Australian Government
Cancer Australia

2 March 2026

Committee Secretary
Senate Standing Committees on Community Affairs
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Dear Committee Secretary

Submission – inquiry into the impact of microplastics and other toxics on human health

Cancer Australia was established by the Australian Government in 2006 to benefit all Australians affected by cancer, and their families and carers. Cancer Australia aims to reduce the impact of cancer, address disparities and improve outcomes for people affected by cancer by leading and coordinating national, evidence-based interventions across the continuum of care.

Cancer is the leading cause of death and burden of disease in Australia, with one in two people being diagnosed within their lifetime. Australia's survival rates for most cancers are among the best in the world, however significant disparities in cancer outcomes exist between specific population groups within Australia. The Australian Cancer Plan (the Plan), released in 2023, is a future-focused plan designed to improve cancer outcomes, particularly for those groups whose health outcomes are poorer. The Plan includes a focus on prevention and early detection of cancer and the application of technology, research and data to improve Australia's cancer outcomes.

This submission will respond to the following item under the inquiry Terms of Reference: *The impact of microplastics, toxics and forever chemicals on human health, with particular reference to: (d) links between endocrine disrupters and increased rates of cancer in young people, fertility issues, hormone dysregulation, respiratory diseases, inflammatory conditions and immune system dysfunction.*

Early-onset cancer is defined as cancer diagnosed beyond adolescence but before the age of 50 years of age (20-49 years). Since 2000, there has been an increase in incidence in a number of 'early-onset' cancers, including bowel (colorectal), thyroid, breast, prostate and kidney cancer, both in Australia and other parts of the world.

Cancer Australia is responding to this emerging issue via the following initiatives:

- An epidemiological report on trends in cancer incidence in people under 50 years to understand the impact of early-onset cancers in Australia.

- An evidence review of risk factors for early-onset cancers, to understand the potential reasons behind the increase, identify research gaps and interventions aimed at altering the trajectory of early-onset cancer rates in Australia.
- In July 2025, Cancer Australia launched the Cancer Australia Research Initiative (CARI), a new grant program aimed at addressing emerging needs in cancer research and including a commitment of $7 million over three years to fund research into early-onset cancers.
- Reviewing and updating evidence-based guidance for GPs to facilitate earlier diagnosis of people with signs and symptoms of cancer, including those under 50 years. These updates will be made to *Optimal Care Pathways Step 2* section, which focus on presentation, initial investigations and referral.

Early-onset cancers are likely caused by a complex interplay of several risk factors. These may include changes over recent decades in lifestyle, diet, obesity rates, environmental exposures, and other factors. Microplastics are of considerable scientific and public interest as a potential risk factor for cancer, including early-onset cancers such as colorectal cancer. However, more research is needed to better understand the specific drivers of this upwards trend in incidence of early-onset cancer.

The epidemiological report and the evidence review commissioned by Cancer Australia have both examined the potential link between microplastics and early-onset cancer. A summary of systematic reviews of microplastics in relation to cancer (*see Attachment A*) found that these studies consistently described similar potential mechanisms, suggesting convergence across the evidence base. Proposed mechanisms included DNA damaging potential which may contribute to genomic instability, plausible biological links between microplastic exposure and the development of metabolic syndrome and colorectal carcinogenesis, and 'Trojan horse' effects (i.e. microplastics as carriers of other toxins). However, microplastics as an emerging potential risk factor for early-onset cancer has not been reported by the International Agency for Research on Cancer, and more evidence is needed from longitudinal studies to establish whether these exposures are in fact causally related to cancer.

Cancer Australia will continue to monitor the emerging research evidence in relation to the increasing incidence of early-onset cancers.

Yours sincerely

Professor Dorothy Keefe PSM MD

Chief Executive Officer

Attachment A. A summary of systematic reviews on microplastics in relation to cancer.

