



PLASTICS RESEARCH
COUNCIL
FACTS FOR A BRIGHTER FUTURE

MICROPLASTICS EVIDENCE REVIEW

WHAT THE SCIENCE
ACTUALLY SHOWS

Reviewed by independent scientific,
medical & toxicological experts

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Abstract

Dust and particles are everywhere. They are an unavoidable consequence of living in a physical world: soil, minerals, pollen, skin, fibers, soot, food, metals, glass, ceramics, wood and countless other materials generate particles that people inhale and ingest every day. Plastic is one small component of this much larger particulate background, and the exact fraction varies by setting, size cutoff and analytical method. The scientifically relevant question is therefore not whether plastic particles can be detected, but whether they occur at a meaningful dose and show hazard beyond the particles already present around us.

Across decades of polymer toxicology, particle toxicology, exposure science, occupational-dust research, biomonitoring and environmental studies, no reliable evidence shows ordinary common-polymer plastic dust to be more hazardous than the broader low-toxicity dust background at realistic exposure. At sufficiently high, atypical particle loading, plastic particles can produce irritation, inflammation or other responses, just as many poorly soluble particles can. By contrast, several non-plastic particle classes have established serious human hazards that ordinary plastic dust has not demonstrated, including respirable crystalline silica (quartz), asbestos, occupational wood dust and certain toxic metal-containing particles. There is therefore no scientific or logical basis for treating ordinary plastic dust as a special hazard class simply because it is plastic.

The evidence on plastic particles is not an emerging blank slate. The wider literature spans more than five decades, and the Plastics Research Council comprehensive 2026 synthesis catalogued 1,661 canonical evidence records in addition to the broader polymer and particle literature. Taken together, the evidence supports a solid conclusion: normal real-world microplastic exposure has not been shown to cause human disease. Public-health communication should remain proportional to demonstrated risk. Disproportionate fear can itself increase stress and anxiety and drive unnecessary behavior changes. The rational response is accurate measurement, realistic dose comparison, sensible control of total dust where appropriate, and attention to any particle hazards that are actually demonstrated.

Key Findings

The evidence in brief

- **Dust and particles are everywhere and cannot be eliminated entirely.** Exposure to particles is a normal part of life: people continuously encounter mineral, biological, combustion, textile, food, metal, glass, ceramic and other particles in air, water, food and ordinary dust.
- **Plastic is a small or trace fraction of the total particle background in many measured settings.** The exact fraction varies by environment and method, but plastic is generally not the dominant component of the dust and particles people encounter.
- **No reliable evidence shows ordinary common-polymer plastic dust to be more hazardous than the broader low-toxicity dust background at realistic exposure.** In contrast, respirable crystalline silica, asbestos, occupational wood dust and certain toxic metal-containing particles have established serious human hazards that ordinary plastic dust has not demonstrated.
- **At sufficiently high, atypical doses, plastic particles can cause particle-related effects just as other dusts can.** Irritation, inflammation, oxidative stress and impaired clearance at very high particle loading are general particle-toxicology phenomena, not evidence that plastic is a uniquely hazardous particle class. There is no scientific or logical justification for treating ordinary plastic dust as a special case simply because it is plastic.
- **Dose is decisive, and real exposure is extremely small.** Best mass-based exposure estimates show that normal intake and modeled internal burden are small in absolute terms. Principal higher-confidence comparisons place normal exposure approximately 3 million to 600 million times below selected study-specific doses that produced no adverse effects in animal studies and far below occupational limits for low-toxicity dust.
- **The evidence base is large and mature, not an emerging blank slate.** The wider literature spans more than five decades, and the 2026 comprehensive PRC synthesis catalogued 1,661 canonical evidence records. The integrated conclusion is solid: normal real-world microplastic exposure has not been shown to cause human disease.
- **The vast majority of ingested particles pass through the body.** Meaningful accumulation of microplastics in humans has not been demonstrated. Detection in stool is evidence of elimination, not evidence of danger.
- **Claims about plastic in blood, brain, organs, placenta, arteries, fertility, cancer, dementia, and cardiovascular disease remain unproved.** Some studies report particles or polymer-associated signals, but they do not prove realistic exposure, intact particles, accumulation, toxicity, disease, or causation.
- **Alarming studies do not represent real life.** They often use extreme doses, artificial polystyrene beads, forced exposure routes, weak contamination controls, method-limited

detection, or particle counts without mass context. These studies may be useful for screening or method development, but they do not prove everyday risk.

- **Particle count is not dose.** A very large number of extremely small particles can still represent a negligible mass. Risk assessment requires identity, mass, dose, size, exposure route, retention, and demonstrated harm.
- **Microplastics have not been shown to be an important toxic-chemical delivery pathway for humans.** Plastic particles can contain or sorb chemicals in principle, but current evidence does not show that they deliver a meaningful toxic dose compared with direct exposure from food, air, water, dust, medicines, cosmetics, consumer products, and the environment.
- **Additives, dissolved chemicals, and microplastic particles are different risk questions.** Claims about BPA, phthalates, PFAS, monomers, oligomers, dyes, pigments, or leachables should not be converted into claims about plastic particles unless the evidence proves that connection.
- **Avoiding plastics because of microplastics is not justified by current evidence.** Replacing plastics with heavier or less efficient alternatives can increase waste, greenhouse gas emissions, transport burden, breakage, food spoilage, cost, or other environmental impacts.
- **Disproportionate concern about plastic dust is not scientifically justified and is not harmless.** Public-health communication should distinguish demonstrated hazards from detection alone. Unnecessary fear can increase stress and anxiety and drive poor decisions; this does not mean any particular symptom is imaginary or psychogenic. Litter should still be prevented, waste managed responsibly and measurements made accurately.
- **The conclusion should change only if strong evidence proves harm at realistic exposure.** That would require confirmed intact plastic particles, realistic exposure levels, strong contamination controls, measured mass as well as particle count, meaningful retained dose, reproducible adverse effects, causation rather than association, and independent replication.

Bottom line

Microplastics are measurable and widespread because dust and particles themselves are ubiquitous. Plastic is only a small part of that much larger particle background, and ordinary common-polymer plastic dust has not been shown to be more hazardous than the broader low-toxicity dust mixture at realistic exposure. Several non-plastic particle classes - including respirable crystalline silica, asbestos, occupational wood dust and certain toxic metal-containing particles - have established serious hazards that ordinary plastic dust has not demonstrated. After decades of research and a comprehensive synthesis of 1,661 canonical evidence records, the conclusion is solid: there is no scientific or logical basis for treating ordinary plastic dust as a special public-health threat.

Executive Summary

Dust and particles are everywhere. They are an unavoidable part of living in a physical world, and people inhale and ingest mineral dust, cellulose, food particles, fibers, skin flakes, pollen, soot, metals, glass, ceramics, wood and many other particles every day. Sensitive analytical methods therefore find particles almost everywhere, including biological samples. The presence of particles is normal; the risk question is what the particles are, how much is present, and whether the dose and material are capable of causing harm.

Plastic is one small component of this much larger particulate background. The exact fraction varies by setting, size cutoff and method, but measured indoor, outdoor, water, blood and lung evidence consistently shows why plastic should not be interpreted in isolation from the many non-plastic particles present alongside it.

This conclusion is not based on dismissing or ignoring studies. The wider literature spans more than five decades, and the 2026 comprehensive Plastics Research Council synthesis catalogued 1,661 canonical evidence records across occurrence, exposure, analytical reliability, kinetics, toxicology, comparative particle science and human evidence. The central risk conclusion is therefore not an unresolved blank slate: normal real-world microplastic exposure has not been shown to cause human disease.

Microplastics are measurable and widespread, but detection is not danger. Their widespread detection is expected because plastic is used widely and all solid materials shed particles. Finding a particle, a plastic-like signal, or a polymer-associated chemical fragment does not prove toxicity, accumulation, disease, or causation. A valid health-risk claim must show that intact plastic particles were present, that contamination was excluded, that exposure was realistic, that the dose was meaningful, and that harm occurred because of the plastic particles.

No reliable evidence shows ordinary common-polymer plastic dust to be more hazardous than the broader low-toxicity dust background at realistic exposure. The evidence is asymmetric in an important way: respirable crystalline silica (quartz), asbestos, occupational wood dust and certain toxic metal-containing particles have established serious human hazards, whereas comparable hazards have not been demonstrated for ordinary plastic dust. This is why focusing on plastic while excluding the much larger and sometimes more hazardous non-plastic particle background can distort risk interpretation.

At sufficiently high, atypical particle loading, plastic particles can produce irritation, inflammation, oxidative stress or impaired clearance, just as many other poorly soluble particles can. That is generic particle toxicology. It demonstrates that dose matters; it does not show that plastic is a special hazard class or that ordinary environmental exposure is harmful.

There is therefore no scientific or logical justification for treating ordinary plastic dust as a special case merely because it is plastic. The generic properties most often invoked as reasons for concern - small size, surface area, persistence, hydrophobicity, charge, sorption, biofilms, inhalation and translocation - are shared by many other particle classes. Plastic-specific risk requires evidence of disproportionate harm at realistic exposure, not simply evidence that plastic particles exist.

Dose is decisive. Best available mass-based estimates show that normal human exposure to microplastics is extremely small. Modeled external intake and modeled internal burden are both small in absolute terms, although they should not be divided to infer a retention percentage because the models cover different particle-size domains. Normal exposure is approximately 3 million to 600 million times below the principal higher-confidence study-specific doses that produced no adverse effects in animal studies and is far below occupational

limits for low-toxicity dust. The exact benchmark differs by polymer and study, but even large uncertainty in exposure estimates does not bring normal exposure close to those no-adverse-effect doses.

Many studies used to support public alarm do not represent real-world exposure. They often use concentrations hundreds, thousands, or millions of times higher than realistic exposure. They often use artificial, perfect, spherical polystyrene beads rather than the mixed, irregular, weathered particles found in the environment. They may report cellular stress markers, associations, or detection rather than disease. Such studies may be useful for hazard screening or method development, but they do not prove that normal human exposure is harmful.

Accumulation has not been demonstrated. Most ingested particles are expected to pass through the digestive tract. The body has normal particle-clearance systems, including gut elimination, mucociliary clearance, macrophage uptake, lymphatic handling, and renal or biliary clearance for sufficiently small or soluble material. Finding particles in stool is therefore evidence of elimination, not evidence of danger. Claims of large accumulation in organs must fit realistic intake, realistic retention, and basic mass balance. Many do not.

Claims about plastic in blood, brain, placenta, arteries, reproductive tissues, and other organs require particular caution. These studies are technically difficult, contamination is easy, and some methods detect chemical signals rather than intact particles. Even when particles or polymer-associated signals are reported, that does not prove accumulation, toxicity, disease, or plastic-specific behavior. Detection in a biological sample is not the same as proof of harm.

The claim that microplastics meaningfully deliver toxic chemicals to humans is also not supported at normal exposure levels. Plastic particles can contain additives or sorb chemicals in principle, but the practical question is dose. The chemical exposure carried by tiny amounts of microplastic must be compared with direct exposure from food, water, air, dust, medicines, cosmetics, consumer products, and the surrounding environment. Current evidence does not show that microplastics are an important toxic-chemical delivery pathway for humans.

Environmental detection also requires proportion. Microplastics can be found in water, soil, sediment, air, dust, wildlife, and food. But detection in the environment is not proof of human-health risk or ecological collapse. The strongest risk assessment depends on measured concentration, dose, particle type, exposure route, retention, and demonstrated biological effect. In many cases, plastic is a small minority of total particles, while natural fibers, mineral particles, cellulose, soot, pigments, and other non-plastic materials dominate.

The public has often been misled because headlines confuse detection with harm, particle count with dose, association with causation, and high-dose laboratory effects with real-world risk. A large number of tiny particles can still represent a negligible mass. A laboratory effect at an extreme dose does not prove everyday risk. A polymer signal does not prove intact particles. A correlation does not prove causation. A dramatic headline does not override decades of toxicology, exposure science, and particle science.

This review does not argue that litter should be ignored. Waste should be managed responsibly. Unnecessary particle release should be reduced where practical. Large plastic debris, lost fishing gear, and poor waste management are real environmental problems. However, those problems should not be confused with unproven claims that normal microplastic exposure causes human disease.

Nor does the evidence support fear-based avoidance of plastics. Replacing plastics with heavier or less efficient alternatives can increase waste, greenhouse gas emissions, transport burden, breakage, food spoilage, cost, or other environmental impacts. Material choices should be based on full-system evidence, not on fear of one tiny, low-toxicity component of dust.

The findings of this review can be summarized as follows:

- Dust and particles are ubiquitous and unavoidable at some level; particle exposure is a normal part of everyday life.
- Plastic is a small component of the much larger dust and particle background in many measured settings; widespread detection does not make it the dominant exposure.
- No reliable evidence shows ordinary common-polymer plastic dust to be more hazardous than the broader low-toxicity dust background at realistic exposure.
- Several non-plastic particle classes - including respirable crystalline silica, asbestos, occupational wood dust and certain toxic metal-containing particles - have established serious hazards that ordinary plastic dust has not demonstrated.
- At sufficiently high, atypical doses, plastic particles can produce generic particle effects such as irritation or inflammation, just as other poorly soluble particles can; this is not evidence of a unique plastic hazard.
- Normal real-world exposure is approximately 3 million to 600 million times below the principal higher-confidence study-specific doses that caused no detected adverse effects in animal studies.
- Many alarming studies use unrealistic doses, artificial particles, weak controls, or methods prone to false positives.
- Detection in food, water, air, blood, organs, or the environment does not prove disease.
- Accumulation has not been demonstrated at meaningful levels.
- Microplastics are not proven to be an important toxic-chemical delivery pathway for humans.
- Avoiding plastics because of microplastics is not justified by current evidence.
- Public concern should be proportional to demonstrated risk. Disproportionate focus on plastic dust has no scientific basis when the much larger particle background is ignored, and unnecessary fear can itself increase stress and anxiety.

The evidence supports a proportionate conclusion: ordinary microplastic exposure is a low-dose particle issue, not a demonstrated public-health crisis. Plastic should be evaluated in the context of total particulate exposure and compared with particle classes whose hazards are actually established, rather than singled out because of its name.

The burden of proof lies with anyone claiming that normal microplastic exposure is harmful. To overturn the current conclusion, a study would need to show confirmed intact plastic particles, realistic exposure, strong contamination control, meaningful retained dose, reproducible adverse effects, and causation rather than mere detection or association.

The scientifically justified conclusion is clear: **normal real-world exposure to microplastics has not been shown to harm human health; ordinary plastic dust has not been shown to be more hazardous than the broader low-toxicity dust background; and there is no scientific or logical reason to treat plastic dust as a special public-health threat.**

Funding, Conflicts of Interest, and Scientific Review

Purpose of this Review

This review was prepared to help the public, journalists, educators, policymakers, and decision-makers understand what the scientific evidence shows about microplastics, exposure, toxicity, accumulation, detection reliability, and real-world risk.

The purpose is not to defend or attack any material, but to evaluate claims by evidence quality.

Scientific claims in this review are judged by the same questions throughout:

1. Did the study prove the material was plastic?
2. Did it prove intact particles were present?
3. Were contamination controls strong enough?
4. Was the dose realistic?
5. Was the particle type realistic?
6. Was mass measured, or only particle count?
7. Did the study prove harm, or only detection?
8. Did it prove causation, or only association?
9. Has the result been independently replicated?
10. Does the claimed amount fit measured exposure estimates?

Studies are not accepted or rejected based on whether they are favorable or unfavorable to plastics. They are judged by whether the methods and conclusions support the claim being made.

Limits of Review

This document is a scientific evidence review, not a regulatory risk assessment, medical diagnosis, or substitute for agency determinations. It summarizes and evaluates published evidence available at the time of writing.

Evidence cutoff for this release: Literature and agency material identified by the review team through 19 August 2026 are included where relevant. Evidence identified after that date will be evaluated in subsequent updates.

The conclusions should be updated if strong new evidence shows confirmed plastic particles at realistic exposure levels causing reproducible harm in humans or credible animal models.

Current evidence does not show that normal real-world exposure to microplastics causes human disease.

Commitment to Correction

Science changes when better evidence appears. If readers identify an error, missing study, or stronger contrary evidence, the Plastics Research Council will review it using the same evidence-quality criteria applied throughout this document.

A claim will be revised if the evidence shows that revision is warranted. The standard is not whether a study is alarming or reassuring. The standard is whether the study proves what it claims.

Funding Statement

This review was prepared without external funding or influence. No company, trade association, advocacy organization, donor, or government agency controlled or influenced the content, conclusions, source selection, evidence grading, wording, or publication decisions. The review evaluates scientific claims by evidence quality alone.

Conflicts of Interest

The author and reviewers may have professional experience in polymer science, toxicology, materials, life-cycle assessment, environmental science, risk assessment, or related fields. Relevant affiliations, professional roles, and potential conflicts are disclosed so readers can judge the work transparently.

Author

Dr. Chris DeArmitt is a polymer scientist and President of Phantom Plastics LLC, a technical consulting company focused on polymer formulation, materials selection, product development, failure analysis, and related plastics-science work. Phantom Plastics is not paid to produce this review, and this review was not prepared for, funded by, or directed by any client, company, trade association, advocacy group, or political organization. He is a Fellow of the Royal Society of Chemistry, a Fellow of the Institute for Materials, Minerals and Mining, and a Chartered Chemist.

