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Invisible Invaders: Making Sense of the Health Risks Posed by Microplastics and Nanoplastics

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Abstract

Plastic was supposed to make life easier, and in many ways it has. What nobody planned for is what happens once that plastic starts to fall apart. Microplastics and nanoplastics fragments ranging from a few millimetres down to a few billionths of a metre are now turning up in places no one expected to find them in bottled water, in the fish on our plates, and as several recent studies show inside our own bodies. Researchers have already recovered these particles from human blood, placental tissue, lung tissue, testis and semen, carotid artery plaque, and even brain tissue. People are exposed mainly by eating and drinking, by breathing indoor and outdoor air, and to a smaller degree through skin contact. Laboratory and animal work paints a fairly consistent picture that these particles can trigger oxidative stress, provoke inflammation, cross biological barriers most substances cannot cross because they often act as passengers for additives such as bisphenol A, phthalates and adsorbed pollutants which interfere with hormonal, immune, and metabolic signalling. What is missing is not evidence of biological plausibility but evidence of long-term consequence in real human populations, since most of what we know still comes from cells in a dish or animals in a cage rather than people followed over decades. This review pulls together what the human-relevant literature currently shows about exposure routes, organ-specific effects, and the research and policy gaps that still need to be closed, and closes with a look at where mitigation and regulation stand today.

Keywords: Microplastics, Nanoplastics, Human exposure, Long-term health effects, Policy actions

Introduction

Twenty years ago, the word "microplastic" did not really exist in the scientific vocabulary. Marine biologist Richard Thompson changed that in 2004, when he and his colleagues described tiny plastic fragments accumulating in European coastal sediments and gave the phenomenon a name [1]. Thompson later recalled that the finding "became a worldwide news story" within weeks, and that the obvious next question is this harmful? has driven two decades of research since [1]. Two decades on, that early curiosity looks almost quaint next to what has actually been found: plastic particles small enough to pass through cell membranes have been detected in human blood, lungs, reproductive organs, and brain tissue [2,4,6,9].

Microplastics are generally defined as particles smaller than 5 millimetres, while nanoplastics fall below 1 micrometre, small enough to behave less like debris and more like a chemical contaminant [7]. Some are manufactured deliberately at that scale, as "primary" microplastics used in cosmetics, industrial abrasives, and synthetic textiles. Far more originate as "secondary" fragments, chipped off larger plastic items by sunlight, mechanical wear, and weathering [25]. Either way, once particles reach this range of size, they are essentially impossible to remove from the environment, and they move freely between soil, water, air and the food chain.

What makes the last five years different from the two decades before them is the sheer volume of human tissue evidence. A 2022 Dutch study was the first to confirm plastic particles circulating in human blood, detecting them in roughly three-quarters of the healthy adults it tested [2]. Since then, similar particles have turned up in placental tissue [3], lung biopsies [4], testicular tissue and semen [8,9], arterial plaque removed during surgery [5], and, most recently, in brain tissue collected at autopsy [6]. None of this proves that microplastics cause disease. Correlation and contamination are genuine methodological headaches in this field, and some of these findings have drawn pointed scientific criticism [32]. But together they have moved the conversation from "are we exposed?" to "what is that exposure doing to us?". This review works through the exposure pathways, the organ systems where evidence is accumulating, and the considerable gaps that remain before that second question can be answered with confidence.

