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FROM EXPOSURE TO ONCOGENESIS: THE ROLE OF MICROPLASTICS AND ASSOCIATED POLLUTANTS IN CANCER - A LITERATURE REVIEW

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ABSTRACT

Introduction and purpose: Microplastics (MPs) are defined as particles smaller than 5 mm. They are ubiquitous environmental pollutants. They enter the human body primarily through food, water and inhaled air. This paper focuses on a collection of scientific studies concerning the accumulation of MPs in human tissues and their impact on cancer development, also considering the role of MPs as carriers of known carcinogens.
Brief description of the state of knowledge: In recent years, MPs have attracted considerable scientific attention. Their effects on human health, including oncology, have begun to be investigated. This area remains poorly studied, although new publications are emerging rapidly. Systematic reviews specifically addressing the oncological consequences of MPs are also lacking. Therefore, we see the need to summarize the current state of knowledge in this aspect.
Summary (conclusions): Increased levels of polyethylene (PE), polypropylene (PP), polystyrene (PS) and polyvinyl chloride (PVC) have been detected in tumor tissues such as breast, colorectal, pancreatic, prostate, lung and cervical cancers. In vitro and in vivo studies show that MPs stimulate tumorigenesis by enhancing cell proliferation, epithelial-mesenchymal transition, migration and activation on oncogenic PI3K/AKT and MAPK/ERK pathways. Moreover, MPs can serve as vectors for carcinogens (for example, polycyclic aromatic hydrocarbons and bisphenols). We hope this review will help guide future research directions.

KEYWORDS
Microplastics, Carcinogens, Neoplasms, Tissue Distribution, Environmental Exposure, Environmental Pollutants

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Introduction

Microplastics (MPs) are defined as plastic fragments with diameters below 5 mm. They are ubiquitous in the human environment [1]. Globally, nearly 400 million tons of plastic are produced annually. Over time, plastic degrades into ever smaller particles that enter the environment and threaten human health [2].

MPs enter the human body via various ways. The main routes are food, water and inhaled air, but also can penetrate through skin contact [3]. Among these, the gastrointestinal route is the most significant [4, 5]. MPs have been detected, for example, in honey, seafood, drinking water, milk, soft drinks and beer [6–8].

Worldwide, MPs have been found in 83% of tap water samples [9]. On average, humans ingest between 0, 1 and 5 g of MPs via food (roughly the mass of a credit card) [10].

Advances in MP detection are owed to modern technologies. Currently used methods include Fourier-transform infrared spectroscopy (FTIR), mass spectrometry, scanning electron microscopy with energy-dispersive X-ray spectroscopy (SEM-EDX), Raman spectroscopy and thermal analysis techniques. These allow far more precise chemical identification compared to visual inspection [11].

Analyses of human fecal samples have revealed nine types of MPs, dominated by polypropylene and polyethylene terephthalate [12]. From the gastrointestinal tract, MPs can translocate into the bloodstream and then into tissues, where they accumulate [13–15]. MPs have been detected in blood and clotted blood, various bodily fluids, urine, semen and tissues including lung, kidney, liver, heart, prostate and placenta, as well as in newborn meconium [8, 13, 16].

Experimental studies have shown that MPs possess toxic properties. Observed effects include oxidative stress, immune system activation and inflammation [17–19]. Furthermore, harmful substances (including chemicals or toxins) can absorb onto the MP surface, increasing their toxicity [20].

As reported by the World Health Organization (WHO), cancer ranks among the top causesof death worldwide. Cancer develops in stages from genetic mutations that disrupt the cell cycle, leading to uncontrolled cell division and tumor formation, ultimately resulting in metastasis and suppressed antitumor immune responses [21]. MP may potentially promote each of these stages, a concern supported by growing evidence that MPs may increase cancer risk [22].

Objective

The objective of this review is to summarize the current state of knowledge on the presence of MPs in tumor tissues and their influence on oncogenic processes.