Dr. DeArmitt is also Founder of the Plastics Research Council, a nonprofit organization created to review and communicate evidence on plastics, microplastics, materials, and environmental claims. His work for the Plastics Research Council, including the review of peer-reviewed literature and preparation of this evidence review, is unpaid. He spends roughly 1,000 hours per year on this unpaid nonprofit work, reading scientific studies and making evidence-based information available to the public at no charge. Over 700 microplastics studies were reviewed directly over a 7-year period for this public Q&A review; the broader 2026 Plastics Research Council comprehensive synthesis separately catalogued 1,661 canonical evidence records.

Because Dr. DeArmitt works professionally in polymer science, readers may reasonably consider that background when evaluating this review. However, expertise in plastics is not a disqualification from assessing plastics evidence. It is the reason he is able to understand polymer chemistry, analytical methods, exposure estimates, toxicology claims, material substitution, degradation mechanisms, and life-cycle comparisons in technical detail. Dismissing a plastics scientist for explaining plastics would be no more rational than dismissing a medical doctor for explaining medical evidence. The relevant question is not whether the reviewer has expertise, but whether the claims are accurate, transparently sourced, logically reasoned, and open to correction.

For that reason, the review is designed to be transparent and checkable: claims are supported by cited sources, evidence is graded using explicit criteria, and conclusions are separated from detection claims, association claims, and high-dose laboratory findings.

The findings have also been reviewed and checked by independent experts, including professors, a toxicologist, a life-cycle-assessment expert, and other technically qualified reviewers. Their role was to examine the scientific reasoning, evidence interpretation, and consistency of the conclusions with the cited literature.

This review reflects scientific assessment of the published evidence. It was not externally funded or influenced. Corrections will be made if credible evidence shows that any statement, source, or interpretation is inaccurate.

Scientific Reviewers and Endorsers

This report was independently reviewed by scientific and technical experts with relevant experience in polymer science, toxicology, environmental chemistry, analytical methods, risk assessment, and/or life-cycle assessment. The reviewers listed below reviewed the report for scientific accuracy, balance, evidence quality, interpretation of the cited literature, and clarity of the conclusions. Their listing indicates that they approve the report being released with their names, credentials, and affiliations shown.

Reviewer participation does not imply that each reviewer read every section of the report, nor that their employer or institution formally endorses the report unless explicitly stated. All conclusions remain those of the Plastics Research Council and are based on the cited scientific evidence.

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Key to abbreviations

DFACMT - Distinguished Fellow of the American College of Medical Toxicology

MD - Medical Doctor

DABT - Diplomate of the American Board of Toxicology

P.E. - Professional Engineer

Dr./PhD - Doctor of Philosophy

FRSC – Fellow of the Royal Society of Chemistry

FIMMM – Fellow of the Institute of Materials Minerals and Mining

CChem – Chartered Chemist

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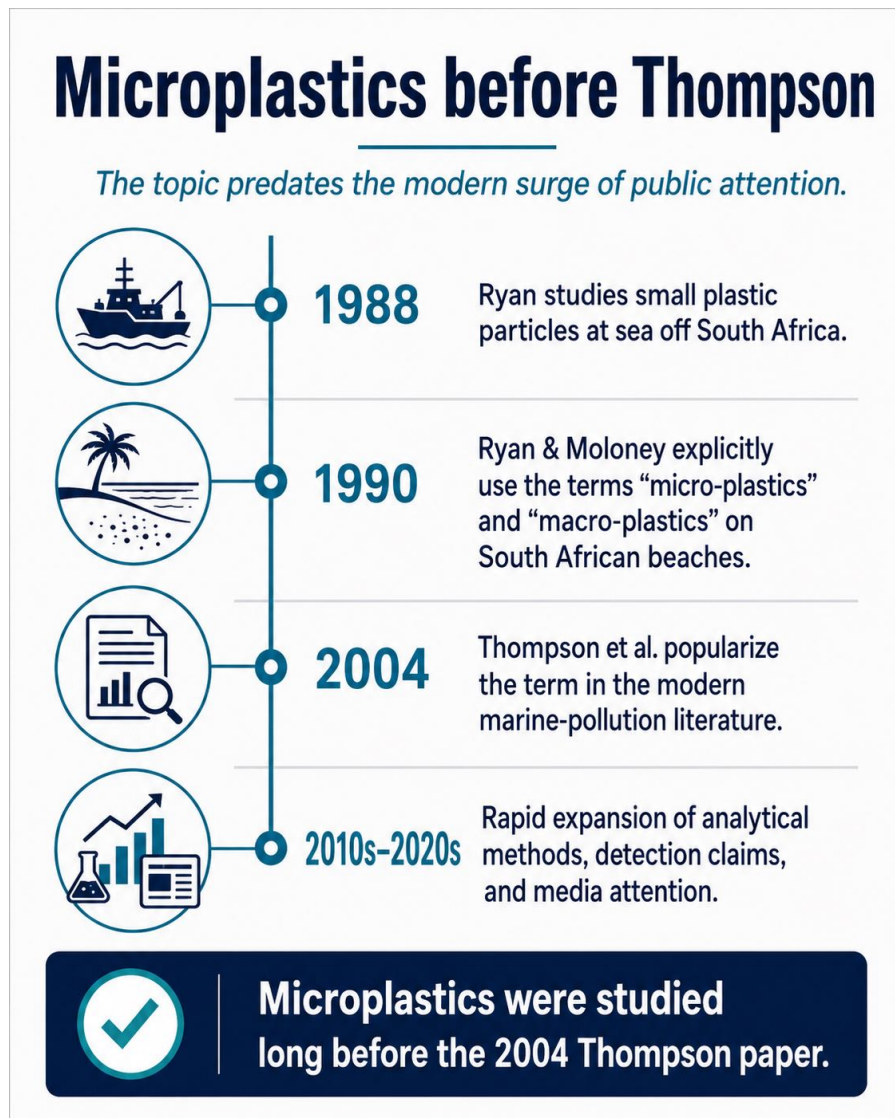
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1. Microplastics Evidence Review

Introduction

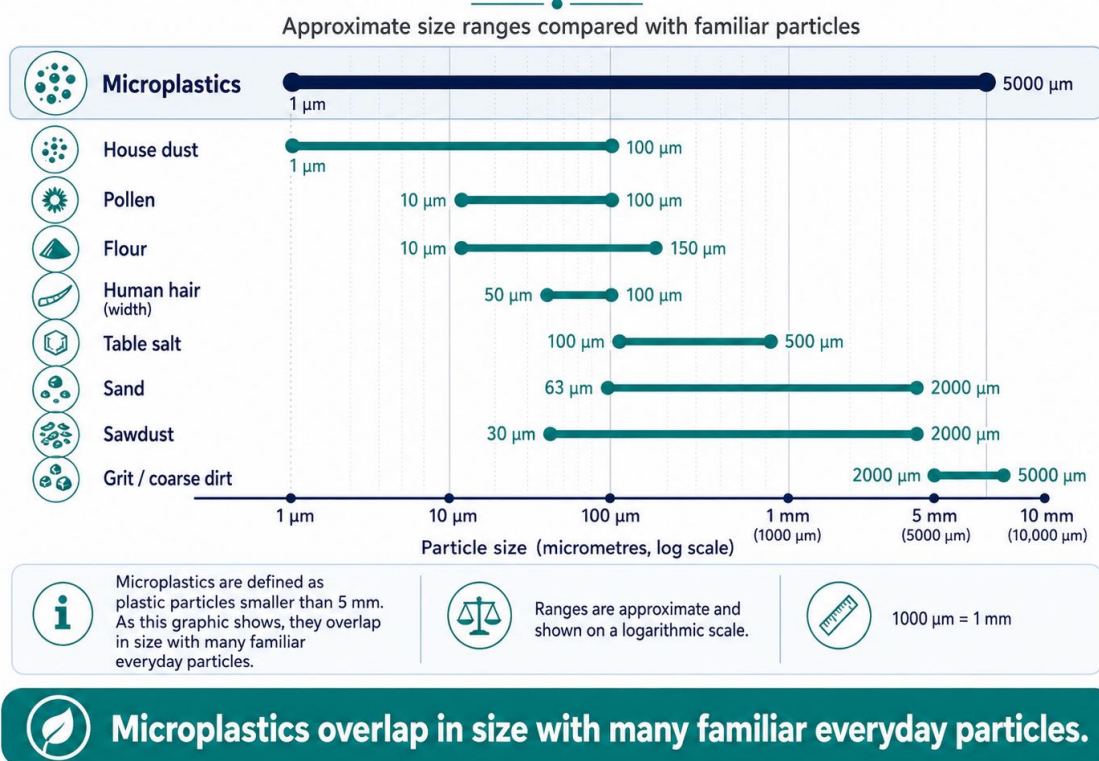
The term and concept of “micro-plastics” predate the widely cited Thompson et al. 2004 paper. Ryan and Moloney explicitly studied “micro-plastic particles” on South African beaches in 1990, separating micro-plastics from macro-plastics and grouping them into industrial pellets, expanded polystyrene, and fragments of plastic articles. Their surveys found that plastics comprised 98% of micro-artefacts and that mean micro-plastic particle density increased from 491 m⁻¹ of beach in 1984 to 678 m⁻¹ in 1989. Thompson et al. 2004 later brought broader attention to microscopic plastic fragments and fibers, but the scientific study and terminology of micro-plastics were already present in the literature more than a decade earlier. It is worth noting that studies on plastic particles, including toxicology studies, were published decades before the word microplastics was coined.



Microplastic: A solid plastic particle, fiber, or fragment smaller than 5 mm in its largest dimension, formed either intentionally at small size or by the breakdown of larger plastic items.

The term nanoplastic is applied to plastic particles below 1 micron in size.

How Big Are Microplastics?



Dust and particles are everywhere and cannot be eliminated entirely. They are an unavoidable consequence of living in a physical world. All solid materials wear, abrade, weather, corrode, fracture, crumble or shed material over time, producing mineral, organic, metallic, biological, glass, ceramic, textile and other particles. People routinely inhale and ingest this background without treating every particle class as a special public-health threat. Sensitive methods therefore find particles almost everywhere: in air, water, dust, food, soil, buildings, vehicles and biological samples. Plastic is one small part of this much larger particulate background. The scientific question is not whether plastic can be detected, but whether it is present at a meaningful dose and whether it shows disproportionate hazard compared with the many other particles present. No reliable evidence shows ordinary common-polymer plastic dust to be more hazardous than the broader low-toxicity dust background at realistic exposure, while several non-plastic dust classes have established serious hazards. Plastic deserves special concern only if evidence demonstrates special risk, not simply because the material is plastic.

Sources: Mohamed Nor 2021; Heindel 2020; Gustafsson 2018; Mukherjee & Agrawal 2017; IARC 1995; IARC 2012; WHO 2022; Ryan 1990; Thompson 2004

All solids shed particles

Particles are a normal consequence of living in a physical world.



How We Grade the Evidence

Not all microplastics studies are equal. Some studies deserve more weight because they use realistic exposure levels, validated analytical methods, strong contamination controls, confirmed particle identification, and conclusions that match the data. Other studies deserve less weight because they use extreme doses, artificial particles, weak controls, contamination-prone methods, or claims that go beyond what the experiment actually showed.

This review applies the same evidence-quality questions to all studies, whether the result appears favorable or unfavorable to plastics. The goal is not to defend a material. The goal is to separate strong evidence from weak claims.

The central rule is simple: a headline is not evidence. A study must prove what it claims to prove.

Core grading questions

- Did the study prove the material was plastic?
- Did it prove intact particles were present, not merely chemical fragments (including monomers and oligomers), dissolved substances, additives, or contamination?
- Were blanks, controls, and sample-handling procedures strong enough to rule out contamination?
- Was the exposure dose realistic compared with measured human or environmental exposure?
- Were the particles realistic, or were they artificial materials such as perfect laboratory polystyrene beads?
- Was the exposure route realistic?
- Did the study measure mass as well as particle count?
- Did it prove harm, or only detection?
- Did it prove causation, or only association?
- Has the result been independently replicated?

Evidence-quality scorecard

Criterion	Stronger evidence	Weaker evidence
Particle identity	Polymer confirmed by a validated method with transparent quality criteria.	Dye staining, weak spectral match, or bulk chemical signal only.
Intact particles	Particle size, shape, and location are shown.	Only pyrolysis fragments or indirect chemical signals are reported.
Contamination control	Strong procedural blanks, clean sampling, and blank correction.	Weak or missing blanks; unclear handling; plastic-rich lab environment.
Dose realism	Exposure matches measured real-world levels.	Doses hundreds, thousands, or millions of times above real exposure.
Particle realism	Particles reflect environmental materials, sizes, shapes, and weathering.	Perfect spherical laboratory beads or polymers not representative of normal exposure.
Exposure route	Route matches human or environmental exposure.	Forced bolus, injection, cell bath, or other artificial route.
Dose metric	Mass, particle count, size, and polymer type are reported where possible.	Particle count alone, especially for tiny particles, with no mass context.
Biological outcome	Clear adverse effect at realistic exposure.	Stress markers, irritation, or cellular effects only at extreme doses.
Causation	The study shows the plastic caused the effect.	Detection or association is treated as harm.
Replication	Independent studies confirm the result.	Single unconfirmed study or method-development result.
Conclusion	The conclusion matches the data.	The headline or abstract overstates what was proved.

Evidence grades

Strong: confirmed identity, strong contamination control, realistic exposure, relevant particles, evidence of harm if harm is claimed, and conclusions that match the data.

Moderate: useful evidence with limitations; enough to inform discussion, but not enough by itself to prove broad human health risk.

Weak: detection or hazard-screening evidence with major limits such as unrealistic dose, uncertain identity, weak blanks, artificial particles, or no replication.

Not proved: the claim is not supported by evidence strong enough for public-health or policy conclusions.

Why this matters

The strongest studies in this field are not the loudest studies. They are the studies that measure realistic exposure, prove what the particles are, control contamination, and avoid turning detection into disease.

Many alarming microplastics claims come from studies that would score poorly under normal toxicology or analytical-chemistry standards. That does not make those studies worthless. It means they should not be used to frighten the public or justify policy unless they actually prove real-world risk.

This is why this review emphasizes evidence quality. The evidence does not divide into pro-plastic and anti-plastic. It divides into strong science and weak science.

Sources: Lenz 2016; Koelmans 2019; Koelmans 2022; Gouin 2022; Cowger 2020; Hermesen 2018; EFSA 2025; WHO 2022

How this review handles contrary evidence

This review does not ignore studies that report detection or harm. It separates them into categories: confirmed exposure, uncertain detection, high-dose laboratory hazard studies, animal studies, association studies, and human evidence. A study is accepted only for what it actually proves. Detection is not treated as disease. Association is not treated as causation. High-dose laboratory effects are not treated as normal human risk.

How quantitative claims are sourced

For the most important numerical claims, the source is named immediately next to the number or sentence it supports. This makes it clear which study supports which claim and prevents readers from having to infer support from a general source list at the end of a section.

What is the difference between microplastics, nanoplastics, plastic additives, and dissolved chemicals?

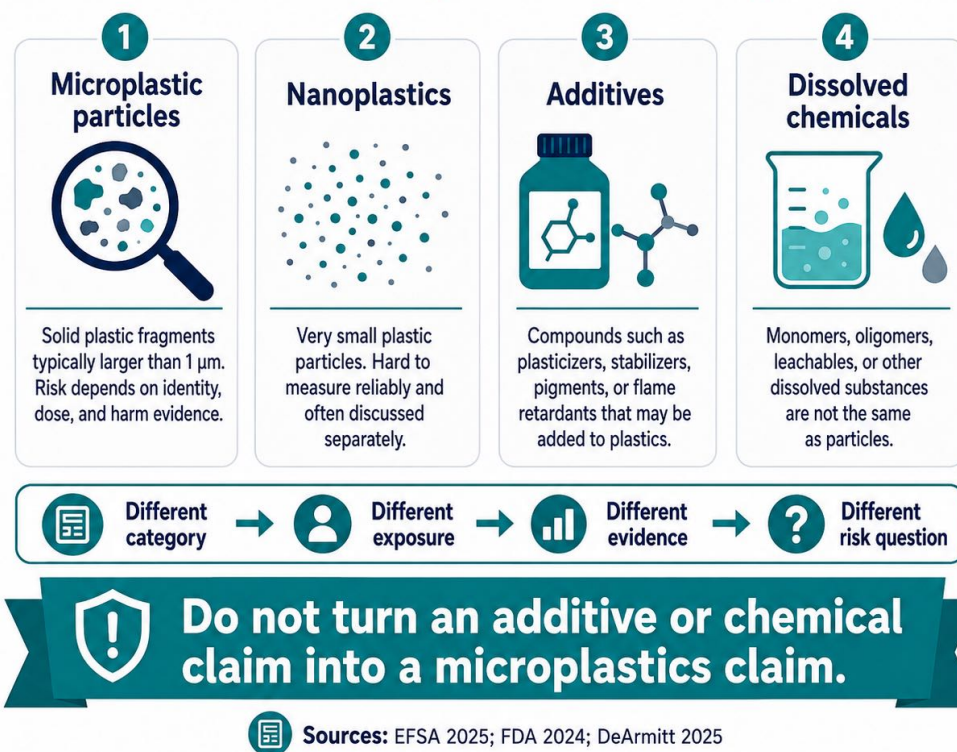
Bottom line: Microplastics and nanoplastics are solid particles. Additives and dissolved chemicals are different, and exposure questions are separate.