Paper	Topic	Key findings	Study design summary	Strengths and limitations of study
Albukhari et. al. (2025)	Existing human, animal, and in vitro evidence on the dual effects of microplastic exposure in metabolic dysregulation and the molecular pathways associated with colorectal carcinogenesis	The findings suggest a biologically plausible connection between microplastic exposure and the development of both metabolic syndrome and colorectal carcinogenesis pathway, rather than a direct clinical association with early onset colorectal cancer (i.e. correlations rather than causative links) Human studies: exposure to microplastics – especially BPA and phthalates – correlated with an increased risk of metabolic syndrome features (e.g., central obesity, insulin resistance, dyslipidaemia) Animal studies and *in vitro* studies: exposure to microplastics contributed to heightened inflammatory responses, alterations in gut microbiota composition, and dysfunction of the epithelial barrier Animal studies and *in vitro* studies: Chronic exposure led to colonic inflammation and an elevation in tumorigenic markers, such as β-catenin (a key oncogenic protein in the *Wnt* signalling pathway) and COX-2 (an inflammatory enzyme implicated in tumour progression), suggesting a potential role in colorectal tumour development	Systematic review of 45 studies (18 human observational studies [mostly cross-sectional studies], 17 *in vivo* animal studies, 10 *in vitro* studies) 4 databases (PubMed, Scopus, Web of Science, EMBASE) + grey literature + relevant reports from international organisations (e.g., WHO, UNEP): final search performed in Apr. 2025	<u>Strengths</u> Followed PRISMA 2020 guidelines, including risk of bias assessment Integrated evidence from diverse study designs (human participants, animal models, and in vitro results) <u>Limitations</u> Considerable heterogeneity regarding exposure types and outcome definitions Potential selection bias and residual confounding from cross-sectional studies: correlations rather than causative links Publication bias (e.g. favourable oxidative or inflammatory effects are more likely to be published)
Tan et. al. (2025)	Detection, characterisation, and potential health implications of microplastics in	Microplastics are ubiquitously present in human gastrointestinal tract biospecimens (faeces, colonic tissues, and liver samples), with higher concentrations found in faecal samples from gastrointestinal diseases patients (e.g., inflammatory bowel disease and CRC).	Systematic review of 13 studies (description of included studies not described) Search was performed in July 2023 from Scopus and PubMed	<u>Strengths</u> Followed PRISMA 2020 guidelines, including risk of bias assessment <u>Limitations</u> Differences in microplastics prevalence across sample types are potentially

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	the human gastrointestinal tract	Microplastics exposure is influenced by dietary habits, geographical location, and lifestyle choices		related to limitations in current detection and analysis methods
Alimba et. al. (2025)	DNA damage and genotoxicity, in turn associated with health outcomes including cancer	DNA damaging potentials of microplastics and nanoplastics may contribute to increased genomic instability, the hallmark of genetic-related diseases including cancer Mechanisms of microplastics and nanoplastics-induced DNA damage: ○ Generate free radicals and oxidative stress (i.e. generate too many unstable atoms or molecules and overproduce reactive oxygen and nitrogen species, and overwhelm the body's antioxidant defences) ○ Trigger inflammation in the body, causing the release of cytokine like TNF-α ○ Damage cellular proteins involved in cell division and signalling, ultimately damage DNA stability ○ Somatic and germ-line cells are susceptible to the genotoxic effects	Systematic review of 141 studies (in situ, *in vivo* or/and *in vitro* models) 5 databases (Scopus, PubMed, Science Direct, Google Scholar, and Web of Science) searched to May 2025	<u>Strengths</u> Followed PRISMA 2020 guidelines Articles not conducted following standard experimental designs were eliminated
Wen et. al. (2025)	Association between microplastics and nanoplastics exposure and CRC carginogenesis	Microplastics and nanoplastics enter the body through gastrointestinal ingestion, respiratory inhalation, and dermal contact When ingested, microplastics and nanoplastics translocate across biological barriers to induce DNA damage and oxidative stress through reactive oxygen species overproduction Microplastics and nanoplastics disrupt intestinal barrier function by reducing tight junction proteins, trigger chronic inflammation via pro-inflammatory cytokines, and cause gut microbiota dysbiosis	Systematic review of 87 studies PubMed, Scopus, Google Scholar, Science Direct, ProQuest, EMBASE, Semantic Scholar, and Web of Science from 2004 to 2024	<u>Strengths</u> Followed PRISMA 2020 guidelines <u>Limitations</u> Did not specify details of the included studies (e.g. details of included studies, and data extraction and synthesis) Risk of bias assessment, quality appraisal not performed Resembles a narrative review supplemented with a systematic search