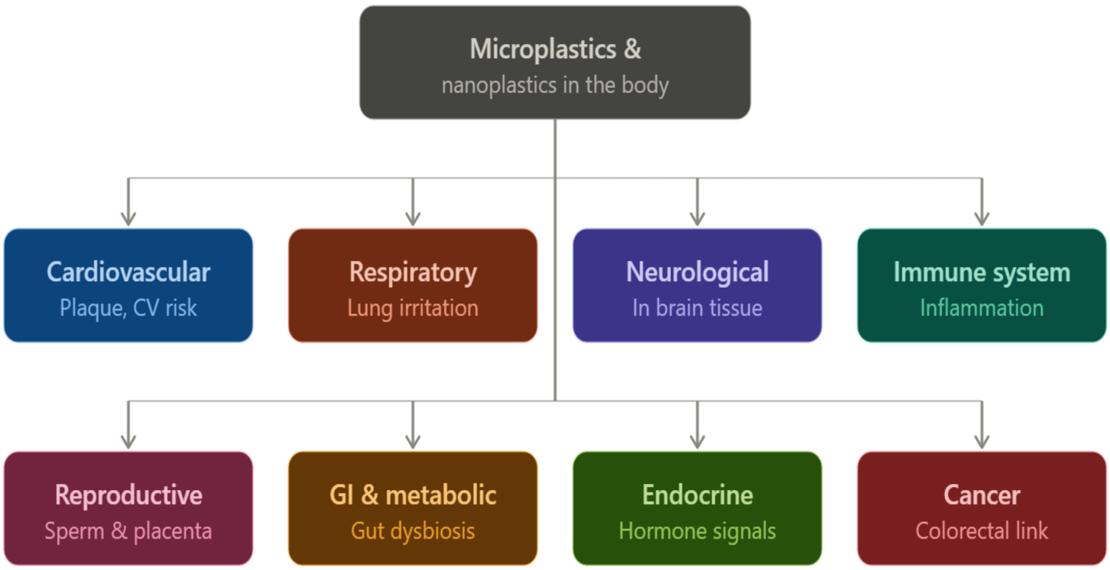
How Microplastics and Nanoplastics Get into the Body

Three routes account for essentially all known human exposure: ingestion, inhalation, and playing a much smaller role in skin contact [18,25]. Ingestion is probably the route people find most unsettling once they hear the numbers. A 2024 Columbia University team used a new Raman-scattering imaging technique sensitive enough to count individual nanoplastic particles, something older methods simply could not do, and found that a single litre of bottled water can contain on the order of 240,000 detectable plastic fragments, roughly 90 percent of them nanoplastics too small for previous studies to have picked up at all [7,33]. Seafood, table salt, honey, and even fresh produce have all tested positive for microplastic contamination in various studies, which is unsurprising given how thoroughly these particles have infiltrated soil and waterways.

Inhalation matters more than most people assume, particularly indoors, where synthetic carpets, upholstery, and clothing shed fibres continuously into household dust. Lung tissue removed during surgery has been shown to contain microplastic fragments and fibres, with higher concentrations detected in the lower lobes of the lung, a pattern the study's authors suggested may reflect gravitational settling of inhaled particles over time [4]. Dermal absorption remains the least significant of the three pathways for most people, since intact skin is a reasonably effective barrier, though there is some concern about nanoplastics in cosmetics and personal-care products penetrating compromised or damaged skin [18].

Exposure Route	Typical Sources	Representative Human Evidence
Ingestion	Bottled and tap water, seafood, salt, honey, food packaging	Up to ~240,000 particles/litre in bottled water, 90% nanoplastic [7,33]
Inhalation	Indoor dust, synthetic textiles, urban air, occupational settings	Microplastic fibres recovered from human lung biopsy tissue [4]
Dermal contact	Cosmetics, personal care products (minor route)	Limited direct human data; concern centres on compromised skin barriers [18]

Health Implications



Physical and Cellular Effects

At the most basic level, these particles cause trouble simply by being physically present where they should not be. Nanoplastics in particular are small enough to cross the intestinal lining and enter the bloodstream, from where they can reach essentially any organ [2,19]. Once lodged in tissue, they behave as any foreign material would: irritating surrounding cells, triggering low-grade inflammation, and in laboratory models, disrupting mitochondrial function and generating reactive oxygen species [16,20,24]. A recent narrative review focused specifically on inflammation and cellular ageing concluded that this oxidative burden is a fairly consistent finding across cell lines, organoids, and animal models, and that it plausibly links microplastic exposure to the same inflammatory pathways implicated in cardiovascular disease and other age-related conditions [24]. None of this is proof of harm at the exposure levels most people actually experience, but it does explain why researchers keep circling back to inflammation as the common thread running through nearly every organ system examined so far.