Many public claims mix these categories together. A plastic particle is not the same thing as BPA, phthalates, PFAS, monomers, oligomers, dyes, pigments, or dissolved organic chemicals. In fact, most plastics, including PE, PP, PET, and uPVC, do not contain BPA, phthalates, or PFAS. Each claim should be judged separately by exposure, dose, and any evidence of harm.

Sources: EFSA 2025; FDA 2024; Hahladakis 2018; Hartmann 2019; Gigault 2021

Particle, additive, or dissolved chemical?

These are different categories with different risk questions.



Are all plastics the same?

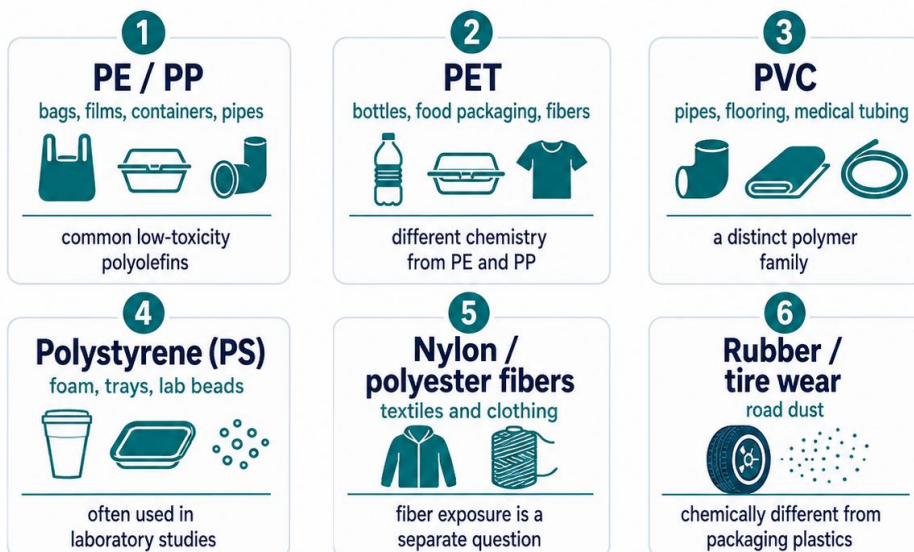
Bottom line: No. Plastic is not one material.

Polyethylene, polypropylene, PET, PVC, polystyrene, nylon, acrylics, coatings, and other resins are different plastic materials with different chemistries and uses. Rubber is an elastomeric material and is treated separately in this review rather than classified as plastic. A claim about one polymer should not automatically be applied to another.

Sources: Gilbert/Brydson 2017; Hahladakis 2018; ISO 2020; Andrady 2011

Not all plastics are the same

A result for one material does not apply to every plastic.



A study on polystyrene beads does not automatically apply to PE, PP, PET, PVC, or fibers.



Plastic is not one material.
Claims must match the actual material studied.



Sources: Brydson 2017; Gilbert 2017; DeArmitt 2025

Key: PE - polyethylene, PP – polypropylene, PVC – polyvinyl chloride, nylon is polyamide 6 or 6,6

Are plastic particles different from other particles?

Bottom line: No. Plastic particles are not a special biological category simply because they are plastic. Most claimed “microplastic mechanisms” - inhalation, ingestion, irritation at very high dose, inflammation, oxidative stress, chemical sorption, microbial biofilms, surface charge, translocation and environmental breakdown - are general particle behaviours shared by many other materials. Risk depends on dose, size, shape, surface chemistry, durability, bioavailability and intrinsic toxicity, not on the word “plastic.”

Plastic particles can be inhaled or ingested, but so can dust, soot, pollen, mineral fragments, cellulose fibers, tire particles, metal particles, silica, and many other small solids. Once exposure occurs, the relevant toxicological question is not “is it plastic?” but “what is the dose, where does it deposit, how long does it remain, and does the material have hazardous chemistry or structure?”

Several mechanisms often described as special properties of microplastics are not special to plastics:

- Particle irritation or inflammation: At sufficiently high doses, many poorly soluble particles can overwhelm clearance systems and cause inflammation or oxidative stress. This is a general particle-overload phenomenon, not a plastic-specific mechanism.
- Chemical sorption: Plastics can sorb hydrophobic chemicals, but so can soot, black carbon, clay, organic matter, fibers, sediments, and minerals. In real environments, natural particles and food usually dominate chemical exposure compared with microplastic-mediated transfer.
- Biofilms and microbes: Microbes can colonize wet surfaces. Plastic can host biofilms, but so can metal, glass, minerals, natural fibers, plant matter, sediments, and biological tissue.
- Environmental movement: Plastic particles move according to the same basic physics as other particles: size, density, shape, turbulence, settling, buoyancy, aggregation, and surface fouling determine transport.
- Degradation: Plastic particles can weather, fragment, oxidize, and biodegrade. The rate varies by polymer and environment, but “particle breakdown over time” is not unique to plastic.

Are Plastic Particles Unique?

Most claimed ‘microplastic mechanisms’ are shared by many other particles.

Claimed behavior	Unique to plastic?	Reality	What is somewhat distinctive about plastic?  Different polymer chemistry  Some polymers are low-density and can float  Hydrophobic surfaces can sorb chemicals  Some products contain additives These features do not prove unique toxicity.
 Be inhaled or ingested	No	All small particles can be inhaled or ingested.	
 Cause irritation at high dose	No	A general particle-overload effect.	
 Trigger inflammation or oxidative stress	No	Common to many particles, especially at high doses.	
 Adsorb chemicals	No	Soot, clay, fibers, and minerals also adsorb chemicals.	
 Carry microbes or biofilms	No	Any wet environmental surface can host biofilms.	
 Degrade over time	No	Plastic particles weather, fragment, and degrade; smaller particles degrade faster.	
 Many particles are more hazardous  Silica / quartz  Asbestos  Diesel soot  Metal dust  Wood dust			
 Risk depends on dose, size, shape, surface chemistry, bioavailability, and toxicity — not simply on whether a particle is plastic.			

The scientific implication is important. There is no known generic hazard-relevant particle property commonly attributed to microplastics that is unique to plastic. Small size, irregular shape, surface area, hydrophobicity, surface charge, chemical sorption, biofilm formation, inhalation, ingestion, translocation, environmental transport and persistence are all shared by other particles such as mineral dust, soot, pollen, spores, cellulose fibers, road dust, metal particles, glass, ceramics, pigments, clay and organic matter. At high particle loading, many poorly soluble particles can cause irritation or inflammation. A plastic-specific hazard claim therefore requires evidence of disproportionate harm at realistic dose, not simply the presence of a generic particle mechanism.

What is distinctive about plastics is their polymer chemistry, but polymer chemistry alone does not make a particle uniquely hazardous. The relevant question is whether that chemistry produces greater harm at realistic exposure than the much larger non-plastic particle background. For several important non-plastic particle classes the human hazard is already established: respirable crystalline silica is a recognized carcinogenic dust; asbestos is a well-established fibrous carcinogen; occupational wood dust is a human carcinogen; and certain metal-containing dusts can produce serious systemic toxicity depending on composition and exposure. Comparable hazards have not been demonstrated for ordinary common-polymer plastic dust at realistic environmental exposure.

Conclusion: Plastic particles should be evaluated within the full particle mixture, not automatically elevated into a special toxic category. The evidence should compare realistic plastic dose and potency with the much larger background of non-plastic particles people already inhale and ingest. Unless plastic is shown to contribute disproportionate harm, a plastic-only focus is scientifically incomplete and can divert attention from particle hazards that are better established.

Sources: Styles & Wilson 1973; Driscoll 2020; ECETOC 2013; Wieland 2022; IARC 1995; IARC 2012; Koelmans 2016; Hartmann 2017; Wang 2018; Zhao 2023; Loftly 2023

2. Human Health and Real-World Risk

Are microplastics harmful to human health?

Bottom line: No credible evidence shows that normal real-world exposure to microplastics harms human health. This conclusion is strengthened, not weakened, by particle context: plastic is a small component of ubiquitous dust, ordinary plastic dust has not been shown to be more hazardous than the broader low-toxicity dust background, and several common non-plastic dust classes have better-established hazards.

Actual doses people encounter in food, water, air, and dust have not been shown to cause effects in humans. Direct oral animal studies support very low intrinsic toxicity for several specific thermoplastics, including weathered polypropylene, PA-6, PTFE, cellulose acetate, and a PE/PET mixture. The evidence is not interchangeable across polymers: no verified repeated-dose oral NOAEL has been identified for real fragmented or weathered polystyrene, and PVC evidence is formulation-specific rather than a dedicated polymer-only NOAEL. There is not an absence of evidence because polymer toxicology spans many decades.

Sources: WHO 2019 and 2022; FDA 2024; Koelmans 2022; Merski 2008; Kim JS 2021; Buesen 2025; Lee 2022; Thomas 1991; Kim et al. 2025; Feron 1981

What does the evidence actually show?

Bottom line: The evidence shows that very small amounts of plastic particles may be reported in food, water, air, dust, and some biological samples. It does not show that these findings cause disease.

The key distinction is simple: detection is not danger. Dust is virtually everywhere, plastic is used widely, and all solid materials shed particles, so finding some plastic wherever dust is present is not surprising. A health claim must go further: confirm particle identity, quantify a realistic dose, show relevant uptake or deposition, compare plastic with the other particles present, and demonstrate harm attributable to the plastic. A plastic-only interpretation that omits the larger particulate background is incomplete.

Sources: WHO 2019; WHO 2022; EFSA 2016; FDA 2024; Koelmans 2022; SAPEA 2019; Gouin 2022

Are microplastics toxic, or are they mostly inert particles?

Bottom line: Many common high-molecular-weight thermoplastics have very low intrinsic oral toxicity, but no single toxicity value applies to every plastic.

Toxicity depends on material, particle preparation, dose, route, and duration. Animal studies of specific thermoplastics found no adverse effects at 25 mg/kg/day for weathered PP; 1,000 mg/kg/day for PA-6; approximately 1,600-2,000 mg/kg/day of each PE and PET component given together in a 50:50 dietary mixture; at least 2,000 mg/kg/day for fragmented PTFE; and 5,000 mg/kg/day for cellulose acetate. These are toxicological safety benchmarks from animal studies, and the exact benchmark differs by polymer and study. A fragmented-PET mouse study reported lower no-adverse-effect levels of 1.75 mg/kg/day in males and 7 mg/kg/day in females, but important particle-verification and pathology limitations prevent treating those values as a general PET threshold. No verified repeated-dose oral no-adverse-effect level has been identified for real fragmented or weathered PS, and PVC evidence is formulation-specific. Historical 25%-diet PTFE findings were preparation-dependent because residual dispersants affected outcomes. These values are not legal limits or one universal threshold for every plastic.

Sources: Merski 2008; Kim JS 2021; Buesen 2025; Lee 2022; Thomas 1991; Kim et al. 2025; Feron 1981; Naftalovich 2016; Haskell Laboratory Reports 56-61 and 224-68

Do microplastics in the environment prove a human health risk?

Bottom line: No. Environmental detection is not proof of human harm.

A material can be detectable in water, air, soil, sediment, or wildlife without being present at a dose that causes disease in people. For particulates not otherwise regulated (PNOR), OSHA Table Z-1 sets permissible exposure limits (PELs) of 15 mg/m³ for total dust and 5 mg/m³ for the respirable fraction as 8-hour time-weighted

averages. These are OSHA regulatory limits, not NIOSH recommended exposure limits (RELs). NIOSH lists PNOR in Appendix D as a substance category with no established REL and notes that it questioned whether the proposed OSHA limit was adequately protective. The OSHA values are therefore useful as occupational scale context, not as no-effect thresholds.

Sources: OSHA 2023; OSHA Table Z-1; NIOSH 2026; NIOSH Appendix D; Wieland 2022

Should people be worried about microplastics in the body?

Bottom line: Current evidence does not support avoiding plastics or changing everyday behavior because of ordinary microplastic exposure.

People are exposed to many particles every day: food particles, mineral dust, soot, textile fibers, skin flakes, pollen, cellulose, and other debris. Plastic appears to be only a small part of total particle exposure. Concern should be proportional to measured dose and proven harm (which is absent).

Sources: Plastics Research Council; WHO 2022

Do microplastics cause disease?

Bottom line: No human disease has been proved to be caused by normal microplastic exposure.

Some studies report associations or laboratory effects. That is not proof of disease in humans. Many alarming studies use artificial particles or doses far beyond real life, sometimes a million times higher than realistic environmental levels. Those studies do not show that normal exposure makes people sick.

Sources: Lenz 2016; WHO 2022; Koelmans 2022; SAPEA 2019; FDA 2024

What is the difference between finding microplastics and proving harm?

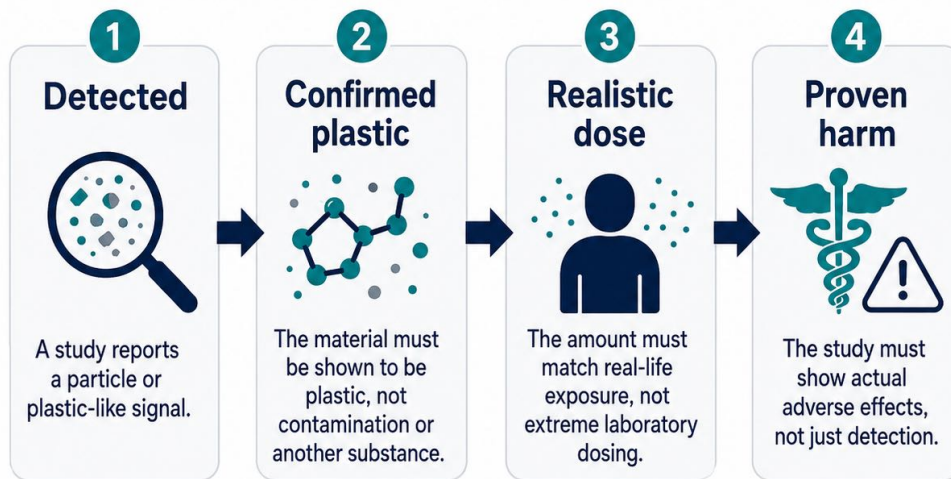
Bottom line: Finding something is not the same as proving it is harmful.

A laboratory may report a plastic-like particle or polymer signal. That only answers the question "what might be present?" It does not answer "is it dangerous?" Harm requires proof of dose, exposure route, persistence, and biological damage at realistic levels. There is no evidence of harm even after extensive testing.

Sources: WHO 2022; FDA 2024; Koelmans 2022; SAPEA 2019; Bradford Hill 1965

Detection does not equal danger

Finding a particle is only the first step.



Most headlines stop at step 1.



Detection is not proof of health risk.



A claim must pass all four steps to prove danger.

How much microplastic are people actually exposed to?

Bottom line: Best mass-based estimates indicate that exposure is very small.

Mohamed Nor et al. estimated median adult external intake at about 583 nanograms per day for microplastics in the 1–5000 μm range, or roughly 0.015 g over 70 years before elimination. The authors separately modeled adult tissue burden for the smaller 1–10 μm domain at about 40.7 ng by age 70. Both outputs are small in absolute mass, but they cover different particle-size domains and different model stages, so dividing 40.7 ng by 0.015 g does not yield a valid measured or model-based retention fraction. The defensible conclusion is that modeled ordinary external exposure and modeled internal burden are both small.

Sources: Mohamed Nor 2021; Cox 2019; Welle 2018; Senathirajah 2021

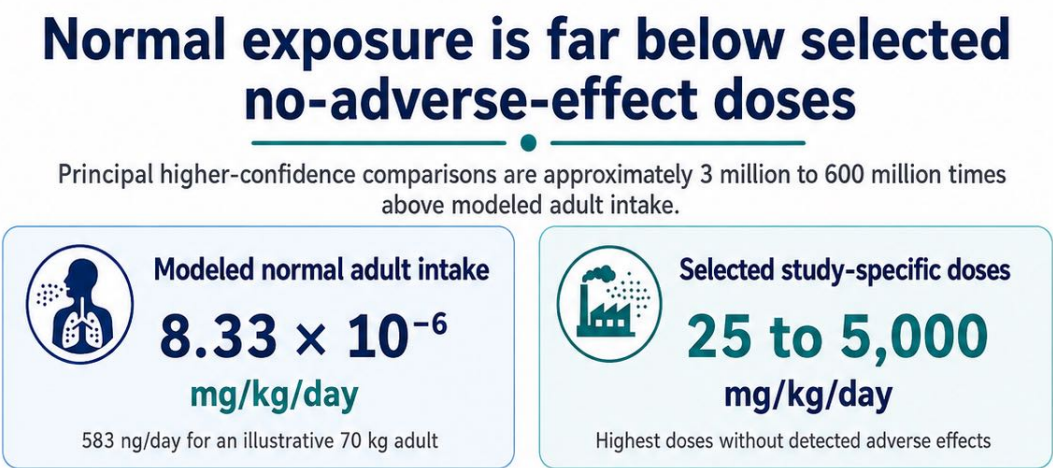
How far below no-effect and occupational benchmark levels is normal microplastic exposure?

Bottom line: Normal exposure is approximately 3 million to 600 million times below the principal higher-confidence study-specific doses that caused no detected adverse effects in animal studies and is also far below workplace dust limits. The exact benchmark differs by polymer and study; these are not legal limits or one universal threshold for every plastic.

For ingestion, Mohamed Nor et al. estimated median adult intake at 583 nanograms per day. For an illustrative 70 kg adult, this is about 8.33×10^{-6} mg/kg/day. On the same external-mass scale, normal exposure is about 3.0 million times below the weathered-PP no-harm dose; 120 million times below the PA-6 no-harm dose; roughly 190–240 million times below the PE and PET component doses that caused no harm when administered together

in the Merski mixture; about 240 million times below the PTFE no-harm dose; and about 600 million times below the cellulose-acetate no-harm dose.

