J Clin Haematol. 2026;7(1):51–56.

Journal of Clinical Haematology Review Article

Could Microplastics or Nanoplastics Be Driving the Lymphoma Epidemic?

Naveen Kumar[1,*]

[1]Assistant Professor, Department of Zoology, School of Basic and Applied Sciences, Sanskaram University, Patauda - 124108, Jhajjar, Haryana, India

[*]Correspondence should be addressed to Naveen Kumar, Naveentak72@gmail.com

Received date: December 11, 2025, **Accepted date:** April 10, 2026

Citation: Kumar N. Commentary: Could Microplastics or Nanoplastics Be Driving the Lymphoma Epidemic?. J Clin Haematol. 2026;7(1):51–56.

Copyright: © 2026 Kumar N. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Lymphoma therapeutics research revealed an unsettling parallel: the plastic we consume daily might contribute to the very disease we're trying to cure. Microplastics-and particularly nanoplastics with their superior ability to enter the bloodstream-are now detected in human blood, lungs, and placentas. While direct evidence linking them to lymphoma remains absent, our recent comprehensive mechanistic review hypothesized biological plausibility through five convergent pathways-inflammation, oxidative stress, immune dysregulation, genotoxicity, and endocrine disruption. Nanoplastics, with their enhanced bioavailability and cellular penetration, may represent the primary etiological agents, though microplastics serve as the persistent environmental reservoir from which nanoplastics continuously fragment. Unlike therapeutic compounds that kill cancer cells through acute pathway activation, chronic low-dose plastic exposure may promote cancer through the same mechanisms. Critical evidence gaps exist-no studies have measured plastic particles in lymph nodes or compared exposures between lymphoma patients and controls-making urgent investigation necessary. This hypothesis, while mechanistically plausible based on our recent comprehensive review, requires rigorous testing through epidemiological studies, lymph node tissue analysis, and animal carcinogenesis bioassays before any causal claims can be made.

Introduction

Microplastics are everywhere. Studies detect concentrations up to 1000 particles/L in seawater and up to 10 particles/cm^3 in the atmosphere [1]. Humans can be exposed to more than 48,000 microplastic particles/day through inhalation alone [2]. These tiny fragments-smaller than 5 mm–are now in human blood, lungs, placentas, and breast milk [3,4].

Our recent review termed microplastics the "hidden poison"–a phrase reflecting a troubling reality we create daily yet fail to see [5,6]. These particles persist for thousands of years, continuously releasing toxic chemicals throughout their slow degradation [6]. As larger plastics break down into smaller fragments, microplastic concentrations will keep rising for millennia, even if all plastic production stopped today. Critically, this fragmentation process generates nanoplastic-particles <1 μm that exhibit fundamentally different biological behavior and substantially greater toxicity potential than their larger microplastic precursors. We're not just dealing with today's pollution; we're creating a toxic legacy that will accumulate and intensify for generations. While scientists debate their immediate health effects, an urgent connection may be going unrecognized: nanoplastics, more so than microplastics, could be contributing to the global rise in lymphoma.

From Curing Lymphoma to Proposing Its Cause

Research on lymphoma therapeutics has revealed how certain compounds kill cancer through oxidative stress and mitochondrial damage. Nucleoside analogs like gemcitabine, fludarabine, and cladribine induce lymphoma cell death through rapid reactive oxygen species (ROS) generation that overwhelms mitochondrial defenses [7,8]. Our laboratory work with FNC (2′-deoxy-2′-β-fluoro-4′-azidocytidine, also known as Azvudine) demonstrated similar mechanisms–when cancer cells experience intense, sudden stress, a burst of ROS flooding their mitochondria triggers death [9–11]. That's how treatment works.

Recent work on environmental toxins, particularly micro- and nanoplastics, has uncovered something striking. Multiple research groups have shown that these plastic particles trigger the same mechanisms–ROS generation, mitochondrial dysfunction, inflammation–but differently [12–15]. Instead of an intense burst that kills cells, plastic particles create chronic, low-level stress over years. Nanoplastics, with their ability to penetrate cells and reach mitochondria directly, may exert these effects more potently than larger microplastics that remain primarily extracellular [16]. Same pathways, opposite outcome: instead of killing cancer, this chronic stress may cause it [6].