Chemical Toxicity

Plastic particles rarely travel alone. Manufacturing additives such as bisphenol A, phthalates, and flame retardants are chemically bound into the polymer and can leach out once inside the body, and many of these compounds are recognised endocrine disruptors [19,35]. On top of that, microplastics are hydrophobic and have a large surface area relative to their volume, which lets them act like a sponge for other environmental pollutants such as heavy metals, polychlorinated biphenyls, and polycyclic aromatic hydrocarbons, picked up from soil and water long before they're ever ingested [25]. This double burden, the plastic itself plus whatever it has picked up along the way is part of why toxicologists tend to treat microplastic exposure as much as a chemical problem as a physical one.

Immunological response

The immune system treats microplastics the way it treats any foreign particle when it notices them. A 2025 systematic review of nanoplastic-immune interactions pooled evidence from *in vitro* and animal studies and found a consistent pattern of oxidative stress, cytokine imbalance, and activation of pro-inflammatory signalling pathways following exposure [21]. The concern is less about a single acute reaction and more about what happens with chronic, low-level exposure, the kind most people actually experience. Several reviews link to persistent low-grade inflammation, a known contributor to cardiovascular disease, metabolic syndrome, and autoimmune conditions [19,21]. Whether repeated exposure eventually leaves the immune system hyper-reactive, under-reactive, or simply dysregulated is still an open question that the authors of these reviews are careful not to overstate.

Gastrointestinal and metabolic Effects

Because ingestion is the dominant exposure route, the gut has attracted a lot of research attention, and the human evidence here is more advanced than in most other organ systems. A quasi-experimental study found that people served hot food on disposable plastic tableware for one month showed measurable shifts in gut microbial composition compared with those served on non-disposable ware [15], and a cross-sectional study in preschool children found microplastic exposure inversely correlated with microbial diversity and reduced levels of certain probiotic bacterial taxa [36]. A broader synthesis of this literature reports that microplastic exposure tends to increase potentially pathogenic bacterial families while depleting genera associated with short-chain fatty acid production, changes that plausibly weaken the gut barrier and increase intestinal permeability, the mechanism behind so-called "leaky gut" [13,14]. These shifts have been linked with obesity, insulin resistance, and inflammatory bowel disease [13,14], though the authors of these reviews are careful to note that diet, medication use, and other confounders make it genuinely difficult to isolate microplastics as an independent cause in cross-sectional human data [13].3

Reproductive and developmental effects

Reproductive tissue turned out to be one of the more startling places microplastics have been found. A 2024 University of New Mexico study examined testicular tissue from 47 human donors and 28 dogs and detected microplastics in every single sample which had twelve different polymer types in all and the most abundant is polyethylene [8]. In the canine samples, higher polyvinyl chloride content correlated with lower sperm counts, though the human samples were not analysed for sperm count directly [8]. Independent work from China has detected microplastics directly in human semen and testicular tissue [9], and a larger 2024 multi-site study of 113 men across three Chinese provinces found microplastics in every semen and urine sample tested, with a measurable dose-response relationship between the number of polymer types detected and reduced sperm motility [10]. It is worth being cautious here: these are observational, cross-sectional findings, and the studies themselves generally describe the results as correlational rather than causal, calling for confirmation in larger, longitudinal cohorts [10]. Still, the consistency across independent research groups and countries is difficult to dismiss.

Respiratory Effects

Airborne microplastics are inhaled continuously in both outdoor and indoor environments, and once inhaled they can lodge deep in lung tissue. Researchers using infrared spectroscopy identified microplastic fibres and fragments in lung biopsy samples from living patients, with a noticeably higher concentration in the lower airway regions [4], a pattern consistent with animal studies showing that inhaled nanoplastics can trigger pulmonary inflammation and, in some models, cross into systemic circulation [19]. Whether chronic inhalation contributes meaningfully to conditions such as asthma or chronic bronchitis in humans is not yet established, but the biological plausibility, inflammation plus a foreign, non-degradable particle sitting in lung tissue indefinitely is hard to dismiss out of hand.