A fragmented-PET mouse study reported lower no-adverse-effect values of 1.75 and 7 mg/kg/day. The lower value is about 210,000 times above modeled adult exposure, but important particle-verification and pathology limitations make this a low-confidence quantitative anchor. It is therefore discussed separately and is not used to define the principal 3-million-to-600-million-fold comparison. The exact benchmark differs by polymer, particle form, species, duration and dosing method.



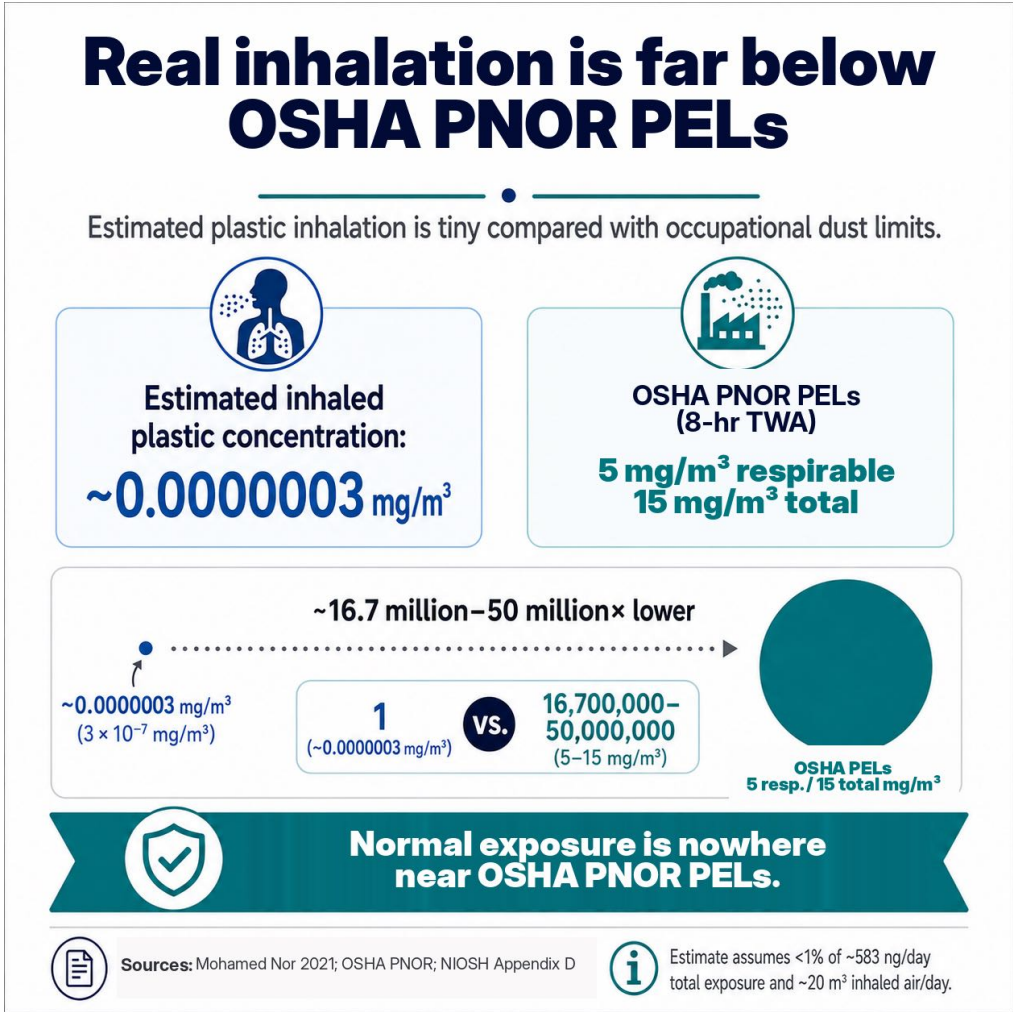
 Polymer / study context	 No-adverse-effect dose	 Times above modeled exposure
Weathered fragmented PP	25 mg/kg/day	~3.0 million-fold
PA-6	1,000 mg/kg/day	~120 million-fold
PE + PET components, administered together	1,600–2,000 each	~190–240 million-fold
Fragmented PTFE	≥2,000 mg/kg/day	≥240 million-fold
Cellulose acetate	5,000 mg/kg/day	~600 million-fold

 A separate low-confidence fragmented-PET study reported 1.75–7 mg/kg/day; it is discussed separately and is not used as the principal benchmark.

Study-specific values are context, not legal limits or one universal threshold for every plastic.

For inhalation, the same dose reality applies. OSHA PNOR PELs are 15 mg/m³ for total dust and 5 mg/m³ for the respirable fraction, each as an 8-hour time-weighted average (OSHA 2023; OSHA Table Z-1). They are OSHA regulatory limits; NIOSH does not publish a PNOR REL and directs users to Appendix D. These values provide occupational scale context rather than a toxicological no-effect threshold. Normal environmental inhalation of plastic particles is far below these workplace concentrations because modeled plastic mass in air is extremely small.

Using the OSHA respirable PNOR PEL alone, 5 mg/m³ over an 8-hour workday can correspond to tens of milligrams of inhaled particulate mass, depending on breathing volume. Modeled microplastic inhalation is in the nanogram-per-day range (Mohamed Nor 2021; Eberhard 2024), placing it millions of times below the OSHA PNOR concentration benchmark. This comparison is for magnitude only; it does not imply that a PEL is a no-effect level or that every dust exposure below a PEL is harmless.



This does not mean people should deliberately breathe dust or eat plastic. It means that risk has to be proportional to dose. When actual exposure is millions of times below levels that produce no observed harm or below workplace limits for low-toxicity dust, claims of a normal-exposure health crisis need extremely strong evidence. Current evidence does not provide that.

Sources: Mohamed Nor 2021; Merski 2008; Kim et al. 2025; Kim JS 2021; Buesen 2025; Lee 2022; Thomas 1991; OSHA 2023; OSHA Table Z-1; NIOSH 2026; NIOSH Appendix D; Wieland 2022

How do real-world exposures compare with doses used in toxicology studies?

Bottom line: Many laboratory studies use doses far above real life.

Many laboratory studies use doses hundreds, thousands, or millions of times higher than real life. That is not a small technical detail. It changes the meaning of the result. At extreme doses, even ordinary dust or soil particles can cause reactions. Lenz showed that many studies use plastic-particle concentrations a million times or more above real environmental levels (Lenz 2016). Those studies do not prove that everyday exposure is harmful.

A recent example is Jung et al. 2026 in Science Advances. Male mice were gavaged with 2 mg of commercial polyethylene particles (10–150 µm) per mouse per day for 8 weeks. That daily mass is about 3,430 times the modeled 583 ng/day adult human intake before accounting for the mouse’s much smaller body mass; on a body-mass basis it is roughly 8–12 million times higher for a 20–30 g mouse. That mg/kg comparison is an exposure-scale check, not an interspecies toxicokinetic conversion. The study used no realistic-dose arm and no dose-response series. Its spatial transcriptomics and PPARα–ANXA2 work are mechanistically informative at high dose, but the experiment does not demonstrate hepatotoxicity from ordinary human microplastic exposure.

Sources: Lenz 2016; Jung 2026; Mohamed Nor 2021; Eberhard 2024; Burns 2018



Why do many harm studies fail to represent real-world microplastics?

Bottom line: They often use the wrong dose, the wrong particles, or the wrong exposure conditions.

Common problems include unrealistically high concentrations, perfect spherical polystyrene beads that do not represent normal exposure, weak controls, poor contamination prevention, and results that are never compared with real human dose. These are not minor weaknesses. They can turn an artificial laboratory result into a frightening headline that does not apply to real life.

Sources: Lenz 2016; Gouin 2022; Eberhard 2024; Koelmans 2019; Cowger 2020; Hermesen 2018

Why did some early microplastics harm studies mislead the public?

Bottom line: Several early high-profile microplastics harm studies raised alarms before the field had strong agreement on realistic dose, representative particles, contamination control, or appropriate controls.

Early microplastics studies attracted wide public attention because they appeared to show dramatic harm to fish, oysters, and other aquatic organisms. Those studies were important in bringing attention to particle pollution, but many were later criticized because they did not represent normal environmental exposure. Common weaknesses included extreme particle concentrations, artificial laboratory polystyrene beads, nanoparticles rather than the larger particles most often measured in the environment, chemically pre-loaded particles, weak comparison particles, missing or limited dose-response data, and conclusions that were treated as broader than the experiment justified.

Examples illustrate the problem. Wardrop et al. reported transfer of sorbed pollutants from ingested microbeads to fish; that result depended on chemically contaminated microbeads and should not be read as proof that normal microplastic exposure is an important chemical-delivery pathway. Sussarellu et al. reported reproductive effects in oysters exposed to polystyrene microplastics, but the relevance of the tested particles and concentrations to normal environmental exposure was later questioned. Barboza et al. reported increased mercury bioconcentration and oxidative stress in European seabass exposed to microplastics and mercury, but that is a combined chemical-plus-particle exposure scenario, not evidence that ordinary plastic particles alone cause harm at realistic concentrations. Lu et al. reported uptake and liver effects in zebrafish exposed to polystyrene microplastics and nanoplastics, but the study used laboratory polystyrene particles and was later specifically criticized for uncertainty about particle identity and interpretation. Mattsson et al. reported behavioral and metabolic effects in fish exposed to polystyrene nanoparticles, but nanoparticle experiments should not be generalized to ordinary mixed environmental microplastics without realistic exposure comparison.

The most serious example was the high-profile Lönnstedt and Eklöv larval-fish paper in *Science*, which claimed environmentally relevant concentrations of polystyrene microplastics affected larval fish ecology. That paper was later retracted after investigation and serious misconduct concerns. A retraction does not invalidate the whole microplastics field, but it does show why dramatic claims must be checked for transparent data, appropriate controls, realistic exposure, replication, and conclusions that match the experiment.

The broader lesson was stated clearly by Lenz et al.: microplastic exposure studies should be environmentally realistic. High-dose laboratory effects, artificial particles, and special exposure scenarios may be useful for hazard screening, but they do not prove real-world ecological or human health risk. A credible study must show that the material was plastic, that the dose was realistic, that the particles were representative, and that harm occurred under conditions relevant to actual exposure.

Sources: Wardrop 2016; Sussarellu 2016; Barboza 2018; Lu 2016; Baumann 2016; Mattsson 2015; Lönnstedt & Eklöv 2016/retracted; Berg 2017; Lenz 2016; Gouin 2024

Why are polystyrene bead studies often misleading?

Bottom line: Many harm studies use perfect spherical polystyrene beads at extreme doses.

Those beads are easy to buy and easy to measure, so they are useful for laboratory experiments. They are a poor model for real human exposure. Such beads are not chemically identical to the consumer polystyrene found in the environment. For example, laboratory-made polystyrene beads are often cross-linked and may contain residues of the cross-linker divinylbenzene and surfactants from polymerization. A high-dose study using lab-made polystyrene beads does not show that the irregular, mixed, tiny amounts of plastic found in real life harm people.

This problem was apparent early in the literature: many high-profile harm studies used laboratory polystyrene beads or nanoparticles, while environmental plastic exposure is typically a mixture of weathered PE, PP, PET, fibers, coatings, and irregular fragments. Tire-rubber particles are a separate nonplastic particle class and should not be folded into plastic exposure without an explicit definition.

Sources: Lenz 2016; Gouin 2024; Wieland 2024; Koelmans 2022, DeArmitt 2020

What is wrong with saying “the science is emerging”?

Bottom line: The measurement methods are still improving, but the main safety question is not new. Plastic particles and common plastic materials have been studied for decades, and the evidence does not show harm at realistic exposure levels.

Calling the central risk question “emerging” can mislead the public. Analytical methods continue to improve, as they do in every active scientific field, but the safety question is not new. There are thousands of relevant publications across polymer toxicology, particle toxicology, dust exposure, food-contact materials, medical polymers, environmental particles, microplastics and nanoplastics. The 2026 comprehensive PRC synthesis alone catalogued 1,661 canonical evidence records. That depth of evidence means the risk conclusion does not reset to uncertainty every time a new detection study is published.

The important question is not whether microplastics can be detected. They can. The important question is whether normal real-world exposure causes harm. On that question, the evidence is already strong: realistic exposure levels are extremely small by mass, most ingested particles pass through the body, retained burden is tiny, and no credible evidence shows human disease caused by normal microplastic exposure.

It is also unlikely that future evidence will overturn this conclusion unless it is exceptionally strong. That is because plastic particles do not have a special physical property that makes them uniquely dangerous. The properties often used to raise concern — small size, hydrophobicity, surface area, persistence, surface charge, biofilm formation, chemical sorption, inhalation, ingestion, and particle transport — are also found in many ordinary dust and environmental particles. These include mineral dust, cellulose fibers, pollen, soot, clay, road dust, pigments, metal particles, glass, ceramics, spores, and organic matter. If those general particle properties do not make the much larger background of ordinary low-toxicity dust uniquely dangerous at normal exposure, they do not automatically make the tiny plastic fraction dangerous either.

This is where Bayesian reasoning matters. Science does not restart with every new headline. Each new claim must be weighed against everything already known. When decades of exposure science, toxicology, materials testing, and particle science show very low risk, a new study must be unusually strong to change the conclusion. It would need to prove confirmed intact plastic particles, realistic exposure, strong contamination control, meaningful retained dose, reproducible harm, and causation rather than mere detection or association.

Most alarming studies do not meet that standard. Many use extreme doses, artificial polystyrene beads, forced exposure routes, weak contamination controls, or particle counts without mass context. Such studies may be useful for method development or hazard screening, but they do not show that everyday exposure harms people.

Therefore, the phrase “the science is emerging” should be used carefully. Analytical methods are developing, and measurements will continue to improve. But the central risk conclusion is not an open blank slate. The current weight of evidence shows no credible reason for public concern about normal real-world exposure to microplastics. Future evidence should still be evaluated, but it should not be allowed to override decades of science unless it is strong enough to prove realistic-dose harm.

Sources: WHO 2022; SAPEA 2019; Koelmans 2022; FDA 2024; Hermesen 2018

Why does decades of evidence matter more than one alarming new study?

Bottom line: Science is cumulative. One alarming study does not reset the evidence to zero.

When scientists say “no credible evidence of harm,” that does not mean nobody has looked. It means the opposite: common plastics have been studied for decades, and realistic human exposure has not been shown to cause disease.

This is the simple idea behind Bayesian updating. Evidence is not judged one headline at a time. Each new claim is weighed against everything already known. If decades of toxicology and exposure evidence show very low risk, then a new study must be exceptionally strong to overturn that conclusion.

A new study should change the conclusion only if it uses realistic doses, proves the particles are plastic, controls contamination, shows harm at real exposure levels, and is independently repeated. Most alarming microplastics

studies do not meet those tests. Many use doses far above real life, artificial particles, weak methods, or detection claims that do not prove harm.

The practical conclusion is clear: microplastics are not an untested unknown. New research should continue, but one weak or unrealistic study should not outweigh decades of evidence showing no demonstrated human disease from normal exposure.

Sources: Lenz 2016; Merski 2008; Stock 2021; Koelmans 2022; WHO 2022; FDA 2024; Bradford Hill 1965; Goodman 1999; Ioannidis 2005; Bayes 1763

Do microplastics accumulate in the human body?

Bottom line: Accumulation has not been demonstrated.

Most ingested particles are expected to pass through the digestive tract. Mohamed Nor et al. modeled about 0.015 g of lifetime external intake for a 1–5000 μm particle domain and, separately, about 40.7 ng of adult tissue burden for a 1–10 μm model domain. These values should not be divided to infer a retention fraction because the size windows differ. Claims of large accumulation require direct proof, not assumption. Some studies use the word “accumulation” when they have only detected particles or polymer-associated signals. Accumulation and detection are not the same: accumulation requires evidence that retained burden increases over time.

Sources: Mohamed Nor 2021; Duis 2016; Prata 2022; Inoue 2025; Jovanović 2018; Schwabl 2019

Modeled tissue burden is nanogram-scale

The external-intake and tissue-burden models answer different questions.



External intake model

1–5,000 μm

~0.015 g over 70 years

Includes modeled ingestion +
inhalation pathways



Tissue-burden model

1–10 μm domain

~40.7 ng by age 70

Model-based internal burden estimate



Different size windows → no valid direct retention percentage

Dividing 40.7 ng by 0.015 g produces a numerical ratio, but not a measured or model-valid absorption/retention fraction.



What the model does support

**Most ingested particles are expected to be eliminated,
and modeled internal burden is extremely small in
absolute mass.**

Source: Mohamed Nor et al. 2021

Can fear of microplastics itself cause harm?

Bottom line: Disproportionate fear is not harmless. Sustained stress and anxiety can affect wellbeing, symptom perception, healthcare use and decision-making. That does not mean any particular symptom is imaginary or psychogenic. It means public-health communication should not create a level of fear that is disproportionate to the demonstrated hazard.

Public-health communication should distinguish ubiquitous particle detection from demonstrated disease risk. When plastic represents only a small part of a much larger particle background and no special hazard has been demonstrated at ordinary exposure, repeatedly presenting plastic dust as uniquely threatening can create unnecessary concern without a corresponding health benefit. People should be told what is known, what is not proved, how small the exposure is, and how plastic compares with better-established particle hazards.