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		Microplastics and nanoplastics act as "Trojan horses", carrying absorbed toxic chemicals (e.g., bisphenol A) and pathogens that heighten cytotoxic effects and trigger carcinogenic pathways		
Liu et. al (2025)	An evaluation of the association between microplastic exposure and of incidence and mortality of all cancers in cohort and case-control studies	Exposure to microplastic pollutants was significantly associated with overall cancer incidence for case-control studies (OR: 1.10, 95% CI: 1.01-1.19), but not for cohort studies (OR: 1.04, 95% CI: 0.96-1.12). There was no significant association with overall cancer mortality in cohort studies (OR: 1.06, 95% CI: 0.96-1.22) There were significant associations in subgroup for phthalates and bisphenol A with cancer incidence	Systematic review and meta-analysis of 43 studies (32 case-control studies, 11 cohort studies) Three databases (PubMed, Embase, the Cochrane Library) searched to 30/6/24 Quality assessment and publication bias assessment performed Pooled analysis performed Dose-response analysis performed for bisphenol A	<u>Strengths</u> Meta-analysis of a large number of studies, examination of multiple types of microplastics as exposures, quality assessment and publication bias assessment performed, dose-response analyses performed for bisphenol A <u>Limitations</u> Majority of included studies were case-control studies which are vulnerable to recall bias, significant heterogeneity in results
Mashayekhi-Sardoo et. al. (2025)	A summary of studies examining microplastic infiltration in colorectal tissue samples in persons with colorectal adenocarcinoma	In two cross-sectional studies (n = 11 and n = 50), microplastic particles were detected in all colorectal adenocarcinoma tumour tissue samples, while in another cross-sectional study (n = 10), microplastic particles were detected in 50% of colorectal adenocarcinoma tumour tissue samples In a case-control study (n = 31; 16 cases, 15 controls), a higher number of microplastic particles were detected in colorectal adenocarcinoma tissue samples in cases compared that in controls, and also compared to that in non-tumour tissue in cases	Systematic review of 4 studies (3 cross-sectional studies, 1 case-control study) Five databases (PubMed, ISI Web of Science, Embase, Scopus, Google Scholar) searched to 30/9/24 Quality assessment performed	<u>Strengths</u> Quality assessment performed, low risk of bias in all included studies <u>Limitations</u> Low number of studies included, low sample sizes, cross-sectional and case-control study design more vulnerable to bias compared to cohort studies, significant clinical and methodological heterogeneity across studies
Baspakova et. al. (2025)	A review of the mechanisms of	A large number of mechanisms by which microplastics may impact of cancer cells were identified, including on the oxidative stress	Systematic review (number and type of included studies unclear)	<u>Strengths</u>

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	impact of microplastics on cancer cells	pathway in cancer cells, on cancer-related genes, on the response of the immune system to cancer, on inflammation pathways in cancer cells, and on mitochondrial function in cancer cells Microplastics may have both inhibitory and promotional effects on cancer cells	Two databases (Google Scholar, PubMed) searched from 2015 to 2024 (search performed on 10/14/24) Molecular docking analysis were performed using online software (an *in silico* analysis) Quality assessment not performed	A large number of mechanisms by which microplastics may impact cancer cells were identified <u>Limitations</u> Key details about the systematic review method and results were not clearly reported (the number of included studies and the characteristics of each study, and whether study selection and data extraction were performed in duplicate to minimise errors), quality assessment not performed, the use of *in silico* analysis
Chartres et. al. (2024)	A review of the effects of microplastic exposure on human health, including cancer	Included 3 human observational studies examining reproductive (n = 2; participants were pregnant women) and respiratory (n = 1; case control study) outcomes 28 animal studies examining reproductive (n = 11), respiratory (n = 7), and digestive (n = 10) outcomes. For reproductive outcomes (sperm quality) and digestive outcomes (immunosuppresion) the overall body evidence was "high" quality. Microplastic exposure is "suspected" to adversely impact them. For reproductive outcomes (female follicles and reproductive hormones), digestive outcomes (gross or microanatomic colon/small intestine effects, alters cell proliferation and cell death, and chronic inflammation), and respiratory outcomes (pulmonary function, lung injury, chronic inflammation, and oxidative stress) the overall body of evidence was "moderate" quality. Microplastic exposure is "suspected" to adversely impact them.	Rapid systematic review Searched 4 databases in July 2022 and April 2024 with no restriction on the date. Risk of bias was assessed. A review protocol that prespecified the review methods was published on Open Science Framework (OSF).	<u>Strengths</u> Rigorous methodology including risk of bias, PICO, search strategy, and data extraction <u>Limitations</u> No direct measure of microplastics on cancer outcomes. Using the ”key characteristics of carcinogens approach” – i.e. identify mechanisms indicative of cancer or reproductive toxicity. Based on animal studies, the authors conclude there is a suspected link to colon cancer.