Neurological Effects

The most attention-grabbing finding in this entire field so far may be a 2025 Nature Medicine study that examined brain tissue collected at autopsy and found measurably higher concentrations of microplastics in samples from 2024 than in comparable samples from 2016, with people who had a dementia diagnosis showing the highest concentrations of all [6]. Polyethylene made up roughly three-quarters of the plastic mass recovered, and electron microscopy confirmed the particles as shard-like nanoscale fragments consistent with plastic debris [6]. The authors were careful to frame the dementia association as correlational rather than causal, and the study drew substantial methodological criticism from other scientists over contamination controls and measurement precision, prompting a published correction to the original paper

[31,32]. Independent of that particular study, animal work has separately shown that nanoplastics are able to cross the blood-brain barrier and provoke microglial activation, offering a plausible mechanism even if the human clinical significance remains unresolved [19].

Cardiovascular Effects

Perhaps the single most consequential human study published so far came out of Italy in 2024, when researchers examined carotid artery plaque removed from 257 patients during routine endarterectomy surgery. Using mass spectrometry and electron microscopy, researchers have detected microplastics and nanoplastics predominantly polyethylene with some polyvinyl chloride embedded within the plaque of just over half the patients [5]. Over roughly three years of follow-up, patients whose plaque contained detectable plastic had a 4.5-fold higher risk of heart attack, stroke, or death from any cause compared with patients whose plaque did not [5]. That is a striking number for an observational study, and the authors themselves acknowledged that unmeasured confounders could account for some of the association. Subsequent correspondence in the same journal raised legitimate concerns about possible sample contamination during surgery [5]. Even with those caveats, it remains the strongest single piece of evidence to date that microplastics found inside human tissue correlate with a hard, clinically meaningful health outcome rather than just a laboratory biomarker.

Endocrine Disruption

Because microplastics so often carry bisphenol A, phthalates, and flame retardants as passengers, they function as a delivery mechanism for endocrine-disrupting chemicals even independent of any physical effect the plastic itself might have [19,35]. These chemicals can mimic, block, or otherwise interfere with the body's own hormone signalling at surprisingly low concentrations, and researchers have linked chronic low-level exposure to thyroid dysfunction, altered reproductive hormone levels, and metabolic disruption [19]. Pregnant women, infants, and adolescents are usually flagged as the populations of greatest concern, given how sensitive hormone-dependent development is during those life stages [3,35].

Cancer Risk

The cancer question is where the field is arguably moving fastest and where the evidence is still most preliminary. New Zealand researchers proposed in 2023 that rising microplastic exposure since the mid-twentieth century could help explain the puzzling global rise in early-onset colorectal cancer, since microplastics could plausibly disrupt the protective colonic mucus layer that normally shields the gut lining from bacterial toxins [11]. The authors described this explicitly as a hypothesis worth investigating rather than an established cause. Chinese researchers have since gone further, directly detecting microplastics mostly polyvinyl chloride and polyethylene inside colorectal tumour tissue itself, with greater variety and density than in adjacent healthy tissue [12]. A separate case-control study of 258 colorectal cancer patients found a dose-response relationship between faecal microplastic concentration and cancer risk [27]. Taken together with older evidence that microplastics can adsorb known carcinogens such as polycyclic aromatic hydrocarbons, the mechanistic story is coherent [25,26,28], but every author working in this space stresses the same caveat. None of this establishes causation in humans, and the field needs prospective cohort data before any firm conclusion is warranted.

Bioaccumulation and Long-Term Exposure

Unlike most substances the body encounters, plastic does not break down once it is inside tissue. Particles that reach the liver, kidneys, lungs, or brain tend to stay there, accumulating gradually over years of continuous low-level exposure [6,17]. The Nature Medicine brain study's most striking secondary finding was arguably not the dementia association but the trend over time: brain samples collected in 2024 contained substantially more plastic than samples from the same institution collected in 2016, suggesting that whatever is accumulating in human tissue is accumulating faster as environmental plastic pollution itself increases [6]. Because these particles are also relatively efficient carriers for other persistent pollutants, their long-term presence may compound risk over decades in ways that are genuinely difficult to study using the short exposure windows typical of laboratory research [17].