Sources: Yaribeygi 2017; McEwen 1998; McEwen 2007; Celano 2016; Horenstein 2020

If microplastics are not shown to cause harm, why are people worried?

Bottom line: Concern is amplified when detection, hazard and risk are presented without particle context. Repeated reports that plastic has been “found” in air, food or the body can make a ubiquitous low-dose exposure appear exceptional, especially when the much larger background of mineral, biological, combustion, textile and metal particles is omitted.

Völker et al. found that scientific articles generally framed microplastic risk as uncertain or unproven, while media coverage overwhelmingly portrayed harm as certain.

Sources: Völker 2020; Yaribeygi 2017; Covello 1992

3. Microplastics in the Human Body

This section is important because body-location claims attract attention. Answers separate detection, proof of intact plastic, dose, and harm. Laforsch questioned the validity of many studies claiming plastic particles in stool, saliva, sputum, bronchoalveolar lavage fluid, blood, lung tissue, placenta, digestive tract, liver, spleen, and kidney, arguing that many reported particles were consistent with airborne laboratory dust and too large to plausibly enter the claimed tissues through the gut or lungs.

Particle movement in the body is not a new microplastics discovery...

Claims that microplastics or nanoplastics can move through biological barriers are often presented as if they reveal a new and unique plastic hazard. They do not. The movement of small solid particles through the intestinal wall has been discussed since the nineteenth century. Herbst reported finding starch granules in chyle and blood after feeding starch flour to a dog in 1844, and later authors described this phenomenon as “persorption”: the passage of undissolved microparticles through the intestinal wall into lymph or blood. Modern studies and reviews have further examined intestinal particle uptake, Peyer’s patches, M cells, macrophage handling, lymphatic transport, and particle clearance.

This history is important. It shows that the biological question is not simply whether a particle can move. Very small particles of many types can sometimes cross or be transported across biological barriers under particular conditions. The relevant risk questions are more specific: What is the particle? How large is it? How much is present? Was the material confirmed to be plastic? Was the exposure realistic? Is the particle retained or cleared? Does the retained dose increase over time? Is there reproducible harm at real-world exposure? Detection or possible translocation is therefore not the same as accumulation, toxicity, or disease.

The digestive tract is not an open pathway into the body. It is a selective biological barrier designed to absorb nutrients while excluding most insoluble particles. Older particle-uptake work showed that very small particles can sometimes cross the gastrointestinal barrier under experimental conditions, but that does not mean normal dietary particles accumulate in meaningful amounts. LeFevre and co-workers showed that chronic administration was needed before 5 micrometer particles accumulated in Peyer’s patches in mice, illustrating that uptake is limited and biology-dependent rather than automatic. Powell and co-workers reviewed the origin and fate of dietary nanoparticles and microparticles in the gastrointestinal tract and emphasized that humans are routinely exposed to many non-plastic dietary particles, including mineral particles, without that implying disease. For microplastics, the relevant conclusion is that detection in stool is expected, because most ingested particles are not absorbed and are eliminated. Claims of accumulation therefore require direct evidence of increasing retained mass over time, not merely detection of particles or polymer signals in a sample.

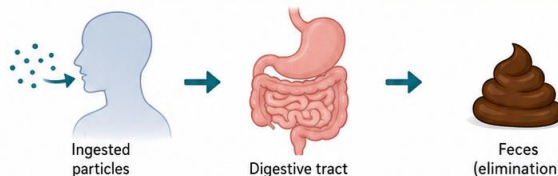
Sources: Herbst 1844; LeFevre 1980; Powell 2010; Mohamed Nor 2021; Schwabl 2019; Zhang 2021; Volkheimer & Schulz 1968; Volkheimer 1974; Geiser 2010; Oberdörster 2004

How the body clears particles

Detection is not the same as accumulation.

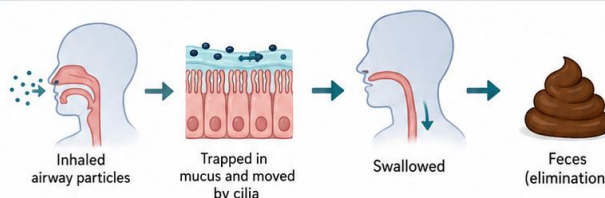
1 Ingested particles

Particles in food, water, or from hand-to-mouth transfer.



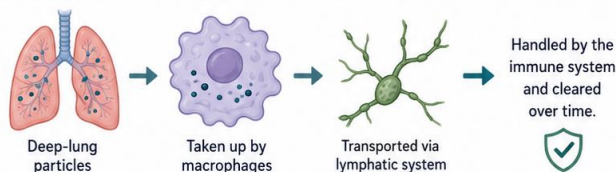
2 Inhaled airway particles

Particles trapped in the nose, throat, and upper airways.



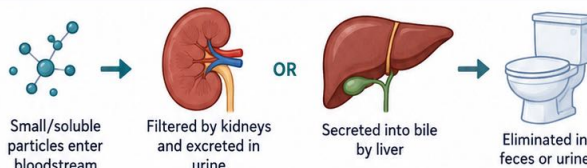
3 Deep-lung particles

Particles that reach the gas-exchange regions of the lungs.



4 Very small, soluble, or nanoscale material

Materials small enough to enter the bloodstream.



The body has normal particle-clearance systems.

Have microplastics been found in blood?

Bottom line: Some studies have reported plastic-like particles or polymer signals in blood at extremely low concentrations, but this does not prove harm and detection is uncertain.

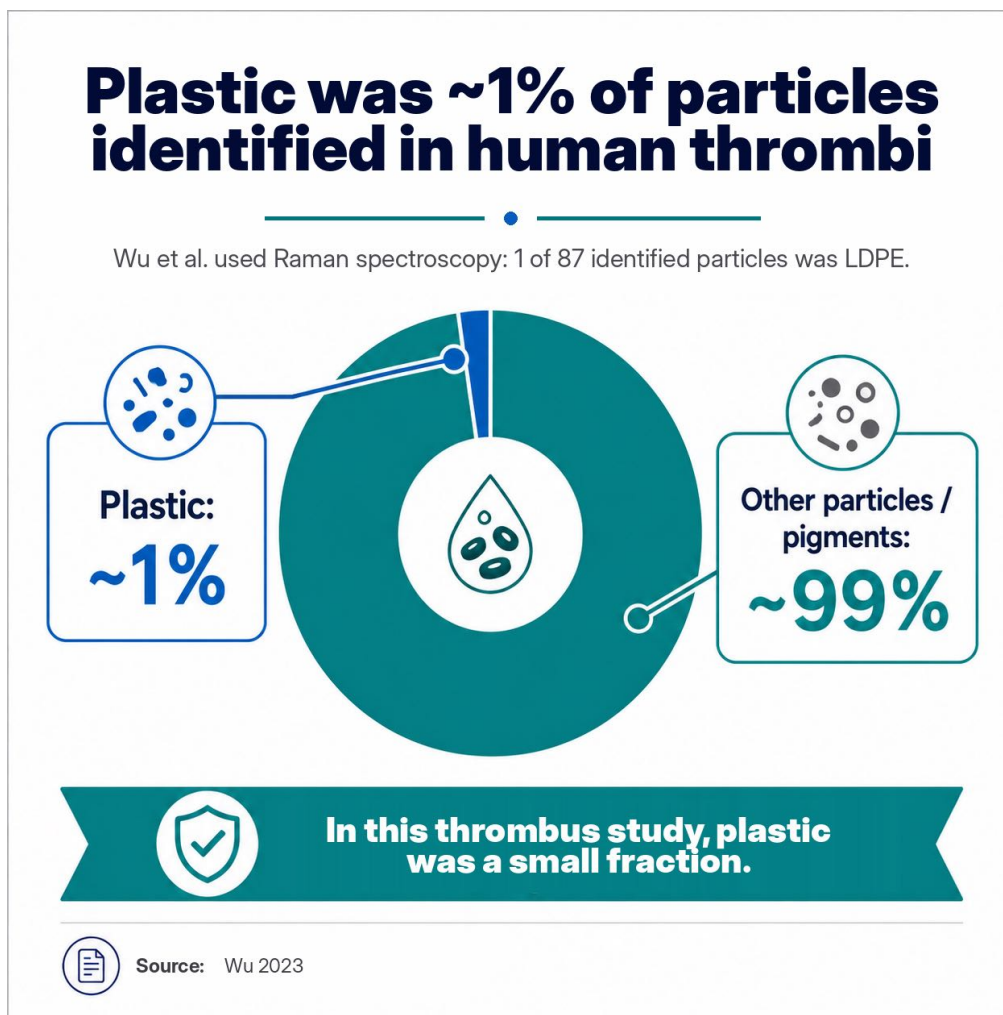
Particles in blood are not a new biological concept and are not unique to plastic. Studies published decades ago showed that inhaled or intratracheally instilled ultrafine particles can move from the respiratory tract into blood circulation under experimental conditions. This does not mean that every particle detected in blood is harmful, and it does not prove that normal microplastic exposure causes disease. It means that blood-detection claims must be interpreted in the broader context of ordinary particle exposure, including mineral dust, combustion particles, pigments, soot, metal oxides, biological particles, and other non-plastic material. The key questions remain the same: what was the particle, how much was present, was contamination excluded, was the dose realistic, and was harm demonstrated?

Blood studies are difficult because contamination is easy and the amounts reported are extremely small. A reported signal must be confirmed as real plastic rather than laboratory contamination, background dust, or a method artifact. For particle-mixture context, Wu et al. 2023 analyzed particles embedded in human thrombi rather than normal circulating blood. Raman spectroscopy identified 87 particles, of which one was low-density polyethylene (about 1%); iron compounds, pigments, and other non-plastic particles dominated. That result does not establish the composition of normal blood and does not show that any detected particle caused disease. Brits

et al. reported polymer-associated chemical signals in blood using Py-GC-MS, but the method destroys the sample and therefore does not by itself prove intact circulating plastic particles.

The thrombus finding provides useful particle-mixture context, but it should not be generalized to normal circulating blood. It shows that a diseased vascular deposit can contain many non-plastic particles and that a plastic-only interpretation can omit most of the particulate mixture. If the biological question is particle exposure, all particle classes should be considered; if the claim is plastic-specific risk, the plastic fraction must be shown to contribute a meaningful dose and disproportionate harm.

Sources: Wu 2023; Brits 2024; Li 2024; Leslie 2022; Rauert 2025; Guan 2023; Nemmar 2002; Shimada 2006; Geiser 2010



Have microplastics been found in the brain?

Bottom line: Some particle-resolved studies provide plausible evidence that plastic particles can occur in selected human brain or olfactory tissues, but current studies do not establish representative brain burden, progressive accumulation, exposure route, or neurological harm.

Evidence quality depends strongly on method. Destructive thermal methods can report polymer-associated mass but do not preserve particle size, shape, count, or location, and lipid-rich brain tissue creates analytical challenges. More recent particle-resolved studies provide stronger occurrence evidence in selected samples, but selected clinical or postmortem tissues still cannot establish representative population burden, long-term accumulation, or disease causation.

Particle movement into the brain is not unique to plastic particles. The translocation of inhaled ultrafine particles from the respiratory tract to other organs, including the brain, has been studied for decades. Oberdörster and co-

workers reported extrapulmonary translocation of inhaled ultrafine carbon particles in rats and later reported evidence consistent with movement of inhaled ultrafine particles to the olfactory bulb and some brain regions. This does not prove that normal environmental microplastic exposure causes human brain disease. It shows something narrower: very small particles of several kinds can sometimes cross biological barriers under experimental conditions. Therefore, a brain-risk claim must show more than possible particle movement. It must show confirmed plastic identity, realistic exposure, realistic particle type, dose, retained mass, and harm beyond the background of non-plastic particles.

Brain-translocation claims also need particle context. Plastic is not the only particle class studied for movement into, or effects on, the nervous system. Studies have reported brain translocation or neurological associations for several non-plastic particles, including ultrafine carbon, manganese oxide, iron-soot, zinc oxide, titanium dioxide, and black carbon. Some of these particles are more environmentally relevant than laboratory polystyrene nanoparticles because people are actually exposed to combustion particles, mineral particles, metal oxides, and soot. The correct comparison is therefore not “plastic particles versus no particles.” It is plastic particles versus the much larger and better-established background of airborne mineral, combustion, biological, and metal-containing particles.

Sources: Oberdörster 2002; Oberdörster 2004; Oberdörster 2005; Kopatz 2023; Rauert 2025; Nihart 2025; Monikh 2025; Li 2026a; Lane 2026; Mohamed Nor 2021; Chuang 2020; Suglia 2008; Hopkins 2018; Liu 2017

Is the claim that the brain contains a large amount of plastic credible?

Bottom line: The reported ~6 g brain estimate should not be interpreted as ~6 g of confirmed intact plastic particles. It is a disputed polymer-associated mass estimate with major analytical and mass-plausibility limitations.

The ~6 g value is difficult to reconcile with current exposure and internal-burden models, but the comparison must be made carefully because those models cover defined particle-size domains rather than every possible nanoplastic. The stronger methodological objection is that destructive polymer-mass methods do not by themselves establish intact-particle number, size, morphology, or location. A gram-scale brain-burden claim therefore requires independent confirmation with particle-resolved methods, rigorous contamination control, source and intake plausibility, and a defensible mass balance; those requirements have not been met.

The microscopy images are also limited because the reported particles were described as “putative” microplastics. “Putative” means presumed or suspected, not confirmed. Images or arrows pointing to particles do not establish polymer identity unless the particles are analytically confirmed as plastic and contamination is excluded.

Independent expert reaction to the brain-accumulation claim also raised concerns about interpretation, contamination, analytical uncertainty, and whether the study proved intact plastic particles were present. The Science Media Centre published expert comments from Oliver Jones, Tamara Galloway, Ben Henry, and Antonis Myridakis on the reported accumulation of microplastics in human brain tissue. Those reactions did not establish that the study was false, but they reinforced the key point: the claim requires cautious interpretation because detecting chemical signals or putative particles is not the same as proving intact plastic particles, realistic accumulation, or harm.

Sources: Nihart 2025; Rauert 2025; Monikh 2025; Laforsch 2025; Science Media Centre 2025; Mohamed Nor 2021

The “6 g brain” claim is not a validated intact-particle burden

The reported value should not be interpreted as 6 g of confirmed plastic particles in the brain.

1

Destructive method



Py-GC/MS measures polymer-associated thermal products; it does not preserve or count intact particles.

2

Biological matrix



Brain tissue is lipid-rich, so analytical interference, background and method specificity require strong validation.

3

Mass plausibility



The estimate is difficult to reconcile with current external-exposure and internal-burden models; those models also cover different size domains.

4

Causation



Even a valid occurrence measurement would not establish accumulation, disease or neurological harm.



Defensible interpretation

Treat the 6 g value as a disputed polymer-associated mass estimate — not as 6 g of confirmed intact plastic particles.

Sources: Nihart 2025; Rauert 2025; Monikh 2025; BfR 2025; Mohamed Nor 2021

Have microplastics been found in lungs?

Bottom line: Lung findings are more plausible than many other tissue claims because people inhale airborne particles.

Even so, finding particles in lung samples does not prove harm. Particle identity, contamination control, dose, size and biological effect still matter. Hayden et al. 2023 provides essential context: automated in-situ FE-SEM/EDS characterized 124,018 inorganic particles in 38 selected surgical lung-biopsy specimens and assigned them to 45 particle or mineral classes, including clays, feldspars, quartz, titanium dioxide, iron-rich aluminosilicates, aluminum oxide, zircon/zirconia and numerous metal or metal-oxide particles; rare lead-, uranium- and thorium-containing particles were also reported. More than 90% of analyzed particles were below 2.5 μm . Hayden cannot be used to calculate a plastic-to-nonplastic lung ratio because its method was qualitative and optimized for

inorganic particles, but it demonstrates why plastic detections must be interpreted within the much larger and chemically diverse background of nonplastic particles already present in lung tissue.

Sources: Hayden 2023; Laforsch 2025; Wieland 2022; Jenner 2022; Amato-Lourenco 2021; Vianello 2019

Have microplastics been found in placenta?

Bottom line: Some studies report many types of particles (Bongaerts 2020, Buerki-Thurnherr 2012) including possibly plastic in placenta samples, but they do not prove harm to the fetus.

Placenta studies are highly sensitive to contamination. Detection claims require strong blanks, clean handling, confirmed particle identity, and realistic interpretation. A particle report is not proof of disease or developmental harm. The detection of plastic has been questioned by Laforsch and others. Plastic is one of the many types of particles detected in the body and placenta.

Sources: Laforsch 2025; Bové 2019; Liu 2021; Bongaerts 2020; Buerki-Thurnherr 2012; Clough 2026; Ragusa 2021; Liu 2023; Zhu 2023

Have microplastics been found in stool?

Bottom line: Yes. Stool findings are expected and are not a cause for concern.

Stool detections show that particles including plastics can be swallowed and excreted. That is consistent with elimination rather than accumulation.

Sources: Mohamed Nor 2021; Schwabl 2019; Zhang 2021

Do detected particles stay in the body or pass through?

Bottom line: Most ingested particles are expected to pass through; modeled retained amounts are tiny compared with intake

Retention depends on size, shape, surface chemistry, and biology. The evidence does not show meaningful accumulation in humans at ordinary exposure. A small fraction of sufficiently fine particles may cross biological barriers under some conditions, but uptake is strongly size-dependent. Mohamed Nor et al. assumed 0.3% intestinal absorption for 1–10 µm particles in one model stage. Their ~0.015 g lifetime external-intake estimate covers 1–5000 µm, whereas the ~40.7 ng tissue-burden estimate covers 1–10 µm; the two outputs therefore cannot be divided to calculate a whole-exposure retention percentage. The relevant point is that modeled internal burden is nanogram-scale, not gram-scale (Nor 2021).

