Nefrologia 46 (2026) 501407

Sociedad Española de Nefrología

nefrología

Revista de la Sociedad Española de Nefrología

journal homepage: www.revistanefrologia.com

Letter to the Editor

Micro/nanoplastics and kidney—Letter to the Editor

Micro/nanoplásticos y riñón—Carta al Editor

Dear Editor,

The rapid accumulation of plastic waste poses a significant global threat, with micro/nanoplastics emerging as key pollutants. Common plastic polymers degrade into micro/nanoplastics, which contaminate oceans and enter the human body via ingestion, inhalation, or dermal absorption. Once inside, micro/nanoplastics can accumulate in organs such as the lungs, kidneys, and brain, triggering oxidative stress, inflammation, and endocrine disruption. Annual human intake is estimated at 39,000–121,000 particles.[1] Recent studies suggest a growing link between MNP exposure and kidney dysfunction, making nephrotoxicity a rising concern[2,3] (Table 1).

Model organisms

The effects of polystyrene micro/nanoplastics (50 nm polystyrene nanoplastic as well as 4 μm, 600 nm and 300 nm polystyrene microplastic) were examined on male mice kidney.[4] As a result, histological impairment and inflammation of the kidney were observed beside the increase in mortality rate and significant weight loss observed across all treatments.[4] Furthermore, they have identified a plethora of biomarkers such as SOD, IL-6, MCP, CAT, IL-10, MDA and GSH-Px that are altered across control and micro/nanoplastic treatments in mice.[4] A long-term (28 days) polystyrene microplastic (1000 nm) ingestion experiment was conducted on juvenile rats.[5] The outcome showed that microplastics induced oxidative stress in both endoplasmic reticulum and kidney of treated rats.[5] Significant increase in apoptosis associated gene expression (caspase-12, Bax, caspase-3, Bcl-2 and caspase-9) was also detected in microplastic treated rats.[5]

The polystyrene micro/nanoplastic effects was experimented on kidney of model fish species zebrafish (*Danio rerio*) larvae.[6] Both the polystyrene microplastic (5 μm) and polystyrene nanoplastic (100 nm) induced severe kidney damage and innate immune function suppression in zebrafish larvae.[6] These micro- and nanoplastic treated larvae were also discovered to be more susceptible to *Edwardsiella piscicida* bacterial infection as compared to the negative controls. Additionally, the transcriptomic analysis revealed that cytosolic DNA-sensing and C-type lectin receptor pathways were compromised in both micro- and nanoplastics treated zebrafish groups.[6] They concluded that nanoplastics introduced stress within the endoplasmic reticulum whereas microplastics can lead to lipid accumulation in zebrafish larvae.[6]

Organoids

The toxicological effects of renal injury induced by inhalation of airborne polystyrene nanoplastics were investigated using a kidney organoid.[7] First, they set up a whole-body exposure system to allow the simulation of airborne nanoplastic inhalation exposure. Then, they have also established a cost-effective, reliable, and high-performing protocol for culturing 3D human kidney organoids.[7] Their results indicated that airborne nanoplastics deposition in kidney is possible via inhalation alone.[7] Polystyrene nanoplastics showed a tendency to promote apoptosis through the NR4A1/CASP3 signaling pathway and simultaneously activated the TF/F12 pathway, initiating an external coagulation cascade.[7] These polystyrene nanoplastics also contributed to dysfunction in the renin–angiotensin–aldosterone system (RAAS), impairing renal circulation and worsening kidney damage.[7] They deduced that chromic exposure to these airborne nanoplastics can lead to severe embryotoxicity, renal blood flow disturbance and nephrotoxicity.[7] However, due to limitations in experimental models, they mentioned that caution is advised when applying these findings to human health.

Table 1
The research summary of micro/nanoplastics in kidney.

Model type	Species/subtype	Exposure route	Main findings	Limitations
Model organism	Male mice	Ingestion	Mortality increase; weight loss	Small sample size (10–13 mice for each group)
	Juvenile rat	Ingestion	Oxidative stress	Small sample size (8 in each group)
	Zebrafish larvae	Direct exposure	Kidney damage; innate immune suppression	Short period of exposure (7 days)
Organoid	3D human kidney organoid	Whole-body exposure and airborne	Renal blood flow disturbance; kidney damage	Small sample size ($n = 5$)
Human cell line	Immortalized human podocytes and human glomerular endothelial cells	Direct exposure	Alteration in morphology and cytoskeleton	Sample size not reported
	HK-2	Direct exposure	Upregulation of Arg2 gene expression	Moderate sample size ($n = 24$)

https://doi.org/10.1016/j.nefroe.2026.501407
2013-2514/© 2025 Sociedad Española de Nefrología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Human cell line