Material and Methods

For the purpose of searching scientific publications, the PubMed, Google Scholar and ScienceDirect databases were used with the search term”microplastics human”. Publications from 2020 to 2025 were considered. After reviewing abstracts, original open-access in English articles describing the association between MPs and cancer were selected.

Description of State of Knowledge

Presence of Microplastics in tumors

One study assessed MP content in tumor tissues from patients with various cancers identified 13 types of plastics, most frequently polyethylene (PE), polypropylene (PP) and PE-co-PP copolymer. The highest MPs concentration and diversity were found in blood and tumor tissues. Interestingly, they showed higher MP concentrations than adjacent non-tumor tissues [16].

Using FTIR, Raman and laser-direct infrared spectroscopy, MPs were detected in 40% of tumor tissue samples from colorectal, gastric, esophageal, pancreatic, lung and cervical cancers. PE, PS and PVC were most frequent, occasionally co-occurring [23].In colorectal cancer, MP levels were significantly higher than in control samples from healthy individuals [24]. A study on penile cancer found MPs in 85, 3% of patients, identifying nine polymer types sized 20-50 μm, averaging 6, 42 particles/g [25]. In prostate cancer, PE, PP and PVC were detected, while PS was found exclusively in the tumor tissue [26].

Fibers were the predominant form of MPs. In colorectal cancer they constituted 96, 1% of findings. These fibres were typically 0, 8-1, 6 mm long [27].

In cervical cancer, more advanced stages correlated with higher PP and PE concentrations. MP levels also correlated with patient age, BMI and dietary habits. Patients consuming take-away meals in plastic packaging and bottled water had elevated MP levels [16, 26, 28].

The key information on the presence of MPs in cancer has been summarized in Figure 1. These findings demonstrate the diversity and penetrative ability of MPs in the human body and their correlation with cancer presence. More studies are needed to analyze MP content across diverse tumor types.