The body also has normal particle-clearance systems. Inhaled particles deposited in the airways and alveoli can be trapped in mucus, cleared by the mucociliary escalator, or engulfed by macrophages. Geiser reviewed macrophage clearance of inhaled micro- and nanoparticles and noted that particles deposited in the airways and alveoli are readily taken up by resident surface macrophages. This process is part of normal lung defense. It does not mean that no particle can ever persist or cause harm, but it does mean that detection of a particle is not the same as accumulation, toxicity, or disease.

Sources: Mohamed Nor 2021; Duis 2016; Prata 2022; Inoue 2025; Jovanović 2018; Geiser 2010

Have microplastics been found in every human organ?

Bottom line: No. This has not been proved.

Claims of plastic in every human organ are not established. Many reported findings rely on methods that can confuse body fats, lipids, proteins, contamination, or chemical signals with plastic. Some reported particles are also too large to make biological sense in the tissue claimed. The public should not treat these reports as proof that plastic is spread through every organ.

The conclusion is simple: plastic in every organ has not been proved.

Sources: Laforsch 2025; Clough 2026

Have microplastics been found in breast milk, semen, testes, arteries, and other tissues?

Bottom line: Some studies report plastic-associated findings in these samples, but detection is uncertain and does not prove harm.

Each claim must be checked separately. The key questions are whether intact particles were proved, contamination was controlled, dose was realistic, and health effects were actually demonstrated.

Sources: Laforsch 2025; Clough 2026; Ragusa 2022; Zhao 2023; Marfella 2024; Rotchell 2023

Do nanoplastics cross the blood-brain barrier in humans at realistic exposure?

Bottom line: Crossing of the human blood-brain barrier by environmental nanoplastics at realistic exposure has not been demonstrated.

Nanoparticles of many kinds, not just plastic, can cross the blood-brain barrier under some experimental or medical circumstances. That fact does not prove that environmental nanoplastics cross the human blood-brain barrier at normal exposure levels. Drug-delivery particles are used at high doses and are specially designed to cross biological barriers. Environmental particles are not the same.

One study reported that lab-made polystyrene nanoparticles crossed the blood-brain barrier, but it used an extreme artificial exposure. The particles were not typical environmental plastic, and the dose was about 3,000-9,000 times too high by mass. For the 293 nm particles, the particle count was about 2 trillion times too high. That is not evidence of what happens to people in real life.

Sources: Kopatz 2023; Lenz 2016; Shan 2022; Semmler-Behnke 2008

Do microplastics harm unborn babies, infants, or children at measured exposure levels?

Bottom line: No credible evidence proves that normal measured exposure harms unborn babies, infants, or children.

Some studies report particles or plastic-like signals in placenta, infant stool, or related samples. That does not prove harm. Child and pregnancy claims need especially careful wording because they are emotionally powerful and can easily be overstated.

Sources: WHO 2022; FDA 2024; EFSA 2025; Li 2020; Buerki-Thurnherr 2012

Do microplastics cause inflammation at measured exposure levels?

Bottom line: No credible evidence proves that normal measured exposure causes inflammation.

Some studies report inflammatory responses after plastic-particle exposure, but the dose context is often unrealistic. Many poorly soluble low-toxicity particles can produce inflammation, oxidative stress or impaired clearance when experimental systems are subjected to sufficiently high particle loading. This is a generic high-dose particle phenomenon, not evidence that ordinary plastic dust has exceptional toxicity. As Lenz et al. emphasized, many microplastic experiments use concentrations orders of magnitude above measured environmental exposure, limiting their relevance to everyday risk.

Sources: Lenz 2016

Do microplastics harm zebrafish at measured exposure levels?

Bottom line: No credible evidence proves that normal measured exposure causes harm.

Some studies report effects in zebrafish embryos after exposure to plastic particles (Mohan 2023), but the tested concentrations are far above realistic environmental exposure. High particle loading can also produce responses with many nonplastic materials, including ordinary particulate matter, illustrating that an effect at an extreme experimental dose is not automatically plastic-specific. Other zebrafish studies report developmental effects from particulate matter and from high sodium-chloride concentrations. These experiments may be useful for hazard screening but do not establish human-health risk from ordinary microplastic exposure.

Sources: Lenz 2016; Mohan 2023; Morris 2023; Manjunatha 2021; Seli 2024

Do microplastics persist in the body?

Bottom line: Current evidence does not demonstrate meaningful long-term accumulation of microplastics in humans at normal real-world exposure levels.

Most ingested insoluble particles are expected to be eliminated. Mohamed Nor et al. modeled about 0.015 g of lifetime external intake for 1–5000 μm particles and, separately, about 40.7 ng of tissue burden for the 1–10 μm domain; because the size windows differ, these values cannot be divided into a whole-exposure retention percentage. A small fraction of sufficiently fine particles may enter tissues, where clearance can involve macrophage uptake, lymphatic transport, biliary pathways, and renal clearance for sufficiently small or soluble material. Detection, persistence, and progressive accumulation are distinct claims; current human evidence does not demonstrate a meaningful retained burden increasing over time.

Sources: Liebert 1976; Wagner 2020; DeArmitt 2017

4. Food, Water, and Household Sources

Are microplastics in food dangerous?

Bottom line: No credible evidence shows that typical microplastic levels in food are dangerous.

Food naturally contains many particles and biological materials. The relevant question is whether the dose is large enough to cause harm; that has not been shown. Estimated median adult intake of 583 ng/day corresponds to about 8.33×10^{-6} mg/kg/day for a 70 kg adult. Normal exposure is approximately 3 million to 600 million times below the principal higher-confidence study-specific doses that caused no detected adverse effects in animal studies. The exact benchmark differs by polymer and study; these are not legal limits or one universal threshold for every plastic.

Sources: Mohamed Nor 2021; Kim et al. 2025; Merski 2008; Kim JS 2021; Buesen 2025; Lee 2022; Thomas 1991; FDA 2024; WHO 2022; EFSA 2016; Koelmans 2022

Are microplastics in seafood a health risk?

Bottom line: No credible evidence shows that microplastics in seafood are a human health risk at normal consumption levels.

Microplastics can be reported in seafood, especially in digestive tracts. That does not prove danger. The key question is how much plastic is present in the edible portion, whether the particles are confirmed as plastic, and whether the dose causes harm. For example, several studies reported plastic particles in crabs, but it was found that they were not in the edible (meat) portion (Wu 2023). Low levels of microplastics (MP) in wild mussels indicate that MP ingestion by humans is minimal compared with exposure from household fibers falling onto a plate during a meal.

Sources: EFSA 2016; FDA 2024; WHO 2022; FAO 2017; Smith 2018; Hantoro 2019; Catarino 2018

Are microplastics in salt, beer, honey, milk, rice, meat, or vegetables dangerous?

Bottom line: Finding a few particles in food does not prove danger.

Food naturally contains countless particles and biological materials. Risk depends on dose and toxicity. Current evidence does not show that microplastics reported in foods cause human disease. Actual exposure is far below the principal higher-confidence study-specific no-adverse-effect dose scale - approximately 3 million to 600 million times lower. Plastics and other particles are found in salt and have attracted attention. Other studies have also identified heavy metals, including lead, in salt; those findings should likewise be evaluated by actual dose and risk rather than by material label alone (Mohammadi 2025; Fayet-Moore 2020).

Sources: FDA 2024; WHO 2022; EFSA 2016; Toussaint 2019; Karami 2017; Mohammadi 2025; Fayet-Moore 2022

Are microplastics in bottled water dangerous?

Bottom line: A health hazard from bottled-water microplastics has not been demonstrated at measured exposure levels.

Some bottled-water studies report large particle counts, especially when very small particles are included. Counts are not the same as mass or risk. The important questions are whether the particles were accurately identified, how much mass was present, and whether that dose causes harm. Current evidence does not show that bottled-water microplastics are a health hazard.

Sources: NIH 2024; FDA 2024; WHO 2019, Welle 2018; Jüngling 2026; Schymanski 2018; Gambino 2022

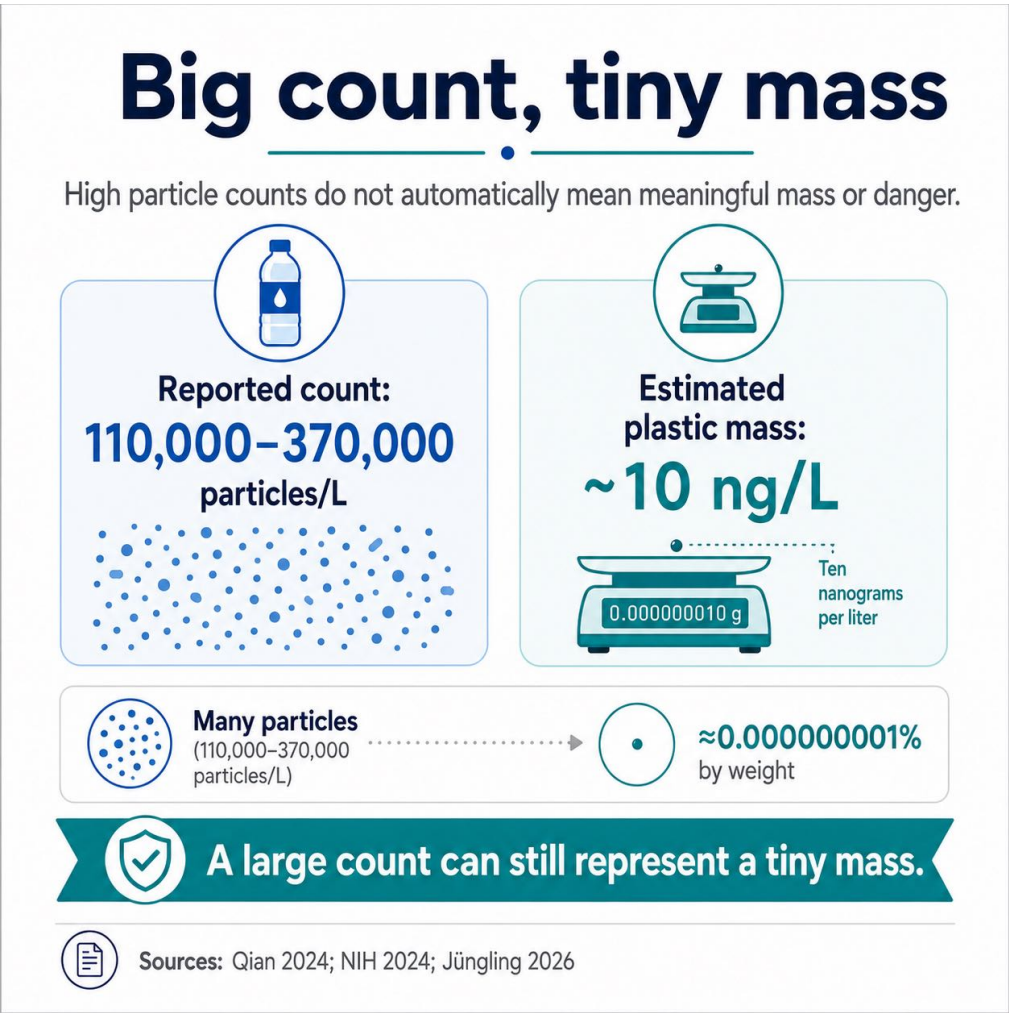
What is the current state of the bottled-water nanoplastics claim?

Bottom line: Recent studies reported very high particle counts, but those counts do not prove danger.

A 2024 study reported roughly 110,000 to 370,000 micro- and nanoplastic particles per liter in three bottled-water brands using stimulated Raman scattering (Qian 2024). That is a particle-count claim, not a health-risk finding. The method is new, the result is an outlier, and the finding has not been independently replicated. Even if

the count were correct, the plastic mass would be around 10 ng/L, or about 0.000000001% by weight, based on converting the reported particle counts and sizes into mass; that is comparable to trace quantities of the same food-contact plastics used to make bottles and caps (Qian 2024; Welle 2018; FDA 2024).

Sources: Qian 2024; NIH 2024; Jüngling 2026; Materic 2024; Qian Reply 2024; Ruggeri 2025



Is bottled water worse than tap water?

Bottom line: Sometimes bottled water reports higher particle counts, but health relevance has not been demonstrated.

Results vary by brand, bottle type, handling, lab method, blank correction, and particle-size cutoff. Reviews of tap and bottled-water studies show that reported occurrence is highly method-dependent and that the literature is not suitable for simple claims that one water source is consistently dangerous or that reported particles establish a health risk (Gambino 2022; WHO 2019; Koelmans 2019). Even if bottled water contains more particles than tap water in some studies, people often consume more tap water than bottled water overall, and no health effect from plastic particles has been demonstrated for either water source. Plastic is the minority of particles in potable water, reported as low as about 0.4% of total particles in one recent analysis (Jüngling 2026). Several studies show that glass bottled beverages can contain substantially more microplastics or other particles than PET-bottled water (Schymanski 2018; Chaïb 2025; Reimann 2010; Krachler 2009). Furthermore, PET has lowest environmental impact for beverage containers (DeArmitt 2025).

Sources: Jüngling 2026; Schymanski 2018; Chaïb 2025; Welle 2018; Reimann 2010; Krachler 2009; DeArmitt 2025; Gambino 2022; Yang 2024

Is glass-bottled water cleaner than plastic-bottled water?

Bottom line: Not necessarily.

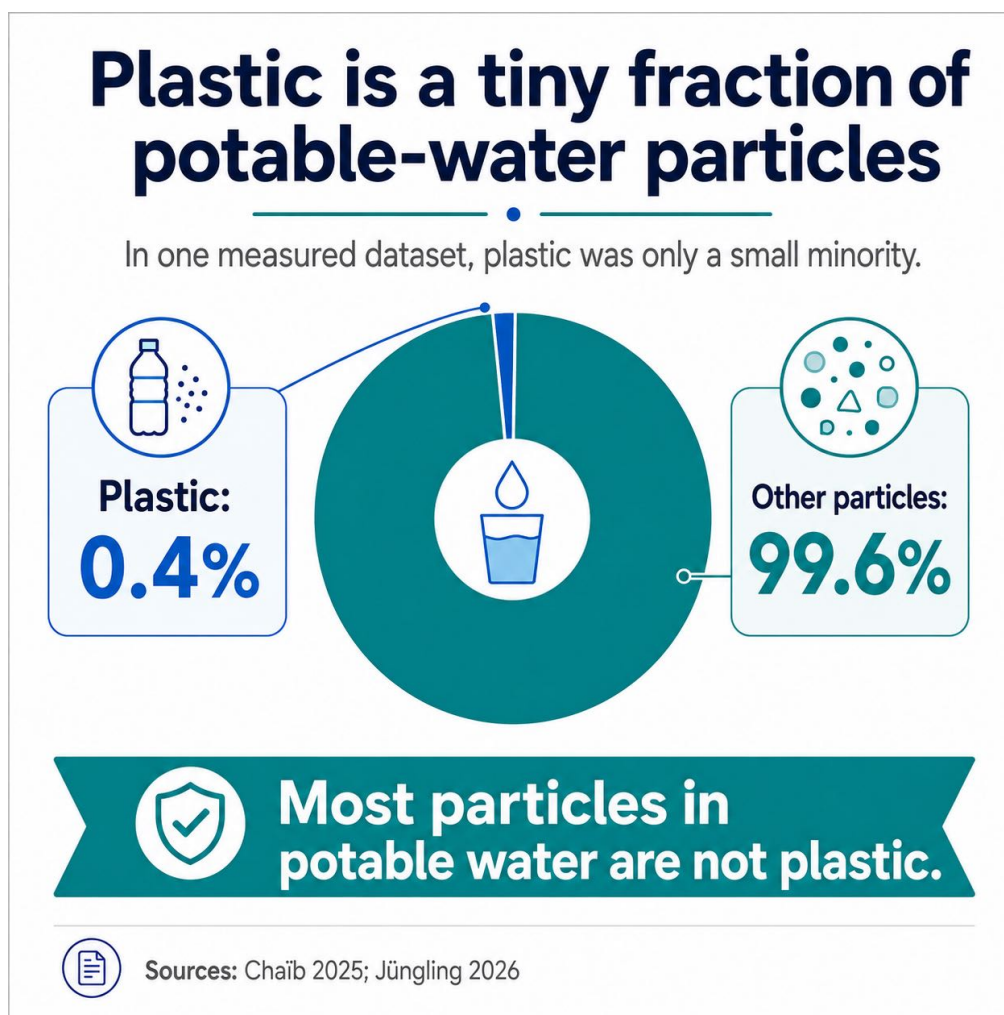
Studies report more particles in glass-bottled beverages than in PET-bottled beverages. Packaging material alone does not determine particle exposure, and particles are not shown to be harmful.

Sources: Schymanski 2018; Chaïb 2025; Reimann 2010; Krachler 2009; Turner 2019

Does tap water contain microplastics?