The toxicological principle is well-established: the dose makes poison [17]. A sudden, high dose of ROS kills lymphoma cells therapeutically. Chronic, low doses may promote cancer through accumulated DNA damage [18]. However, critical gaps exist: Most studies use concentrations potentially exceeding human tissue exposures, and exposure durations fall far short of decades-long human exposures. Whether environmentally relevant micro- and nanoplastic concentrations induce sufficient stress to promote lymphomagenesis over human lifespans remains unknown.

The Nanoplastic Distinction: Size Determines Biological Fate

While microplastics (1 µm–5 mm) dominate discussions in community nowadays, nanoplastics (<1 µm, particularly <100 nm) may represent the primary biological threat due to fundamentally different properties. This size-dependent toxicity mirrors established nanotoxicology principles [19,20].

Enhanced blood entry

Particles <200nm cross epithelial and endothelial barriers through transcytosis with exponentially greater efficiency than microplastics [21,22]. Specialized gut cells preferentially transport particles 50–100 nm [23]. This means nanoplastics achieve systemic distribution while most microplastics remain in the gastrointestinal tract.

Cellular penetration

Nanoplastics enter cells via endocytosis, reaching mitochondria and nuclei-sites of direct molecular damage [16]. Microplastics remain largely extracellular. This intracellular access dramatically amplifies nanoplastic toxicity potential.

Lymphoid accumulation

Biodistribution studies show nanoparticles accumulate preferentially in spleen and lymph nodes at 4–8-fold higher concentrations than other organs [23,24]. This lymphoid tropism makes nanoplastics particularly relevant to lymphoma etiology.

The continuum matters

Microplastics continuously fragment into nanoplastics through environmental weathering [25]. Environmental microplastics serve as persistent reservoirs generating escalating nanoplastic exposure over time. This means nanoplastic concentrations will continue rising for centuries even if plastic production ceased today.

Critical limitation

Current analytical methods cannot reliably detect nanoplastics <500 nm–precisely the size fraction with potentially greatest toxicity [26]. True human nanoplastic burdens remain essentially unknown, meaning we may be dramatically underestimating actual exposure levels.

Where Do Plastics Go after Entering Blood?

Once micro- and nanoplastics cross into the bloodstream-and evidence confirms they do [3,4]–where do they accumulate? Basic anatomy suggests: lymph nodes. Given nanoplastics' superior vascular translocation capacity, they likely reach and accumulate in lymph nodes more efficiently than larger microplastics.

Lymph nodes are biological filters designed to trap foreign particles and activate immune responses [27]. Blood flows through them constantly. If plastic particles circulate in blood, anatomical logic predicts they should concentrate in lymph nodes. And here's the critical point: lymph nodes are where most lymphomas begin [28].

Consider other cancers where microplastic exposure has been directly linked to cancer development: Studies demonstrate that microplastic exposure promotes lung cancer through oxidative stress, inflammation, and epithelial-mesenchymal transition (EMT) pathways [29,30]. Microplastics detected in lung tissues show associations with increased cancer biomarkers and inflammatory responses [31,32]. For gastrointestinal cancers, research links microplastic exposure to colorectal cancer development through gut microbiota disruption, chronic inflammation, and direct genotoxicity [12,33], with higher microplastic concentrations detected in cancerous colon tissues compared to healthy tissues [34]. Skin cancer risk increases with dermal microplastic exposure through UV-induced degradation of plastic particles that generate reactive oxygen species and promote melanoma cell proliferation [35,36]. While these associations do not establish definitive causation-cancer development involves multiple factors including genetics, chronic inflammation, and other environmental exposures-the pattern is compelling: cancer appears where plastic particles accumulate and exert chronic toxic effects. Microplastics may be one of several environmental factors contributing to carcinogenesis, not the

sole cause. Nanoplastics' enhanced tissue penetration and preferential lymphoid accumulation make them particularly concerning for lymphoma development. Yet remarkably, despite clear links between microplastic exposure and lung, skin, and gastrointestinal cancers, no published studies have investigated whether micro- or nanoplastics collect in lymph nodes or examined their role in lymphoma specifically. This represents a critical gap requiring urgent investigation, particularly given that lymph nodes–unlike solid organs where plastic-cancer links have been established–are designed specifically to filter and trap circulating particles.