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		Exposure to microplastics is "unclassifiable" for birth outcomes and gestational age in humans on the basis of the "low" and "very low" quality of the evidence. Microplastics are "suspected" to harm human reproductive, digestive, and respiratory health, with a suggested link to colon cancer.		
Kumar et. al. (2024)	A review of the impact of microplastic pollution on ecosystems including plant health, soil microorganisms, aquatic lives, and other animals	This article covered the distribution of microplastics in water, soil, food and potential routes of human exposure.	Google Scholar and the ISI Web of Knowledge databases were used to do a thorough literature assessment – 2010 to 2024 Journal publications, conference proceedings, poster presentations, and dissertations were included. All relevant information on (i) the method for detecting microplastic, (ii) the presence of microplastic and their routes of exposure, and (iii) effects on the environment has been included.	<u>Limitations</u> Not registered on PROSPERO – formal systematic review format not followed No study quality assessment conducted. No included studies evaluated the impact on human health outcomes. Only one included study was related to cancer: *Polystyrene nanoparticles with diameters of 25 and 70 nm… were found to dramatically alter cell viability, trigger the transcription of inflammatory genes, and alter the expression of proteins related to the cell cycle and pro-apoptosis*.
Symeonides et. al (2024)	An umbrella review of systematic reviews with meta-analyses examining the effect of plastic-associated chemicals on	52 systematic reviews were included, with data contributing 759 meta-analyses. Most meta-analyses (78%) were from reviews of moderate methodological quality. Across all the publications retrieved, there were no meta-analyses evaluating microplastics. Synthesised estimates of the effects of plastic-associated chemical exposure were identified for the following health outcome categories in humans: birth, child and adult reproductive, endocrine, child neurodevelopment, nutritional, circulatory, respiratory, skin-related and cancers.	Umbrella review of systematic reviews with meta-analyses. Focussed on microplastics, plasticisers, flame retardants, bisphenols, and per- and polyfluoroalkyl substances (PFAS) Epistemonikos and PubMed were searched for systematic reviews with meta-analyses, meta-analyses, and pooled analyses evaluating the association of	<u>Strengths</u> Registered on PROSPERO Comprehensive and rigorous approach, including multiple data extractors and quality assessment <u>Limitations</u> Primary studies evaluating cancer outcomes were in relation to polychlorinated biphenyls (PCBs) - a

Paper	Topic	Key findings	Study design summary	Strengths and limitations of study
	human health, including cancer		plastic polymers, particles (microplastics) or any of the selected groups of high-volume plastic-associated chemicals above, measured directly in human biospecimens, with human health outcomes. Search conducted in Sept 2020. No publication period specified. Review quality assessd using 'A MeaSurement Tool to Assess systematic Reviews' (AMSTAR) tool.	group of synthetic industrial chemicals. Not microplastics.
Cho et. al. (2021)	A review of methods for measuring human exposure to microplastics	No standard method exists for measuring human exposure to microplastics. Microplastic characteristics—including size, shape, colour, and chemical composition—affect levels and pathways of human exposure. Two main exposure routes were identified: Inhalation and Ingestion. Exposure through ingestion can occur via the food chain, individual food items, and mineral water, indicating multiple exposure scenarios. The only current human exposure marker is detection in faeces. Evidence remains limited, with few studies assessing the health effects of microplastic exposure in the general population. Observational studies on the human body or population have not been reported to date. The possibility of human exposure and health effects have been suggested indirectly through the	Rapid systematic review Articles published from 2000 to 2019 in PubMed, NDSL, KMBASE, and Cochrane databases 8 review articles and 13 original articles were selected for analysis. Risk-of-bias assessment of individual studies was performed using RoBANS-2.0,	<u>Limitations</u> No data extraction. No reporting on the risk of bias Most of the studies were not actual human studies nor health effect assessment studies that evaluated clear health outcomes Included studies on cancer were editorials

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		measurement of the microplastic content in ecosystems, foods, and household goods. There is a lack of clear evidence of human exposure and health effects.		

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