Organ System	Representative Human Finding	Key Reference(s)
Circulatory system	Microplastics detected in blood of 17/22 healthy volunteers; detected in carotid plaque of >50% of surgical patients, linked to 4.5x higher cardiovascular event risk	[2, 5]
Reproductive system	Microplastics found in 100% of examined human testis samples and in semen; dose-response with sperm motility	[8, 9, 10]
Placenta / fetal interface	First direct evidence of microplastics on both maternal and fetal sides of the placenta	[3]
Respiratory system	Microplastic fibres recovered from human lung biopsy tissue, concentrated in lower lobes	[4]
Nervous system	Higher brain microplastic concentrations in 2024 vs 2016 autopsy samples; highest in dementia cases (contested)	[6, 31, 32]
Gastrointestinal tract	Altered gut microbial composition after one month of exposure to plastic tableware; dysbiosis linked to metabolic and inflammatory outcomes	[13, 14, 15]
Colorectal tissue	Microplastics detected within colorectal tumour and peritumoral tissue; dose-response with faecal microplastic load and cancer risk	[11, 12, 27]

Gaps in Research

For all the tissue-detection headlines of the last few years, the field's biggest weakness has not really changed: there is still very little long-term human data connecting a measured exposure level to a measured clinical outcome. A 2025 systematic review of in vivo human evidence noted that most existing studies simply quantify how much plastic is present in blood, stool, or tissue without establishing whether that presence causally relates to any specific disease [16], which leaves public health authorities in the uncomfortable position of trying to write policy without solid dose-response data to work from [16]. Detection methodology is itself a source of disagreement, the debate that followed the Nature Medicine brain study over appropriate contamination blanks and quantification limits illustrates how much variation still exists between labs and techniques [31,32], and a separate critique of nanoplastic quantification in bottled water raised near-identical concerns about procedural blank contamination [32]. Nanoplastics remain particularly hard to study simply because they are difficult to detect reliably below one micrometre, and co-exposure to the mixture of chemicals that plastics typically carry makes it genuinely difficult to isolate the effect of the plastic particle itself from the effect of whatever it happens to be carrying [18,25]. Closing these gaps will require standardized detection protocols across labs, and most importantly prospective cohort studies that follow real people with measured exposure levels over years rather than relying on cross-sectional snapshots or laboratory extrapolation.

Mitigation and Policy

Governments have started to treat this as more than an environmental curiosity. In March 2022, 175 nations at the UN Environment Assembly adopted Resolution 5/14, committing to negotiate the first legally binding international treaty covering the full lifecycle of plastic from production and design through to disposal with microplastics specifically flagged as a regulatory target [29]. Negotiating sessions have continued since, through Punta del Este, Paris, Nairobi, Ottawa, Busan, and Geneva, with the process proving considerably more contentious than initially anticipated by the organisers. A 2024 academic assessment of the negotiations noted persistent disagreement over whether the treaty should cap plastic production itself or focus narrowly on waste management and recycling [30]. At a national and industry level, the more concrete tools already in use include restrictions on single-use plastics and cosmetic microbeads, extended producer responsibility schemes that shift recycling costs back onto manufacturers, mandatory environmental labelling, and growing investment in standardized detection methods so regulators can actually measure what they are trying to limit [29,30]. Public awareness campaigns and continued biomonitoring research round out the picture, though most researchers in this space agree that meaningful progress will depend on reducing plastic production at the source rather than managing its consequences after the fact.

Conclusion

What remains unproven is how much harm this exposure actually causes ordinary people over a lifetime and closing that gap will take exactly the kind of patient, wellcontrolled, longitudinal human research that has so far been in short supply. Until then, the honest position is neither alarm nor dismissal: the evidence is too consistent to ignore, yet too incomplete to translate into individual risk estimates. Sustained interdisciplinary research, paired with the coordinated policy action now being negotiated at the UN level, offers the most realistic path toward understanding and eventually reducing human exposure to these truly invisible invaders**.**

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