D.; Mistri, T.K. Unseen Toxins: Exploring the Human Health Consequences of Micro and Nanoplastics. *Toxicol. Rep.* **2025**, *14*, 101955. [[CrossRef](#)] [[PubMed](#)]
75. Li, Y.; Tao, L.; Wang, Q.; Wang, F.; Li, G.; Song, M. Potential Health Impact of Microplastics: A Review of Environmental Distribution, Human Exposure, and Toxic Effects. *Environ. Health* **2023**, *1*, 249–257. [[CrossRef](#)] [[PubMed](#)]
76. Tereshchenko, L.G.; Posnack, N.G. Does Plastic Chemical Exposure Contribute to Sudden Death of Patients on Dialysis? *Heart Rhythm* **2019**, *16*, 312–317. [[CrossRef](#)] [[PubMed](#)]
77. Krieter, D.H.; Canaud, B.; Lemke, H.-D.; Rodriguez, A.; Morgenroth, A.; von Appen, K.; Dragoun, G.-P.; Wanner, C. Bisphenol A in Chronic Kidney Disease. *Artif. Organs* **2013**, *37*, 283–290. [[CrossRef](#)] [[PubMed](#)]
78. Trasande, L.; Attina, T.M.; Trachtman, H. Bisphenol A Exposure Is Associated with Low-Grade Urinary Albumin Excretion in Children of the United States. *Kidney Int.* **2013**, *83*, 741–748. [[CrossRef](#)] [[PubMed](#)]
79. Sladič, M.; Smrkolj, Š.; Kavšek, G.; Imamovic-Kumalic, S.; Verdenik, I.; Virant-Klun, I. Bisphenol A in the Urine: Association with Urinary Creatinine, Impaired Kidney Function, Use of Plastic Food and Beverage Storage Products but Not with Serum Anti-Müllerian Hormone in Ovarian Malignancies. *Int. J. Mol. Sci.* **2025**, *26*, 4811. [[CrossRef](#)] [[PubMed](#)]
80. Carwile, J.L.; Michels, K.B. Urinary Bisphenol A and Obesity: NHANES 2003–2006. *Environ. Res.* **2011**, *111*, 825–830. [[CrossRef](#)] [[PubMed](#)]
81. Kang, J.-H.; Asai, D.; Toita, R. Bisphenol A (BPA) and Cardiovascular or Cardiometabolic Diseases. *J. Xenobiot.* **2023**, *13*, 775–810. [[CrossRef](#)] [[PubMed](#)]
82. Vaziri, N.D.; Wong, J.; Pahl, M.; Piceno, Y.M.; Yuan, J.; DeSantis, T.Z.; Ni, Z.; Nguyen, T.-H.; Andersen, G.L. Chronic Kidney Disease Alters Intestinal Microbial Flora. *Kidney Int.* **2013**, *83*, 308–315. [[CrossRef](#)] [[PubMed](#)]
83. Emanowicz, P.; Średnicka, P.; Wójcicki, M.; Roszko, M.; Juszczyk-Kubiak, E. Mitigating Dietary Bisphenol Exposure Through the Gut Microbiota: The Role of Next-Generation Probiotics in Bacterial Detoxification. *Nutrients* **2024**, *16*, 3757. [[CrossRef](#)] [[PubMed](#)]

84. Rochester, J.R. Bisphenol A and Human Health: A Review of the Literature. *Reprod. Toxicol.* **2013**, *42*, 132–155. [[CrossRef](#)] [[PubMed](#)]
85. Buoso, E.; Masi, M.; Limosani, R.V.; Oliviero, C.; Saeed, S.; Iulini, M.; Passoni, F.C.; Racchi, M.; Corsini, E. Endocrine Disrupting Toxicity of Bisphenol A and Its Analogs: Implications in the Neuro-Immune Milieu. *J. Xenobiot.* **2025**, *15*, 13. [[CrossRef](#)] [[PubMed](#)]
86. Ullah, S.; Ahmad, S.; Guo, X.; Ullah, S.; Ullah, S.; Nabi, G.; Wanghe, K. A Review of the Endocrine Disrupting Effects of Micro and Nano Plastic and Their Associated Chemicals in Mammals. *Front. Endocrinol.* **2023**, *13*, 1084236. [[CrossRef](#)] [[PubMed](#)]
87. Hobby, G.P.; Karaduta, O.; Dusio, G.F.; Singh, M.; Zybailov, B.L.; Arthur, J.M. Chronic Kidney Disease and the Gut Microbiome. *Am. J. Physiol. Ren. Physiol.* **2019**, *316*, F1211–F1217. [[CrossRef](#)] [[PubMed](#)]
88. Demarquoy, J. Effects of Micro and Nano Plastics on Renal Health. *World J. Nephrol.* **2026**, *15*, 115039. [[CrossRef](#)] [[PubMed](#)]
89. Lee, Y.-H.; Zheng, C.-M.; Wang, Y.-J.; Wang, Y.-L.; Chiu, H.-W. Effects of Microplastics and Nanoplastics on the Kidney and Cardiovascular System. *Nat. Rev. Nephrol.* **2025**, *21*, 585–596. [[CrossRef](#)] [[PubMed](#)]
90. Baaten, C.C.F.M.J.; Vondenhoff, S.; Noels, H. Endothelial Cell Dysfunction and Increased Cardiovascular Risk in Patients with Chronic Kidney Disease. *Circ. Res.* **2023**, *132*, 970–992. [[CrossRef](#)] [[PubMed](#)]

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