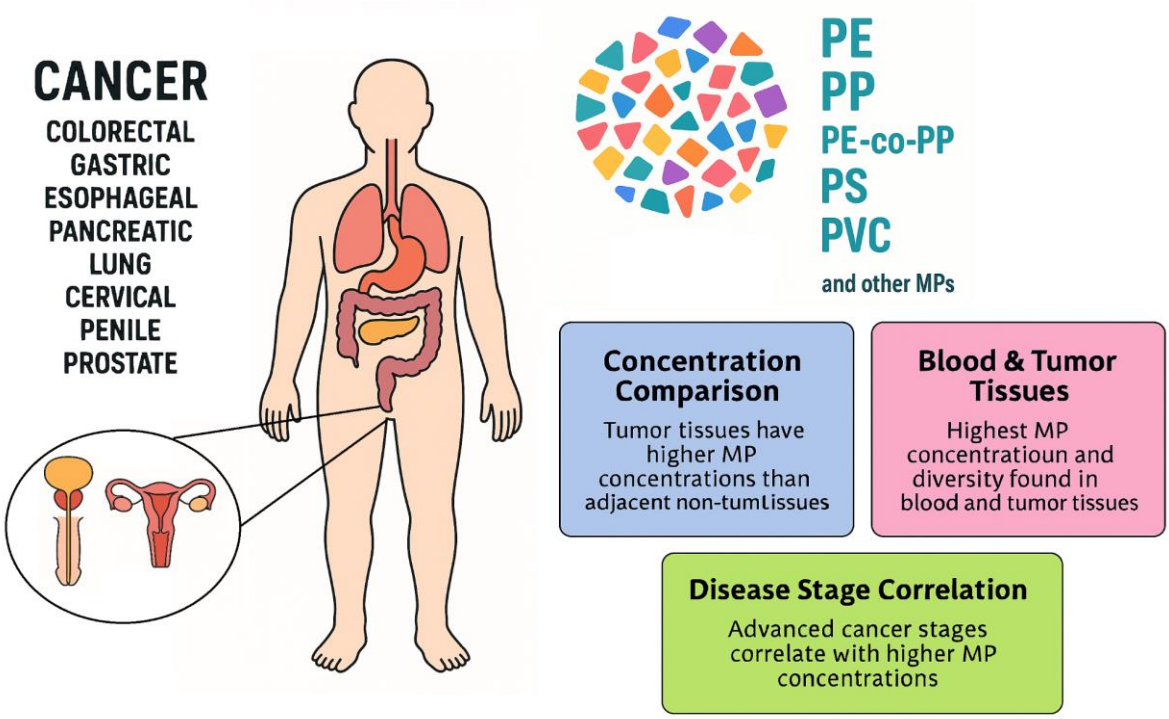

Fig. 1. *Summary of key information on MPs detected in cancers [16, 23-28]*

Microplastics' effects on tumor biology

In *In vitro* studies, the effects of irregular polypropylene particles (PPMP) on breast cancer cells were examined. The particles were selected to resemble those naturally found in the environment - irregular in shape and with sharp edges. Changes in the cell cycle were observed in the sample. The S/G2/M phases were prolonged. The expression of genes related to adenylate cyclase activity and cell-matrix adhesion was abnormal. Fatty-acid metabolism was disrupted, and there was also an increase in the secretion of the pro-inflammatory cytokine IL-6. Notably, only cancer cells were affected. Normal cells showed no changes [29]. Another study found that breast cancer cell endocytosed PS-MPs via anexin A2 binding, leading to mitophagy due to IL-17 exocytosis inhibition, damaging mitochondria [30]. In cutaneous squamous cell carcinoma, which

is known for its sensitivity to environmental stimuli, it was observed that plastic accumulates in a dose-dependent and time-dependent manner. Among the tests performed were the MTT assay, laser confocal microscopy, flow cytometry and Western blot. An increase in mitochondrial reactive oxygen species (ROS) was observed, which led to changes in mitochondrial membrane activity and the release of mtDNA. This ultimately activated the NLRP3 inflammasome and induced cell division. In healthy epidermal cells, MPs caused cell death [31].

Upon exposure to PS, colorectal cancer cells internalized its particles, which subsequently accumulated in the perinuclear region. After a short period of exposure, an increase in cell migration was observed, indicating a potential risk of metastasis [32]. Ps-MPs accumulated in a gastric cancer model. It was found that they induced an increase in N-cadherin expression and decreased in E-cadherin expression, promoting epithelial-mesenchymal transition. Additionally, the expression levels of CD44 and ASGR2 were elevated, which contributed to an unfavorable outcome by increasing resistance to multiple drugs [33]. Polyethylene terephalate (PET) in breast cancer cells demonstrated strong cytotoxicity towards breast cancer cells. They inhibited the PI3K/AKT and MAPK/ERK signaling pathways, which are HER2-dependent [21]. In glioma models, PE exposure increased proliferation, migration, and spheroid formation in a dose-dependent manner [34]. Mice were orally administered PE, which led to alterations in their gut microbiota, creating conditions more favorable for tumor development. The immune response was weakened, promoting the growth of colorectal cancer in situ [35]. Chronic PS nanoplastic exposure sensitized cells to oxidative stress, inducing an oncogenic EMT phenotype. The Bhas 42 assay revealed that PET nanoparticles promoted the early stages of tumor development, whereas the bioplastic PLA exhibited a lower carcinogenic potential [36, 37]. In an ovarian cancer model, PSNPLs reduced cell viability *in vitro* but enhanced tumor growth *in vivo* in mice. Gene expression related to immune respons and thrombomodulin was altered, leading to changes in the tumor microenvironment. The tumors were found to have doubled in both mass and volume compared to the control group [3].

Mice injected parenterally with MPs plus lipopolysaccharide (LPS) formed in liver tumor-like structures within two weeks, with overexpression of cancer markers and chemical carcinogenesis genes (SALL2) [38].

A microfluidic model integrating renal, endothelial and testicular cells to closely mimic physiological conditions. The system was exposed to PS nanoparticles for 24 hours. These particles were internalized by the cells, leading to structural alterations and disruption of glucose metabolism. Activation of oncogenic signaling pathways, including MAPK and PI3K/AKT, was also observed [39].