Five Mechanisms, Plausible Hypothesis

Our comprehensive mechanistic review systematically examined how microplastics may affect pathways known to cause lymphoma [6]. While we hypothesized that microplastics could activate these pathways, nanoplastics also appear more biologically plausible as primary drivers due to their superior bioavailability and cellular access. The evidence suggests plastic particles can activate all five canonical pathways:

A. Chronic inflammation: Micro- and nanoplastics activate immune signaling, releasing inflammatory molecules like IL-6 and TNF-α [14,15]. They also activate NLRP3 inflammasome, releasing cytokines that support lymphoma cell proliferation [37]. This persistent inflammation resembles conditions driving lymphomas through chronic bacterial infections [38]. Nanoplastics' ability to penetrate immune cells directly may trigger more potent inflammatory responses than extracellular microplastics.

B. Oxidative stress: While our laboratory demonstrated that intense oxidative stress kills lymphoma cells therapeutically [10,11], plastic particles produce the opposite-mild oxidative stress continuously, damaging DNA slowly over time rather than destroying cells outright [12,13]. Nanoplastics' mitochondrial localization enables direct ROS generation at the source, potentially explaining their greater genotoxic potential compared to microplastics. The difference resembles a knockout punch versus slow poisoning.

C. Immune system disruption: Studies show plastic particle exposure alters T-cell and B-cell function, reduces NK cell activity by ~40%, and promotes T-cell exhaustion through immune checkpoint upregulation [39–41]. The result: reduced cancer protection combined with increased lymphocyte division-precisely the wrong combination. Nanoplastics' systemic distribution and lymphoid accumulation suggest they may drive these immune disruptions more effectively than microplastics.

D. DNA damage: Plastic particles induce chromosomal aberrations, micronucleus formation, and oncogenic translocations [42]. They also cause epigenetic modifications including DNA methylation changes affecting lymphocyte development [43,44]. As vectors for environmental toxicants, plastic particles deliver additional genotoxic compounds to tissues. Nanoplastics' nuclear penetration capacity enables direct DNA interaction, potentially explaining observations of enhanced genotoxicity with smaller particle sizes.

E. Hormone disruption: Plastic-associated chemicals like BPA and phthalates activate estrogen receptors and JAK/STAT signaling, promoting lymphocyte proliferation and inhibiting apoptosis through Bcl-2 overexpression [45,46]. Nanoplastics' greater surface-area-to-mass ratio amplifies chemical leaching, potentially intensifying endocrine disruption compared to larger microplastics.

The logic appears straightforward: Lymphoma develops through pathways A, B, C, D, and E. Micro- and nanoplastics activate pathways A, B, C, D, and E. Nanoplastics activate these pathways more efficiently due to enhanced bioavailability and cellular penetration. Therefore, nanoplastics and microplastics represent a plausible contributing factor worthy of urgent investigation.

However, critical gaps prevent definitive conclusions: Most data derive from non-lymphoid tissues at supraphysiological concentrations. No studies demonstrate that human exposure levels simultaneously activate all five pathways in lymphoid tissues specifically. Crucially, no studies have systematically compared the effects of size-fractionated particles (e.g., 10 µm microplastics vs. 100 nm nanoplastics) on lymphocyte biology. No mechanistic studies link pathway activation to actual malignant transformation in lymphocytes.

Why Lymphoma Instead of Leukemia?

If plastic particles circulate in blood and evidence confirms they do [3,4,47]–why might they affect lymphoma risk more than leukemia (blood cancer originating in bone marrow)? The answer may lie in anatomy. Lymph nodes receive massive blood flow and actively filter particles–creating maximum exposure [27,48]. Bone marrow receives lower blood flow and is partially protected by the blood-marrow barrier [49]. This exposure gradient predicts lymphoma risk should exceed leukemia risk if plastics contribute to hematologic malignancy. Nanoplastics' preferential accumulation in lymphoid tissues rather than bone marrow further supports this anatomical hypothesis.