Bottom line: Some studies report microplastics in tap water, but the amounts are generally small and risk has not been demonstrated because common commodity plastic particles have not been shown to be toxic at realistic exposure levels. Most particles are already removed by coagulation and filtration at water treatment plants. Plastic is the minority of particles in potable water, reported as low as about 0.4% of total particles in one recent analysis (Jüngling 2026). Tap water also contains many non-plastic particles, and reported microplastic levels vary strongly with sampling method, particle-size cutoff, analytical method, blank correction, and polymer-identification criteria (WHO 2019; Koelmans 2019; Yang 2024). A valid risk claim must identify the particles, quantify the dose by mass as well as count where possible, and show harm at realistic exposure. Mere detection is not enough.

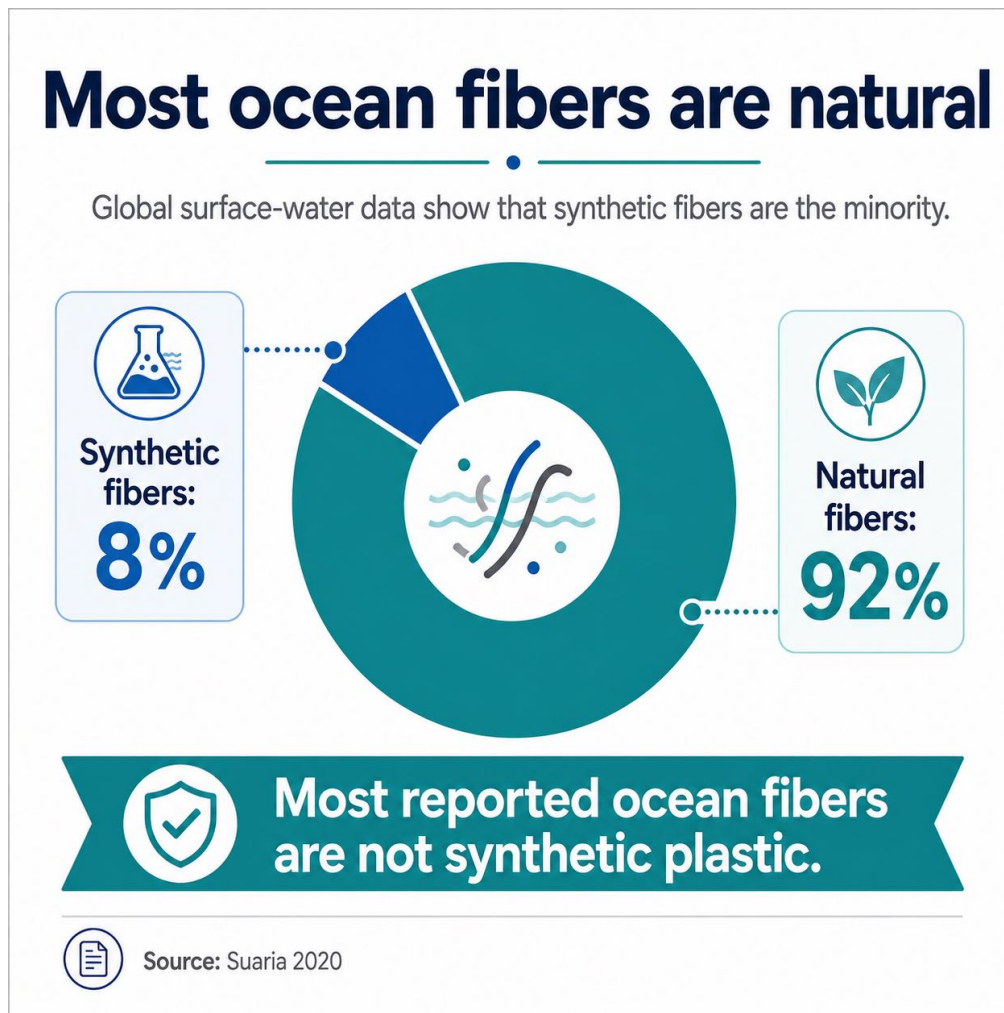
Sources: WHO 2019; EPA Microplastics Research 2026, Jüngling 2026, Brancaleone 2024; Singh 2025, Velasco 2022 & 2023; Pivokonsky 2018; Pivokonsky 2020; Koelmans 2019; Mintenig 2019; Yang 2024



Does ocean water contain microplastics?

Bottom line: Microplastics are present in ocean water, but measured concentrations are generally low and one global assessment characterized currently monitored ecological risk as negligible (Beiras 2020). In a global surface-water fibre dataset, natural cellulosic and animal fibres were about 92% while synthetic fibres were about 8% (Suaria 2020). Those composition data establish relative occurrence, not the toxicity of either natural or synthetic fibres.

Sources: Beiras 2020; Suaria 2020; GESAMP 2016; Cozar 2014; Eriksen 2014



Are microplastics increasing in the ocean?

Bottom line: Claims that ocean microplastics are increasing should be based on measured trends, not assumptions from rising plastic production or modelled leakage.

Measured ocean and wildlife data are variable, and trends differ by region, particle type, sampling method, and time period. One useful long-term indicator is plastic ingestion by seabirds and other marine animals. In *Marine Anthropogenic Litter*, Bergmann, Gutow and Klages summarize long-term seabird studies showing that industrial plastic pellets in seabird stomachs approximately halved from 1980s levels by the early 2000s after attention was paid to pellet losses during production and transport. The same chapter reports that, although plastic ingestion rose from the late 1950s into the 1970s and peaked in some studies around 1985–1995, the trend in plastic consumption decreased and stabilized from 2000 onward, approaching 1980s levels.

That does not mean there is no plastic in the ocean or that litter should be ignored. It means the evidence is more nuanced than the common claim that ocean plastic is simply increasing everywhere. Microplastic trend claims should separate measured data from model projections, should distinguish pellets, fragments, fibers, films, and

fishing-related debris, and should report concentration, mass, particle size, and sampling method. Detection alone is not enough to show a worsening risk trend.

Sources: Bergmann, Gutow & Klages 2015; Van Franeker & Meijboom 2002; Moser & Lee 1992; Robards et al. 1995; Spear et al. 1995; Mrosovsky et al. 2009; Van Franeker et al. 2011; Bond et al. 2013

Are microplastics the main threat to marine animals?

Bottom line: No. Marine animals face many larger and better-proven risks.

Important risks include fishing gear, habitat loss, overfishing, disease, vessel strikes, and water quality.

Microplastics should be kept in proportion to measured exposure and demonstrated effects. The estimated ecological risk from microplastics has been described as negligible in some assessments because measured concentrations are low relative to effect thresholds. (Beiras 2020).

Controlled fish-feeding studies also show why ingestion should not be confused with accumulation or harm. Lu et al. exposed juvenile yellow perch to high-density polyethylene microplastics through feeding and reported no mortality, no apparent signs of significant distress, no adverse effects, and no significant differences in growth performance; no accumulation of HDPE was detected in fish collected 24 hours after feeding. Alomar et al. exposed reared gilthead seabream to low-density polyethylene controlled diets and found that after one month of depuration, no microplastics were found in the gastrointestinal tracts, indicating no long-term retention in the digestive system. Fish-feeding studies are not direct proof of human safety, and they do not mean that all marine debris is harmless. They do show that the mere presence or ingestion of microplastic particles is not the same as demonstrated accumulation, toxicity, or population-level harm. Marine animals face larger and better-proven risks, including entanglement in fishing gear, habitat loss, overfishing, disease, vessel strikes, water-quality problems, and ingestion of large debris. Microplastics should therefore be evaluated by measured exposure, realistic dose, particle type, retention, and demonstrated biological effect, not by the assumption that every ingested particle causes harm.

Sources: Lu 2022; Alomar 2021; FAO 2017; GESAMP 2016; Beiras 2018; Beiras 2019

Are tire-wear particles microplastics?

Bottom line: No. Tire rubber is an elastomer, not a plastic in the classification used here. Tire-wear particles should be evaluated as a separate particle and chemical-exposure category.

Tire particles contain elastomeric rubber, fillers, carbon black, metals, and additives. They should be evaluated separately from polyethylene, polypropylene, PET, and other plastics. Tire-rubber leachate provides a clear example of why particle identity and leached chemicals must be separated: Tian et al. identified 6PPD-quinone (6PPD-Q), an oxidation product of the tire antiozonant 6PPD, as the principal causal toxicant for acute coho-salmon mortality. A subsequent commercial-standard reassessment reported a juvenile coho LC50 of about 95 ng/L. This is strong source-specific aquatic chemical-hazard evidence for tire rubber; it is not evidence that rubber particles themselves have the same toxicity, is not evidence of human-health harm, and is not evidence of plastic or microplastic toxicity.

Sources: Boucher & Friot 2017; Kole 2017; Wagner 2018; OECD 2021; Tian 2021; Tian 2022; McIntyre 2021; DeArmitt 2020

Are paints and coatings a source of microplastics?

Bottom line: Paints and coatings can contribute polymer particles to the environment.

They are different from packaging plastics and should be evaluated separately by chemistry, dose, and exposure route. Mass-based surveys of marine microplastics also indicate that paint and protective coatings can be important contributors. Dibke et al. used Py-GC/MS/thermochemolysis to measure microplastic mass in German Bight waters and reported heterogeneous concentrations and polymer distributions consistent with a potential contribution from marine coatings and ship paint.

Sources: Gaylarde 2021; Turner 2021; Boucher & Friot 2017; GESAMP 2016; Dibke 2021

Do water filters remove microplastics?

Bottom line: Some home filters can remove particles, but performance depends on the filter type and particle size.

Reverse osmosis and membrane systems should remove many particles above their effective pore size. Simple filters vary. No filter should be marketed as a health necessity as there is no evidence of health effects from plastic particles in water. Particles are already removed by coagulation and filtration at water treatment plants.

Sources: Cherian et al. 2023; EPA, Velasco 2022 & 2023; Pivokonsky 2018; Pivokonsky 2020; Velasco 2023; WHO 2019; Cherian 2023

Which water-filter technologies have validated removal data across the full microplastic and nanoplastic size range?

Bottom line: No simple home-filter claim should be accepted unless the filter was tested for the relevant particle sizes.

Water-treatment plants already remove many particles through coagulation, settling, and filtration. Home filters can remove additional particles if their technology and pore size are appropriate, but performance varies by device. There is no scientific reason to present home filtration as a medical necessity for microplastics.

If a household has a real water-quality problem, it should be addressed based on measured contaminants, not fear of plastic particles.

Sources: EPA Lead and Copper Rule; Cherian et al. 2023; Velasco 2022 & 2023; DeArmitt 2023; Velasco 2023; Li 2024; Cherian 2023

Does boiling water remove microplastics?

Bottom line: Boiling does not destroy plastic, but boiling hard water may reduce some particles if scale forms and the water is filtered afterward. There is no scientific or health reason to do so.

A 2024 study reported that calcium carbonate scale formed during boiling hard water can trap some nano- and microplastics. This is not a universal solution. It depends on water hardness, particle type, and filtration after boiling. Kettles can release metals such as nickel, as well as Pb, Ni, Mn, Cr, and Zn, so that should be considered.

Sources: Yu 2024; Berg 2000; Müller 2015

Are nanoplastics more dangerous than microplastics?

Bottom line: There is no evidence showing that normal nanoplastic exposure is dangerous.

Smaller particles are harder to measure accurately. Nanoplastic methods are less mature than methods for larger particles. Claims about nanoplastics should be treated carefully as large numbers of particles can be an insignificant mass of particles.

Sources: WHO 2022; EFSA 2025; Koelmans 2022; Gigault 2021

Do plastic cutting boards release microplastics into food?

Bottom line: Plastic cutting boards can release particles when cut or scraped.

That does not prove danger. The important issue is how much mass is released and whether that realistic dose causes harm. Cutting-board claims should be compared with all other food particles and with hygiene risks from alternative materials.

Sources: IARC 2012; EFSA 2025; Yadav 2023

Are plastic cutting boards dangerous compared with wooden boards?

Bottom line: Danger has not been demonstrated from normal plastic cutting-board use.

A fair comparison must include particle release, food safety, cleaning, bacterial contamination, durability, splintering, and actual exposure. The existence of particles alone does not make plastic boards unsafe. Occupational wood dust has an established human carcinogenic hazard, whereas comparable human carcinogenic evidence has not been demonstrated for ordinary common-polymer plastic dust. Some studies

report antibacterial effects for certain woods, but food-service use also depends on sanitation, surface condition, cleaning method, and regulatory requirements.

Sources: Yadav 2023; IARC 2012; IARC 1995

Do tea bags release microplastics?

Bottom line: Some polymer-containing tea bags can release particles when heated, but this does not imply harm. The risk depends on how much material is released by mass, whether the particles are actually plastic, and whether the dose causes a biological effect. Particle counts alone can exaggerate concern. EFSA reviewed this topic and found methodological errors in some tea-bag studies.

Sources: EFSA 2025; Hernandez 2019

Are black plastic utensils a microplastics issue?

Bottom line: Mostly no. Recent black-plastic concerns are mainly about chemical additives and recycled-material contamination, not microplastic particles.

Chemical-additive questions should be handled separately from microplastic-particle questions. Mixing the two creates confusion.

Sources: Kuang 2018; Liu 2024; FDA 2024; Hahladakis 2018

Does heating plastic containers release microplastics?

Bottom line: Heat and abrasion can increase release from some plastics, but normal use has not been shown to create a microplastic health hazard.

A practical message is simple: follow the product instructions, do not overheat items not intended for heating, and replace badly damaged containers. That is sensible use, not evidence of a proven microplastic disease risk.

Sources: EFSA 2025; Li 2020; Akbulut 2024; Ranjan 2021

Does plastic food packaging make food unsafe?

Bottom line: No. Plastic packaging is not made unsafe by microplastic claims.

Packaging protects food, reduces spoilage, lowers contamination risk, and often reduces environmental impact compared with heavier alternatives. Any claimed particle release must be judged by dose and evidence of harm, not fear of plastic itself. Food packaging and containers must comply with FDA or other regional food contact regulations.

Sources: FDA 2024, EFSA 2025; Welle 2018; Meng 2024; Voulvoulis 2019

Are glass and other alternatives particle-free?

Bottom line: No. Switching away from plastic does not mean switching to a particle-free world.

All solid materials can shed particles during manufacture, use, breakage, abrasion, weathering, or handling. Glass is a clear example. Studies of medical glass ampoules show that opening glass ampoules can introduce glass particles into injectable solutions. Chiannilkuchai and Kejkornkaew reported glass-particle contamination in 449 of 672 ampoules examined, with thousands of particles counted and many in the small-particle size range. Eul Joo et al. reported that 180 tested glass ampoules contained 19,473 glass particles, averaging more than 100 particles per ampoule. Perez et al. described glass ampoules as a high-risk source of particulate contamination because fragments can be introduced when the ampoule is opened and can pass through a needle into a syringe. Yorioka et al. found significantly more small particles after cutting glass ampoules than plastic ampoules for glycyrrhizin injections. These examples are not about ordinary drinking-water exposure, and they do not mean glass is dangerous. They show a narrower but important principle: particles are a property of solid materials and handling processes, not a problem unique to plastics. Therefore, claims about “microplastics” should be compared with the full context, including glass, mineral, metal, cellulose, pigment, and other non-plastic particles.

Sources: Chiannilkuchai 2021; Eul Joo 2016; Perez 2016; Carraretto 2011; Yorioka 2009

Do plastic baby bottles release microplastics, and is that dangerous?

Bottom line: Some studies report particle release from plastic baby bottles under certain preparation conditions. That does not prove harm.

Infant products should be used according to instructions. Any risk claim must compare realistic dose with evidence of biological harm.

Sources: Li 2020; FDA 2024; WHO 2022; EFSA 2025

Do disposable cups, coffee lids, and takeout containers release microplastics?

Bottom line: Heat, abrasion, and repeated use can release small particles from some materials. That does not prove a health hazard.

The key question is the dose by mass and whether it causes harm. Current evidence does not show that normal use of food-contact plastics creates a microplastic health hazard.

Sources: Ranjan 2021; Akbulut 2024; EFSA 2025; FDA 2024

Are compostable or biodegradable plastics safer for microplastics?

Bottom line: Not automatically.

Biodegradable or compostable plastics can still form particles if conditions are not suitable for degradation. “Compostable” does not mean the material disappears instantly in the environment, and it does not automatically mean lower risk.

Sources: Piao 2024; Malafeev 2023; Chamas 2020; ISO 2020; SAPEA 2019

Do microplastics in soil harm crops or soil function?

Bottom line: Current field evidence does not show that realistic microplastic loadings in agricultural soil harm crop yield or key soil microbial functions.

Microplastics can persist in soil, especially where agricultural soils receive organic fertilizers, compost, biosolids, mulch-film residues, or other particle inputs. Persistence, however, is not the same as harm. The relevant question is whether measured or realistic soil concentrations impair crop growth, soil microbial biomass, enzyme activity, carbon cycling, or nutrient function. Schöpfer et al. studied arable soil under field-relevant agricultural conditions and reported that conventional and biodegradable microplastics could persist and accumulate, but current loadings had no detectable immediate negative consequences for soil microbial abundance, microbial activity related to carbon cycling, or crop yield. That is the appropriate distinction: detection in soil may justify monitoring and better source control, but it does not by itself prove ecological damage.