In both *in vitro* (esophageal cells) and *in vivo* (mice) models, the effects of PVC exposure were investigated. It was observed that PVC induced oxidative stress, leading to genetic material damage and impaired DNA repair. The expression of BRCA2 and GRB2 genes was downregulated. Inflammation was triggered by the presence of extracellular DNA. Findings from the in vivo models confirmed these results, showing that the effect persisted on a larger scale [40].

Human colorectal cells were subjected to both short- and long-term exposure to PS. It was observed that they internalized plastic particles, which induced oxidative stress and altered metabolic pathways. Glycolysis, the Krebs cycle, and the pentose phosphate pathway were notably upregulated. The metabolic profile resembled that seen after exposure to the carcinogen azoxymethane (AOM). Further analysis revealed epigenetic changes and increased expression of genes related to tumorigenesis and antioxidant defense [41].

In a murine model of colorectal cancer, the effects of prolonged PS exposure were examined. Mice exposed to MP experienced greater weight loss, more severe genetic damage and an increased number of tumor formations. Levels of ROS, as well as the expression of cancer and mitochondrial stress markers, were elevated. Additionally, the exposed mice exhibited an altered gut microbiome [42].

Microplastics as vectors for carcinogenic substances

The ability of PP, PS and PE to absorb phenanthrene (PHE) and its derivatives was investigated. It was found that these combinations exhibited very high bioavailability in the gastrointestinal tract, ranging from 53 % to 90%. Moreover, the calculated lifetime cancer risk exceeded the acceptable threshold set by the United States Environmental Protection Agency [22].

Over one thousand plastic-related chemical compounds were analyzed in silico to evaluate their impact on detoxification metabolism. The analysis showed that some of these compounds interfere with the cellular defense mechanisms by binding to transporters responsible for removing toxins [43].

Marine samples were analyzed for the presence of MPs. A correlation was found between their presence and polycyclic aromatic hydrocarbons (PAHs). The results suggest that plastics can act as carriers and accumulators of carcinogenic substances [44]. It has been shown that PAHs, which can be transported by MPs,

exhibit a strong affinity for the estrogen receptor alpha. This poses a potential risk of the development of breast cancer [45].

MPs derived from electronic waste were examined to assess their ability to bind PAHs in aquatic environments. Maximum saturation was reached in under one hour and PAH concentrations exceeded permissible limits by a factor one thousand. The calculated lifetime cancer risk surpassed the acceptable threshold established by the United States Environmental Protection Agency [46].

Plasticizers are compounds commonly added to plastics to impart desired properties. The effects of frequently used plasticizers, including bisphenols such as bisphenol A (BPA) and its analogues, were studied in breast cancer cell cultures. The results revealed altered gene expression, disrupted cell cycle regulation and enhanced tumor invasion, angiogenesis and metastasis [47, 48]. The role of MPs as vectors for carcinogenic substances has been presented in Figure 2.

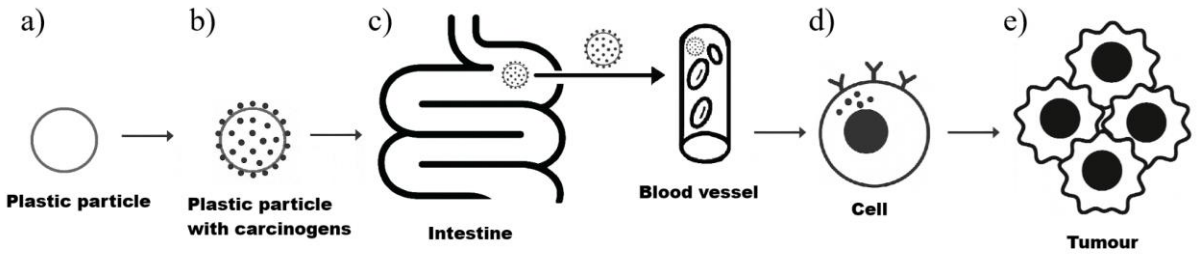

Fig. 2. *The role of MPs as vectors for carcinogenic substances.*
a) Plastic particles for example: PP, PS or PE; b) Adhesion and accumulation of carcinogens (for example PAHs, bisphenols, plasticizers) on the surface of MPs; c) Facilitated absorption of carcinogens from the gastrointestinal tract into the bloodstream; d) Carcinogens stimulate tumorigenesis in the cells; e) Tumor development [22, 43-48]