However, this requires evidence. No studies have compared micro- or nanoplastic concentrations in lymph nodes versus bone marrow. No epidemiological studies have examined whether plastic exposure shows differential associations with lymphoma versus leukemia risk.

What Needs to Happen Now

For clinicians: Lymphoma patient assessments could include environmental exposure histories–occupation, diet, plastic product use. Environmental factors deserve systematic documentation for future research.

For researchers: Several urgent questions demand investigation:

- **Case-control studies** comparing size-fractionated microplastic and nanoplastic concentrations between lymphoma patients and healthy controls, with particular emphasis on measuring the nanoplastic fraction currently invisible to most analytical methods
- **Direct measurements** of plastic particle concentrations in lymph node biopsies from lymphoma patients and controls, using advanced techniques capable of detecting nanoplastics <500 nm
- **Animal studies** testing whether chronic exposure to size-fractionated particles (comparing microplastics vs. nanoplastics) at environmentally relevant concentrations increases lymphoma incidence
- **Mechanistic investigations** examining whether chronic low-dose nanoplastic exposure induces cellular dysfunction at levels that promote rather than prevent malignancy, testing the hypothesis that nanoplastics exhibit greater lymphomagenic potential than microplastics due to enhanced cellular penetration.

For public health: Historical precedents exist. Asbestos regulation reduced mesothelioma. Smoking reduction lowered lung cancer rates [50]. If micro- and nanoplastics significantly contribute to lymphoma risk, this could represent preventable cancer amenable to policy intervention.

The Urgency

Unlike most toxins, microplastics never break down. They persist for centuries [49,51]. Humans inhale over 48,000 particles daily [2]. Infants are born with microplastics already in their bodies [4]. Every year of delay means more plastic accumulates in human tissues. Moreover, as microplastics fragment into nanoplastics through environmental weathering, their toxicity potential increases exponentially while environmental concentrations escalate-meaning the biological threat intensifies over time even as total polymer mass remains constant.

Society didn't wait for absolute proof before regulating asbestos or tobacco. Action came from strong evidence and plausible mechanisms [52]. The nanoplastic-lymphoma hypothesis-while unproven-merits serious investigation given universal exposure, rising lymphoma incidence, anatomical logic, and mechanistic coherence across established lymphoma pathways. This commentary proposes a testable hypothesis requiring urgent investigation, not a proven causal relationship. The precautionary principle suggests that when a plausible mechanism exists alongside universal exposure and rising disease rates, investigation should not await definitive proof-particularly for a contaminant that will persist and accumulate for centuries regardless of future regulatory action.

From Treatment to Prevention

The research journey from lymphoma therapeutics to environmental toxicology has revealed a thought-provoking possibility: the cellular mechanisms exploited for therapy may mirror those that environmental plastics exploit to promote disease. Medicine may be treating lymphoma while overlooking significant environmental contributors to its rising incidence.

This hypothesis demands urgent testing. If nanoplastics prove to be significant contributors, humanity may face a preventable cancer epidemic that grows with every plastic product manufactured-not because of the total plastic mass produced, but because of the invisible nanoplastic particles continuously generated as environmental plastics fragment. The plastic consumed this week might be more than pollution–it could represent a long-term health hazard we're only beginning to recognize.

The question isn't whether there's enough evidence to prove causation-there isn't. The question is whether the mechanistic plausibility, anatomical logic, universal exposure, and potential public health implications justify immediate, well-funded investigation. Given that nanoplastics combine universal human exposure with enhanced bioavailability, cellular penetration, and lymphoid accumulation-all properties absent in larger microplastics–the answer appears clear: this hypothesis warrants rigorous scientific testing through the research priorities outlined above, with particular emphasis on size-fractionated analyses that can distinguish microplastic from nanoplastic effects.

Competing Interests

The author declares no competing interests.