The effects of 0.05 μm polystyrene nanoplastics on the kidney was investigated via cultured immortalized human podocytes and human glomerular endothelial cells.[8] The resultant treated podocytes and glomerular endothelial cells experienced alteration in both morphology and cytoskeleton, but the cell specific markers did not depict significant differential expressions across treated and non-treated cells.[8] Micro/nanoplastics was subjected to normal human adult male kidney HK-2 cell line used commonly in toxicology researches.[9] Interestingly, they unearthed that the Arg2 gene expression was increased significantly upon micro/nanoplastic exposure in HK-2 cells.[9] The Arg2 gene encodes arginase 2, a mitochondrial enzyme involved in arginine metabolism. This enzyme plays indispensable roles in vascular function, metabolic processes as well as inflammation. Following lentivirus-induced knockdown of the Arg2 gene, HK-2 cells exposed to micro/nanoplastics during sepsis exhibited SOD, GSH, and MDA levels comparable to those in non-septic HK-2 cells.[9] This indicates an enhanced resistance to oxidative stress adapted by the HK-2 cells treated with micro/nanoplastics. In the same study, they have also conducted similar treatment to mice and as a result the mortality rate of mice was elevated due to exacerbated kidney damage induced by micro/nanoplastic ingestion.[9] Similarly, the Arg2 gene expression in micro/nanoplastic treated mice was also found to be significantly abundant than their non-treated counterparts.[9]

Future outlooks

Currently, no established method exists to remove nanoplastics from human kidneys, but emerging strategies include enhancing renal clearance through hydration and nephroprotective agents, using nanomedicine tools like plastic-binding nanoparticles, and improving dialysis techniques. Experimental approaches such as magnetic nanocarriers, AI-driven detox planning, and gut microbiota modulation are also being explored. Continued interdisciplinary research is crucial to develop safe, effective removal techniques for this growing environmental and health concern. Standardized exposure models, 3D organoids, and omics approaches[10] are essential for deeper insight. Integrating AI[3] can accelerate research and boost prediction accuracy. Interdisciplinary efforts are vital to understand chronic impacts and guide public health and regulatory responses.

References

1. Cox KD, Covernton GA, Davies HL, Dower JF, Juanes F, Dudas SE. Human consumption of microplastics. Environ Sci Technol. 2019;53:7068–74.
2. Lim LWK. Linking microbiome to cancer: a mini-review on contemporary advances. Microbes. 2025;6100279.
3. Lim LWK. Implementation of artificial intelligence in aquaculture and fisheries: deep learning, machine vision, big data, Internet of things, robots and beyond. J Comput Cogn Eng. 2024;3:112–8.
4. Meng X, Zhang J, Wang W, Gonzalez-Gil G, Vrouwenvelder JS, Li Z. Effects of nano- and microplastics on kidney: physicochemical properties, bioaccumulation, oxidative stress and immunoreaction. Chemosphere. 2022;288132631.
5. Wang W, Guan J, Feng Y, Nie L, Xu Y, Xu H, et al. Polystyrene microplastics induced nephrotoxicity associated with oxidative stress, inflammation, and endoplasmic reticulum stress in juvenile rats. Front Nutr. 2023;91059660.
6. Yang H, Ju J, Wang Y, Zhu Z, Lu W, Zhang Y. Micro-and nano-plastics induce kidney damage and suppression of innate immune function in zebrafish (*Danio rerio*) larvae. Sci Total Environ. 2024;931172952.
7. Chen L, Han B, Yang S, Guo L, Zhao L, Liu P, et al. Toxicological effects and mechanisms of renal injury induced by inhalation exposure to airborne nanoplastics. J Hazard Mater. 2025;488137393.
8. Melina Y, Michelle H, Marie H, Mustafa K, Zeynad M, Mario S, et al. Effects of nanoplastic particles on the kidney. J Am Soc Nephrol. 2024;35(10S), http://dx.doi.org/10.1681/ASN. 2024b4g2q3f2
9. Zha Y, Cheng Y, Yu S, Cheng Y, Wang J, Han X, et al. Long-term micro/nanoplastic ingestion promotes sepsis by worsening kidney damage: a transcriptomics and metabolomics study. Environ Sci Nano. 2025. Retrieved from: https://pubs.rsc.org/en/content/articlelanding/2025/en/d4en01206b
10. Lim LWK. Comparative genomic analysis reveals the origin and global distribution of melon necrotic virus isolates. Gene Rep. 2022;29101685.

Leonard Whye Kit Lim

Faculty of Resource Science and Technology, Universiti Malaysia Sarawak, Kota Samarahan, Sarawak, Malaysia

E-mail address: limwhyekitleonard@gmail.com