Discussion

Scientific interest in MP as a threat to human health has emerged relatively. The vast majority of publications on this topic have been published within the last decade. This review compiles up-to-date data on the presence of MPs in tumor tissues and their impact on oncogenic processes. We have shown that MPs negatively influence tumor development. Moreover, MPs can also act as carriers of carcinogenic compounds.

To date, the most well-documented harmful effects of MPs in humans involve the reproductive system, pregnancy outcome, the gastrointestinal tract and the respiratory system. In contrast, their potential role in oncology - a complex and broad field - remains poorly understood. Given that cancer is among the leading causes of death worldwide and that deepening our understanding of MPs' impact could help reduce incidence and improve prognosis, the current state of knowledge on the MP-cancer relationship should be seen as merely the tip of the iceberg - one that requires further exploration.

The good news is the advancement of technology, which is becoming increasingly precise and widely accessible. This enables improved detection of particles at the micro- and nanoscale, as well as the observation of subtle genetic and metabolic changes.

Future research should aim to determine the quantities and types of MPs present in tumors and clarify their biochemical interactions with oncogenic processes at the cellular level. One particularly intriguing observation is that MP concentrations in tumors are often higher than in the surrounding tissues. A thorough analysis of the mechanisms underlying biological barrier penetration, transport and accumulation is urgently needed. Most studies focus on the individual effects of specific polymers, whereas in reality, humans are exposed to mixtures of various MPs and their associated contaminants. Therefore, research into the combined effects and interactions between these substances is essential. We believe that this review will help raise awareness of the hidden, long-term risks associated with widespread presence of plastics and support efforts aimed at reducing their environmental burden.

Conclusions

The evidence presented above indicates that MPs pose a threat to human health. The most commonly produced polymers include PE, PP, PVC and PS. MPs are most frequently found in fibrous form, with sizes ranging from the micrometer to even the nanometer scale.

Plastic particles can enter the human body through multiple routes, primarily via food, but also through inhaled air and skin contact. Once inside, they are distributed throughout nearly all tissues and compartments of the body. There appears to be no organ that is completely free from their presence. Its presence has been confirmed in various types of cancer, including breast, cervical, esophageal, gastric, colorectal, pancreatic, lung, prostate and skin cancers. What is both concerning and intriguing is that tumor tissues contained higher concentrations of plastic than the adjacent normal tissues. Both *in vitro* and *in vivo* studies have shown that MPs stimulate all stages of carcinogenesis. They disrupt the cell cycle, induce the formation of ROS, suppress defence mechanisms, promote cell proliferation, epithelial-mesenchymal transition, migration and metastasis. Mixtures of polymers may be even more harmful. Moreover, MPs can function as vectors for various substances absorbed on their surfaces. Some of these are proven carcinogens, such as PAHs. This combination results in high bioavailability and may lead to concentrations that significantly exceed acceptable safety thresholds.

The current state of knowledge about MPs remains insufficient and further extensive *in vitro* and *in vivo* studies using advanced techniques are needed. Population-based studies are also necessary to assess the accumulation of MPs in the human body. Monitoring systems for environmental MP levels, along with actions focused on reducing their release into the environment, will help to minimize human exposure to carcinogenic substances.

Conflicts of Interest: No conflicts of interest to declare.