Acknowledgments

I thank anonymous peer reviewers for constructive feedback that substantially improved this commentary.

References

1. Kiran BR, Kopperi H, Venkata Mohan S. Micro/nano-plastics occurrence, identification, risk analysis and mitigation: challenges and perspectives. Rev Environ Sci Biotechnol. 2022;21(1):169–203.
2. Krishnan K. A systematic review on the impact of micro-nanoplastics exposure on human health and diseases. Biointerface Research in Applied Chemistry. 2023;13(4):1–12.
3. Leslie HA, van Velzen MJM, Brandsma SH, Vethaak AD, Garcia-Vallejo JJ, Lamoree MH. Discovery and quantification of plastic particle pollution in human blood. Environ Int. 2022 May;163:107199.
4. Ragusa A, Svelato A, Santacroce C, Catalano P, Notarstefano V, Carnevali O, et al. Plasticenta: First evidence of microplastics in human placenta. Environ Int. 2021 Jan;146:106274.
5. Joseph MM, Nair JB, Joseph AM. Microscopic menace: exploring the link between microplastics and cancer pathogenesis. Environ Sci Process Impacts. 2025 Jul 16;27(7):1768–95.
6. Kumar N, Pachar AK, Singh JK, Mandal M, Lamba M, Yadav S, et al. Investigating the hypothesis role of the hidden poison microplastics in lymphoma development. Discover Environment. 2025 Jun 14;3(1):73.
7. Galmarini CM, Mackey JR, Dumontet C. Nucleoside analogues and nucleobases in cancer treatment. Lancet Oncol. 2002 Jul;3(7):415–24.
8. Genini D, Adachi S, Chao Q, Rose DW, Carrera CJ, Cottam HB, et al. Deoxyadenosine analogs induce programmed cell death in chronic lymphocytic leukemia cells by damaging the DNA and by directly affecting the mitochondria. Blood. 2000 Nov 15;96(10):3537–43.
9. Kumar N, Delu V, Ulasov I, Kumar S, Singh RK, Kumar S, et al. Pharmacological Insights: Mitochondrial ROS Generation by FNC (Azvudine) in Dalton's Lymphoma Cells Revealed by Super Resolution Imaging. Cell Biochem Biophys. 2024 Jun;82(2):873–83.
10. Kumar N, Kumar S, Shukla A, Kumar S, Singh RK, Ulasov I, et al. Mitochondrial-mediated apoptosis as a therapeutic target for FNC (2'-deoxy-2'-b-fluoro-4'-azidocytidine)-induced inhibition of Dalton's lymphoma growth and proliferation. Discov Oncol. 2024 Jan 22;15(1):16.
11. Kumar N, Shukla A, Kumar S, Ulasov I, Singh RK, Kumar S, et al. FNC (4'-azido-2'-deoxy-2'-fluoro(arbino)cytidine) as an Effective Therapeutic Agent for NHL: ROS Generation, Cell Cycle Arrest, and Mitochondrial-Mediated Apoptosis. Cell Biochem Biophys. 2024 Jun;82(2):623–39.
12. Jin W, Zhang W, Tang H, Wang P, Zhang Y, Liu S, et al. Microplastics exposure causes the senescence of human lung epithelial cells and mouse lungs by inducing ROS signaling. Environ Int. 2024 Mar;185:108489.
13. Kadac-Czapska K, Ośko J, Knez E, Grembecka M. Microplastics and Oxidative Stress-Current Problems and Prospects. Antioxidants (Basel). 2024 May 8;13(5):579.
14. Stock V, Böhmert L, Lisicki E, Block R, Cara-Carmona J, Pack LK, et al. Uptake and effects of orally ingested polystyrene microplastic particles in vitro and in vivo. Arch Toxicol. 2019 Jul;93(7):1817–33.
15. Woo JH, Seo HJ, Lee JY, Lee I, Jeon K, Kim B, et al. Polypropylene nanoplastic exposure leads to lung inflammation through p38-mediated NF-κB pathway due to mitochondrial damage. Part Fibre Toxicol. 2023 Jan 10;20(1):2.
16. Hou J, Lei Z, Cui L, Hou Y, Yang L, An R, et al. Polystyrene microplastics lead to pyroptosis and apoptosis of ovarian granulosa cells via NLRP3/Caspase-1 signaling pathway in rats. Ecotoxicol Environ Saf. 2021 Apr 1;212:112012.
17. Klaunig JE, Kamendulis LM, Hocevar BA. Oxidative stress and oxidative damage in carcinogenesis. Toxicol Pathol. 2010 Jan;38(1):96–109.
18. Moustacchi E. DNA damage and repair: consequences on dose-responses. Mutat Res. 2000 Jan 3;464(1):35–40.
19. Nel A, Xia T, Mädler L, Li N. Toxic potential of materials at the nanolevel. Science. 2006 Feb 3;311(5761):622–7.
20. Oberdörster G, Oberdörster E, Oberdörster J. Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles. Environ Health Perspect. 2005 Jul;113(7):823–39.
21. Geiser M, Rothen-Rutishauser B, Kapp N, Schürch S, Kreyling W, Schulz H, et al. Ultrafine particles cross cellular membranes by nonphagocytic mechanisms in lungs and in cultured cells. Environ Health Perspect. 2005 Nov;113(11):1555–60.
22. Hussain N, Jaitley V, Florence AT. Recent advances in the understanding of uptake of microparticulates across the gastrointestinal lymphatics. Adv Drug Deliv Rev. 2001 Aug 23;50(1-2):107–42.
23. Jani P, Halbert GW, Langridge J, Florence AT. Nanoparticle uptake by the rat gastrointestinal mucosa: quantitation and particle size dependency. J Pharm Pharmacol. 1990 Dec;42(12):821–6.
24. Carr KE, Smyth SH, McCullough MT, Morris JF, Moyes SM. Morphological aspects of interactions between microparticles and mammalian cells: intestinal uptake and onward movement. Prog Histochem Cytochem. 2012 Jan;46(4):185–252.
25. Lambert S, Wagner M. Microplastics are contaminants of emerging concern in freshwater environments: an overview. In: Wagner M, Lambert S, Eds. Freshwater Microplastics. The Handbook of Environmental Chemistry, vol 58. Cham: Springer; 2018. pp. 1–23.
26. Gigault J, Halle AT, Baudrimont M, Pascal PY, Gauffre F, Phi TL, et al. Current opinion: What is a nanoplastic? Environ Pollut. 2018 Apr;235:1030–4.
27. Willard-Mack CL. Normal structure, function, and histology of lymph nodes. Toxicol Pathol. 2006;34(5):409–24.