REFERENCES

1. Harms, I. K., Diekötter, T., Troegel, S., & Lenz, M. (2021). Amount, distribution and composition of large microplastics in typical agricultural soils in Northern Germany. Science of the Total Environment, 758, 143615.
2. Phuong, N. N., Zalouk-Vergnoux, A., Poirier, L., Kamari, A., Châtel, A., Mouneyrac, C., et al. (2016). Is there any consistency between the microplastics found in the field and those used in laboratory experiments? Environmental Pollution, 211, 111–123.
3. Chen, G., Shan, H., Xiong, S., Zhao, Y., van Gestel, C. A. M., Qiu, H., et al. (2024). Polystyrene nanoparticle exposure accelerates ovarian cancer development in mice by altering the tumor microenvironment. Science of the Total Environment, 906, 167592.
4. Zarus, G. M., Muianga, C., Hunter, C. M., & Pappas, R. S. (2021). A review of data for quantifying human exposures to micro and nanoplastics and potential health risks. Science of the Total Environment, 756, 144010.
5. Xu, J. L., Lin, X., Wang, J. J., & Gowen, A. A. (2022). A review of potential human health impacts of micro- and nanoplastics exposure. Science of the Total Environment, 851, 158111.
6. Barboza, L. G. A., Lopes, C., Oliveira, P., Bessa, F., Otero, V., Henriques, B., et al. (2020). Microplastics in wild fish from North East Atlantic Ocean and its potential for causing neurotoxic effects, lipid oxidative damage, and human health risks associated with ingestion exposure. Science of the Total Environment, 717, 134625.
7. Shruti, V. C., Pérez-Guevara, F., Elizalde-Martínez, I., & Kutralam-Muniasamy, G. (2020). First study of its kind on the microplastic contamination of soft drinks, cold tea and energy drinks — Future research and environmental considerations. Science of the Total Environment, 726, 138580.
8. Xu, H., Dong, C., Yu, Z., Hu, Z., Yu, J., Ma, D., et al. (2024). First identification of microplastics in human uterine fibroids and myometrium. Environmental Pollution, 360, 124632.
9. The Lancet Planetary Health. (2017). Microplastics and human health—an urgent problem. The Lancet Planetary Health, 1(7), e254.
10. Senathirajah, K., Attwood, S., Bhagwat, G., Carbery, M., Wilson, S., & Palanisami, T. (2021). Estimation of the mass of microplastics ingested – A pivotal first step towards human health risk assessment. Journal of Hazardous Materials, 404, 124004.
11. Huang, Z., Hu, B., & Wang, H. (2023). Analytical methods for microplastics in the environment: A review. Environmental Chemistry Letters, 21(1), 383–401.
12. Schwabl, P., Köppel, S., Königshofer, P., Bucsics, T., Trauner, M., Reiberger, T., et al. (2019). Detection of various microplastics in human stool. Annals of Internal Medicine, 171(7), 453–457.
13. Wu, H., Liu, Q., Yang, N., & Xu, S. (2023). Polystyrene-microplastics and DEHP co-exposure induced DNA damage, cell cycle arrest and necroptosis of ovarian granulosa cells in mice by promoting ROS production. Science of the Total Environment, 871, 161962.

14. Qin, X., Cao, M., Peng, T., Shan, H., Lian, W., Yu, Y., et al. (2024). Features, potential invasion pathways, and reproductive health risks of microplastics detected in human uterus. Environmental Science & Technology, 58(24), 10482–10493.
15. Jin, Y., Lu, L., Tu, W., Luo, T., & Fu, Z. (2019). Impacts of polystyrene microplastic on the gut barrier, microbiota and metabolism of mice. Science of the Total Environment, 649, 308–317.
16. Xu, H., Dong, C., Yu, Z., Ozaki, Y., Hu, Z., Zhang, B., et al. (2024). Detection and analysis of microplastics in tissues and blood of human cervical cancer patients. Environmental Research, 259, 119498.
17. Persiani, E., Cecchettini, A., Ceccherini, E., Gisone, I., Morales, M. A., & Vozzi, F. (2023). Microplastics: A matter of the heart (and vascular system). Biomedicines, 11(2), 264.
18. Çetin, M., Demirkaya Miloglu, F., Kilic Baygutalp, N., Ceylan, O., Yildirim, S., Eser, G., et al. (2023). Higher number of microplastics in tumoral colon tissues from patients with colorectal adenocarcinoma. Environmental Chemistry Letters, 21(2), 639–646.
19. Landrigan, P. J., Stegeman, J. J., Fleming, L. E., Allemand, D., Anderson, D. M., Backer, L. C., et al. (2020). Human health and ocean pollution. Annals of Global Health, 86(1), 151.
20. Koelmans, A. A., Bakir, A., Burton, G. A., & Janssen, C. R. (2016). Microplastic as a vector for chemicals in the aquatic environment: Critical review and model-supported reinterpretation of empirical studies. Environmental Science & Technology, 50(7), 3315–3326.
21. Cui, L., Digiacomo, L., Xiao, S., Wang, J., Amici, A., Pozzi, D., et al. (2023). Insights into the effect of polyethylene terephthalate (PET) microplastics on HER2 signaling pathways. Toxicology In Vitro, 91, 105632.
22. Hu, X., Yu, Q., Gatheru Waigi, M., Ling, W., Qin, C., Wang, J., et al. (2022). Microplastics-sorbed phenanthrene and its derivatives are highly bioaccessible and may induce human cancer risks. Environment International, 168, 107459.
23. Zhao, J., Zhang, H., Shi, L., Jia, Y., & Sheng, H. (2024). Detection and quantification of microplastics in various types of human tumor tissues. Ecotoxicology and Environmental Safety, 283, 116818.
24. Çetin, M., MiLoğlu, F. D., Baygutalp, N. K., Ceylan, O., Yildirim, S., Eser, G., et al. (2022). Detection of microplastics in patients with colorectal adenocarcinoma using various techniques [Manuscript in review]. Research Square. https://www.researchsquare.com/article/rs-1315103/v1
25. Wang, M., Liu, Q., Zhang, X., Jiang, H., & Zhang, X. (2025). Identification and analysis of microplastics in human penile cancer tissues. Science of the Total Environment, 969, 178815.
26. Deng, C., Zhu, J., Fang, Z., Yang, Y., Zhao, Q., Zhang, Z., et al. (2024). Identification and analysis of microplastics in para-tumor and tumor of human prostate. eBioMedicine, 108, 105360.
27. Ibrahim, Y. S., Tuan Anuar, S., Azmi, A. A., Wan Mohd Khalik, W. M. A., Lehata, S., Hamzah, S. R., et al. (2021). Detection of microplastics in human colectomy specimens. JGH Open, 5(1), 116–121.
28. Xu, H., Dong, C., Xiang, T., Shentu, X., Yu, Z., Xu, J., et al. (2025). Microplastic changes during the development of cervical cancer and its effects on the metabolomic profiles of cancer tissues. Journal of Hazardous Materials, 483, 136656.
29. Park, J. H., Hong, S., Kim, O. H., Kim, C. H., Kim, J., Kim, J. W., et al. (2023). Polypropylene microplastics promote metastatic features in human breast cancer. Scientific Reports, 13, 6252.
30. Tian, Z., Ding, B., Guo, Y., Zhou, J., Jiang, S., Lu, J., et al. (2025). Microplastics accumulated in breast cancer patients lead to mitophagy via ANXA2-mediated endocytosis and IL-17 signaling pathway. Environmental Pollution, 364, 125321.
31. Wang, Y., Xu, X., & Jiang, G. (2023). Microplastics exposure promotes the proliferation of skin cancer cells but inhibits the growth of normal skin cells by regulating the inflammatory process. Ecotoxicology and Environmental Safety, 267, 115636.
32. Brynzak-Schreiber, E., Schögl, E., Bapp, C., Cseh, K., Kopatz, V., Jakupec, M. A., et al. (2024). Microplastics role in cell migration and distribution during cancer cell division. Chemosphere, 353, 141463.
33. Kim, H., Zaheer, J., Choi, E. J., & Kim, J. S. (2022). Enhanced ASGR2 by microplastic exposure leads to resistance to therapy in gastric cancer. Theranostics, 12(7), 3217–3236.
34. Rafazi, P., Bagheri, Z., Haghi-Aminjan, H., Rahimifard, M., & Ahvaraki, A. (2024). Long-term exposure of human U87 glioblastoma cells to polyethylene microplastics: Investigating the potential cancer progression. Toxicology Reports, 13, 101757.
35. Yang, Q., Dai, H., Wang, B., Xu, J., Zhang, Y., Chen, Y., et al. (2023). Nanoplastics shape adaptive anticancer immunity in the colon in mice. Nano Letters, 23(8), 3516–3523.
36. Barguilla, I., Domenech, J., Ballesteros, S., Rubio, L., Marcos, R., & Hernández, A. (2022). Long-term exposure to nanoplastics alters molecular and functional traits related to the carcinogenic process. Journal of Hazardous Materials, 438, 129470.
37. Domenech, J., Villacorta, A., Ferrer, J. F., Llorens-Chiralt, R., Marcos, R., Hernández, A., et al. (2024). In vitro cell-transforming potential of secondary polyethylene terephthalate and polylactic acid nanoplastics. Journal of Hazardous Materials, 469, 134030.