28. Swerdlow SH, Campo E, Harris NL, Jaffe ES, Pileri SA, Stein H, et al. WHO classification of tumours of haematopoietic and lymphoid tissues. In: Swerdlow SH, editor. Lyon, France: International agency for research on cancer; 2008 Sep 20.

29. Dong CD, Chen CW, Chen YC, Chen HH, Lee JS, Lin CH. Polystyrene microplastic particles: In vitro pulmonary toxicity assessment. J Hazard Mater. 2020 Mar 5;385:121575.

30. Xu M, Halimu G, Zhang Q, Song Y, Fu X, Li Y, et al. Internalization and toxicity: A preliminary study of effects of nanoplastic particles on human lung epithelial cell. Sci Total Environ. 2019 Dec 1;694:133794.

31. Amato-Lourenço LF, Carvalho-Oliveira R, Júnior GR, Dos Santos Galvão L, Ando RA, Mauad T. Presence of airborne microplastics in human lung tissue. J Hazard Mater. 2021 Aug 15;416:126124.

32. Jenner LC, Rotchell JM, Bennett RT, Cowen M, Tentzeris V, Sadofsky LR. Detection of microplastics in human lung tissue using μFTIR spectroscopy. Sci Total Environ. 2022 Jul 20; 831:154907.

33. Zhao Q, Zhu L, Weng J, Jin Z, Cao Y, Jiang H, et al. Detection and characterization of microplastics in the human testis and semen. Sci Total Environ. 2023 Jun 15;877:162713.