38. Huang, H., Hou, J., Yu, C., Wei, F., & Xi, B. (2024). Microplastics exacerbate tissue damage and promote carcinogenesis following liver infection in mice. Ecotoxicology and Environmental Safety, 286, 117217.
39. Xiao, M., Li, X., Zhang, X., Duan, X., Lin, H., Liu, S., et al. (2023). Assessment of cancer-related signaling pathways in responses to polystyrene nanoplastics via a kidney-testis microfluidic platform (KTP). Science of the Total Environment, 857(Pt 1), 159306.
40. Huang, Y., Li, X., Xu, S., Zu, D., Liu, H., He, H., et al. (2025). Polyvinyl chloride nanoplastics suppress homology-directed repair and promote oxidative stress to induce esophageal epithelial cellular senescence and cGAS-STING-mediated inflammation. Free Radical Biology & Medicine, 226, 288–301.
41. Bonanomi, M., Salmistraro, N., Porro, D., Pinsino, A., Colangelo, A. M., & Gaglio, D. (2022). Polystyrene micro and nano-particles induce metabolic rewiring in normal human colon cells: A risk factor for human health. Chemosphere, 303(Pt 1), 134947.
42. Tian, S., Li, R., Li, J., & Zou, J. (2025). Polystyrene nanoplastics promote colitis-associated cancer by disrupting lipid metabolism and inducing DNA damage. Environment International, 195, 109258.
43. Rosellini, M., Turunen, P., & Efferth, T. (2023). Impact of plastic-related compounds on P-glycoprotein and breast cancer resistance protein in vitro. Molecules, 28(6), 2710.
44. Sui, Q., Yang, X., Sun, X., Zhu, L., Zhao, X., Feng, Z., et al. (2024). Bioaccumulation of polycyclic aromatic hydrocarbons and their human health risks depend on the characteristics of microplastics in marine organisms of Sanggou Bay, China. Journal of Hazardous Materials, 473, 134622.
45. Böckers, M., Paul, N. W., & Efferth, T. (2020). Indeno[1,2,3-cd]pyrene and picene mediate actions via estrogen receptor α signaling pathway in in vitro cell systems, altering gene expression. Toxicology and Applied Pharmacology, 396, 114995.
46. Sharma, M. D., Elanjickal, A. I., Mankar, J. S., & Krupadam, R. J. (2020). Assessment of cancer risk of microplastics enriched with polycyclic aromatic hydrocarbons. Journal of Hazardous Materials, 398, 122994.
47. Böckers, M., Paul, N. W., & Efferth, T. (2020). Bisphenolic compounds alter gene expression in MCF-7 cells through interaction with estrogen receptor α. Toxicology and Applied Pharmacology, 399, 115030.
48. Böckers, M., Paul, N. W., & Efferth, T. (2020). Organophosphate ester tri-o-cresyl phosphate interacts with estrogen receptor α in MCF-7 breast cancer cells promoting cancer growth. Toxicology and Applied Pharmacology, 395, 114977.