34. Ibrahim YS, Tuan Anuar S, Azmi AA, Wan Mohd Khalik WMA, Lehata S, Hamzah SR, et al. Detection of microplastics in human colectomy specimens. JGH Open. 2020 Nov 21;5(1):116–21.

35. Banerjee A, Shelver WL. Micro-and nanoplastic induced cellular toxicity in mammals: A review. Science of the Total Environment. 2021;755:142518.

36. Hwang J, Choi D, Han S, Jung SY, Choi J, Hong J. Potential toxicity of polystyrene microplastic particles. Sci Rep. 2020 Apr 30;10(1):7391.

37. Cao J, Xu R, Wang F, Geng Y, Xu T, Zhu M, et al. Polyethylene microplastics trigger cell apoptosis and inflammation via inducing oxidative stress and activation of the NLRP3 inflammasome in carp gills. Fish Shellfish Immunol. 2023 Jan;132:108470.

38. Isaacson PG. Mucosa-associated lymphoid tissue lymphoma. Semin Hematol. 1999 Apr;36(2):139–47.

39. Deng Y, Zhang Y, Lemos B, Ren H. Tissue accumulation of microplastics in mice and biomarker responses suggest widespread health risks of exposure. Sci Rep. 2017 Apr 24;7:46687.

40. Hirt N, Body-Malapel M. Immunotoxicity and intestinal effects of nano- and microplastics: a review of the literature. Part Fibre Toxicol. 2020 Nov 12;17(1):57.

41. Yang W, Jannatun N, Zeng Y, Liu T, Zhang G, Chen C, et al. Impacts of microplastics on immunity. Front Toxicol. 2022 Sep 27;4:956885.

42. Xuan L, Xiao L, Huang R. The geno-toxicological impacts of microplastic (MP) exposure on health: mechanistic pathways and research trends from a Chinese perspective. Environ Sci Process Impacts. 2023 Jan 25;25(1):26–36.

43. Poma AM, Morciano P, Aloisi M. Beyond genetics: can micro and nanoplastics induce epigenetic and gene-expression modifications? Frontiers in Epigenetics and Epigenomics. 2023 Aug 21;1:1241583.

44. Wade MJ, Bucci K, Rochman CM, Meek MH. Microplastic exposure is associated with epigenomic effects in the model organism Pimephales promelas (fathead minnow). J Hered. 2025 Mar 1;116(2):113–25.

45. Campanale C, Massarelli C, Savino I, Locaputo V, Uricchio VF. A Detailed Review Study on Potential Effects of Microplastics and Additives of Concern on Human Health. Int J Environ Res Public Health. 2020 Feb 13;17(4):1212.

46. Ullah S, Ahmad S, Guo X, Ullah S, Ullah S, Nabi G, et al. A review of the endocrine disrupting effects of micro and nano plastic and their associated chemicals in mammals. Front Endocrinol (Lausanne). 2023 Jan 16;13:1084236.

47. Marfella R, Prattichizzo F, Sardu C, Fulgenzi G, Graciotti L, Spadoni T, et al. Microplastics and Nanoplastics in Atheromas and Cardiovascular Events. N Engl J Med. 2024 Mar 7;390(10):900–10.

48. Choi I, Lee S, Hong YK. The new era of the lymphatic system: no longer secondary to the blood vascular system. Cold Spring Harb Perspect Med. 2012 Apr;2(4):a006445.

49. Kopp HG, Avecilla ST, Hooper AT, Rafii S. The bone marrow vascular niche: home of HSC differentiation and mobilization. Physiology (Bethesda). 2005 Oct;20:349–56.

50. Bianchi C, Bianchi T. Malignant mesothelioma: global incidence and relationship with asbestos. Industrial health. 2007;45(3):379–87.

51. Geyer R, Jambeck JR, Law KL. Production, use, and fate of all plastics ever made. Sci Adv. 2017 Jul 19;3(7):e1700782.

52. Grandjean P. Implications of the precautionary principle for primary prevention and research. Annu Rev Public Health. 2004;25:199–223.