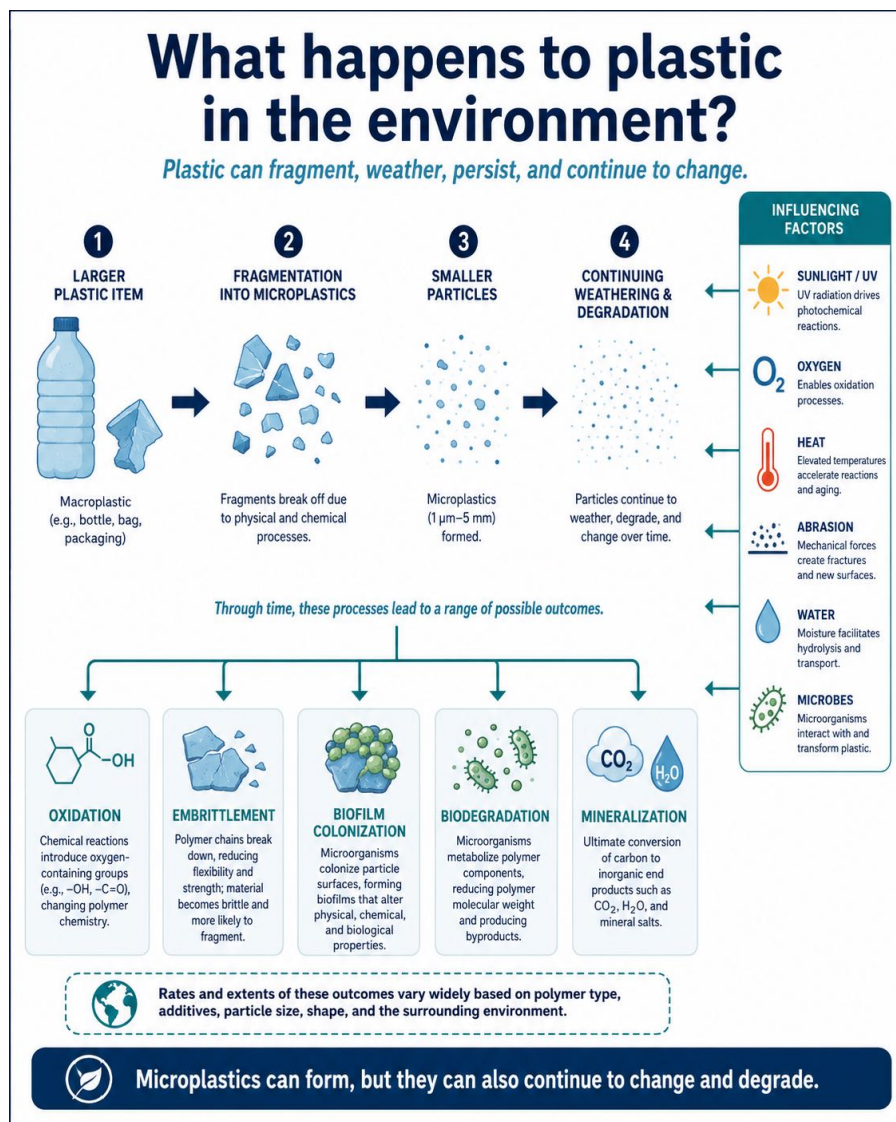
Some laboratory studies report soil effects, but many use simplified systems, high concentrations, artificial particles, or endpoints that do not translate directly into field-level crop loss. Reviews of soil microplastics show that occurrence, sources, and methods remain active research areas, but the evidence should be evaluated by concentration, particle type, exposure duration, soil properties, and real agronomic outcomes. The current evidence supports a proportionate conclusion: soil microplastics are worth measuring and reducing where practical, especially to avoid unnecessary contamination, but realistic soil levels have not been shown to be a major demonstrated cause of crop-yield loss or soil-function collapse.

Sources: Schöpfer 2022; Yang 2021; Li 2021

Do plastics degrade into microplastics, and do microplastics degrade further?

Bottom line: Yes. Plastics can fragment into microplastics, and microplastics can continue to chemically, physically, and biologically degrade. The rate depends strongly on polymer type, additives, oxygen, sunlight, heat, moisture, abrasion, surface area, microbes, and the surrounding environment.

Plastic degradation is not a single process. Weathering by sunlight, oxidation, heat, mechanical abrasion, hydrolysis, and microbial activity can weaken larger plastic items and cause fragmentation into smaller particles. For example, Weinstein et al. found that high-density polyethylene, polypropylene, and polystyrene degraded in salt-marsh habitat and concluded that surface delamination was a primary mechanism by which microplastic particles were produced during early degradation. Expanded polystyrene can also fragment rapidly outdoors under sunlight, producing micro- and nanoplastic particles. This means that macroplastic litter can become microplastic, just as other solid materials can fragment into dust and particles as they age.



The process does not necessarily stop at the microplastic stage. Microplastics have higher surface area than larger items and can continue to oxidize, crack, embrittle, fragment, and be colonized by microorganisms. Polyethylene and polypropylene are often described as persistent, but persistence does not mean permanent. Reviews and experimental studies report biodegradation or biodeterioration of polyethylene and polypropylene by bacteria, fungi, and mixed microbial communities, usually slowly and often more readily after weathering or other pretreatment. Jeon et al. reported long-term biodegradation of buried low-density polyethylene in soil, Arutchelvi et al. reviewed biodegradation evidence for polyethylene and polypropylene, Jeon et al. later reported biodegradation of polyethylene and polypropylene by a soil *Lysinibacillus* strain, and Vaksmaa et al. reported biodegradation and mineralization of polyethylene by a marine fungus after photodegradation.

The correct conclusion is therefore balanced: plastic items can fragment into microplastics, and microplastics can degrade further, but rates vary widely. Degradation can be fast for some materials and conditions, such as sunlight-exposed expanded polystyrene or biologically active environments, and slow for others, especially buried, cold, dark, oxygen-limited, or stabilized plastics. Environmental persistence is a reason to reduce unnecessary litter and improve waste management. It is not proof that every plastic particle is permanent, accumulating forever, or biologically harmful at realistic exposure levels.

Sources: Weinstein 2016; Jeon 1995; Arutchelvi 2008; Jeon 2021; Vaksmaa 2024; Kyoung 2020; Restrepo-Flórez 2014; Mohanan 2020

Do plastic pipes release microplastics into drinking water?

Bottom line: Real-world source-to-tap studies do not support the claim that PE or PVC pipe systems are generally a major net source of microplastics in household tap water. Pipes can shed particles under some abrasion, installation, ageing or abnormal conditions, so “never releases” would be too strong.

A useful example is the Danish tap-water study by Feld et al. The authors sampled drinking water from 17 geographically widespread locations in Denmark, including sites with different water abstraction areas, waterworks, groundwater ages, aquifer geology, and pipe-network materials. The supplementary site information shows that many of the sampled supply networks included plastic pipes, especially PE and PVC, often alongside other pipe materials.

Despite that, the study did not identify PE or PVC particles in the tap water. Across all samples, confirmed plastic particles were rare and consisted of PET, PP, PS, and ABS. For particles larger than 100 µm, all samples were below the limit of detection, and the observed MP-like particles could not be distinguished from background contamination. Most visually suspected particles were not plastic at all; µ-FTIR showed that most were cellulose, with some proteinaceous material and poor or unknown spectra. For the smaller-particle analysis down to 10 µm, only three MP fragments were identified in the subset tested: PET, PP, and ABS. Again, no PE or PVC particles were reported.

This matters because plastic pipes are often treated as an assumed major source of drinking-water microplastics. Feld et al. tested actual tap water from real Danish distribution systems, including systems containing PE and PVC pipe, using contamination controls and chemical confirmation. The result does not prove that plastic pipes can never shed particles under any condition, but it does show that PE and PVC particles were not confirmed in those sampled systems at measurable levels.

The more accurate conclusion is that real-world pipe contribution is condition- and system-specific. Source-to-tap measurements can show very low plastic-particle levels or even net particle removal during distribution, while abrasion, cutting, installation or ageing can create release under other conditions. A source claim therefore requires direct source-to-water evidence rather than assuming that the presence of plastic pipe proves that detected particles came from the pipe.

Sun et al. provides an important supply-chain test of the claim that drinking-water pipes are a major source of microplastics. The study measured microplastics in raw water, treated water leaving drinking-water treatment plants, and household tap water after distribution through the pipe network. If the pipe network were a major net source of plastic particles, household water would be expected to contain more particles than the treated water entering the distribution system.

The opposite pattern was reported. Treated water contained more microplastics than household water, and the authors described the lower household-water concentrations as evidence of non-targeted removal during distribution, possibly because particles adsorb to pipe scale. In other words, the distribution system did not act as a net source of microplastic particles in this study; it appeared to reduce measured particle counts between the treatment plant outlet and household taps.

This does not prove that pipes can never release particles under any condition, and it does not identify the exact fate of each particle. However, it directly contradicts the simple claim that plastic drinking-water pipes

necessarily add measurable microplastics to household tap water. In this real-world supply system, fewer particles came out of the distribution network than entered it.

Water-treatment and distribution systems contain many particle sources. The relevant comparison is total particle exposure and toxicity. Copper and metal pipe corrosion can release substances with known toxicity; plastic pipe particles have not been shown to create a health risk at normal exposure.

Sources: Świetlik & Magnucka 2024; WHO 2019; WHO 2022; Koelmans 2019; Feld 2021; Sun 2024

Do drinking-water pipes release only plastic particles?

Bottom line: No. Drinking-water distribution systems can generate and release many kinds of non-plastic particles, including mineral scale, corrosion products, metals, organic deposits, and biofilm-associated material.

Particles in tap water should not automatically be assumed to come from plastic pipes or plastic packaging. Drinking-water pipes are chemically and biologically active surfaces. Over time, pipe walls can develop scale deposits, corrosion layers, mineral precipitates, organic films, and biofilms. These deposits can act as both sinks and sources for particulate material in water. Wiercik et al. characterized scale deposits in drinking-water pipes using FTIR and ICP-OES and reported that such deposits contain inorganic and organic components, including mineral and metal-associated material. This reinforces a basic point for microplastics interpretation: when particles are found in tap water, they may be mineral scale, pipe corrosion products, metal oxides, biofilm-associated material, fibers, or other non-plastic debris. Polymer identification is therefore essential before calling particles “microplastics.”

This matters because many public claims treat particles in drinking water as if they are automatically plastic and automatically hazardous. That is not scientifically valid. Tap-water particles must be identified by composition, size, morphology, and source. Plastic may be present in some samples, but drinking-water systems also produce non-plastic particulate matter through normal scale formation, corrosion, precipitation, and biofilm processes. The correct conclusion is not “pipes release plastic”; it is that drinking-water systems are complex particle environments, and plastic identity must be proven rather than assumed.

Sources: Wiercik 2026; Makris 2014; González 2022; WHO 2019; Feld 2021

5. Dust, Textiles, and Exposure Reduction

Are plastic particles a major factor in the context of dust as a whole?

Bottom line: No. Dust is a mixture, and plastic should be judged in the context of the whole particulate exposure, not an isolated special category. Airborne and settled dust contain mineral dust, crustal material, combustion particles, soot, road-wear particles, textile fibers, skin fragments, pollen, cellulose, metals, salts and many other materials. Plastic is a small component, with the exact fraction depending on setting and analytical method.

Particulate matter is a well-established health concern, but the major recognized sources are not plastic; global reviews identify crustal matter, road traffic and fuel combustion as major particulate-matter sources. The hazard is also not evenly distributed across particle types. Respirable crystalline silica, asbestos, occupational wood dust and certain metal-containing particles have established serious health hazards, while ordinary environmental plastic dust has not been shown to cause human disease at realistic exposure. Hayden et al. 2023 reinforces the need for this context: 124,018 inorganic particles spanning 45 classes were characterized in only 38 selected lung biopsies, including mineral and metal-rich particles. Hayden cannot provide a plastic/non-plastic denominator because the FE-SEM/EDS method preferentially detects inorganic material, but it demonstrates why a plastic-only interpretation of lung particles is scientifically incomplete.

Concern should be proportional to the dose, composition and toxicity of the whole particle mixture. There is no scientific or logical justification for treating ordinary plastic dust as a special case unless it is shown to be unusually abundant or unusually hazardous. Current evidence shows neither at normal environmental exposure.

Sources: Mukherjee 2017; Heindel 2020; Gustafsson 2018; Hayden 2023; IARC 1995; IARC 2012

Are synthetic clothes a major source of microplastics?

Bottom line: Synthetic clothes can shed fibres, but that does not by itself imply a health risk. The available studies do not establish a textile-fibre-specific human toxicity threshold.

Fibres from clothing, carpets, furniture, and textiles can be found in dust and air, and natural fibres also shed. Cotton and wool have been reported to shed about 10-30 times more fibres than polyester under some test conditions (De Falco 2020; Licina 2019). Merski tested cryogenically milled PE/PET spunbond fabric by oral feeding, not inhaled textile fibres, and Stock used short-term cell systems. Those studies provide relevant polymer context but do not establish a no-harm inhalation level for every PET fibre. No harm from ordinary textile-fibre exposure has been demonstrated.

Sources: FDA 2024; EFSA 2025; Merski 2008; Stock 2021; Suaria 2020; De Falco 2020; Licina 2019; Napper 2016

Do natural fibers like cotton and wool also shed particles?

Bottom line: Yes. Natural textiles shed fibers too.

People are exposed to cellulose, cotton, wool, skin flakes, food debris, and many other non-plastic particles. A scientific comparison should include all particles, not just plastic particles. Cotton and wool textiles have been reported to release about 10-30x more fibers than synthetic polyester under some test conditions (De Falco 2020; Licina 2019). This helps explain why one global ocean-fiber study found only about 8% synthetic fibers and about 92% natural fibers in oceanic surface waters (Suaria 2020).

Sources: Suaria 2020; De Falco 2020; Licina 2019; Athey 2022

What are the main sources of human microplastic exposure?

Bottom line: Likely sources include indoor dust, air, food, drinking water, packaging-related particles, textiles, and household abrasion.

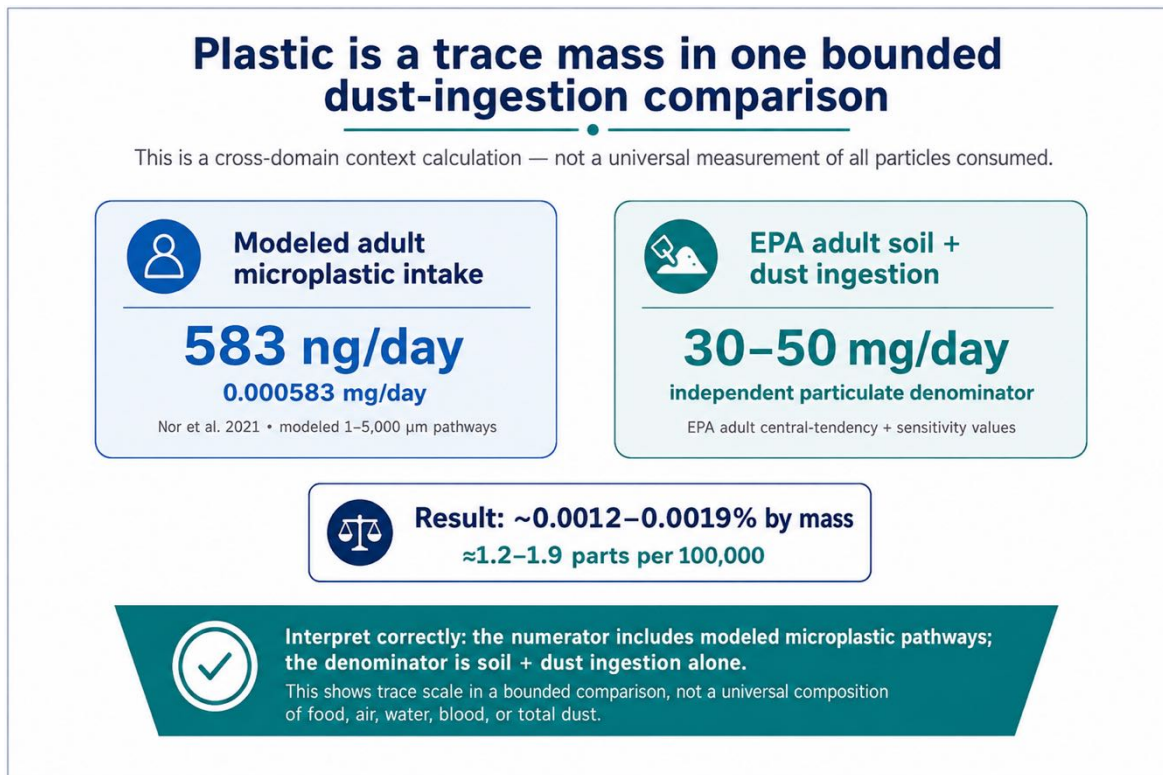
The size of each contribution is still somewhat uncertain because studies use different methods and size cutoffs. Exposure should be described by mass as well as particle count.

Sources: Mohamed Nor 2021; WHO 2022; Cox 2019; Eberhard 2024

Do people get more microplastics from food, water, or air?

Bottom line: The answer is still uncertain and depends on the method used.

Mohamed Nor et al. modeled about 583 ng/day of adult microplastic intake over its included pathways and 1–5000 µm particle domain. As a separate contextual comparison, 583 ng/day is about 0.0012–0.0019% of the U.S. EPA adult soil-plus-dust ingestion values of 30–50 mg/day - roughly 1.2–1.9 parts per 100,000 by mass. This is a bounded cross-domain comparison, not a measured universal fraction of all particles consumed: the microplastic numerator and soil-plus-dust denominator are independently derived and cover different pathways. Same-sample indoor, fibre and potable-water studies nevertheless also place plastic as a minority component of the broader particle background.



Sources: Mohamed Nor 2021; U.S. EPA soil/dust ingestion factors; Cox 2019; Eberhard 2024; Catarino 2018

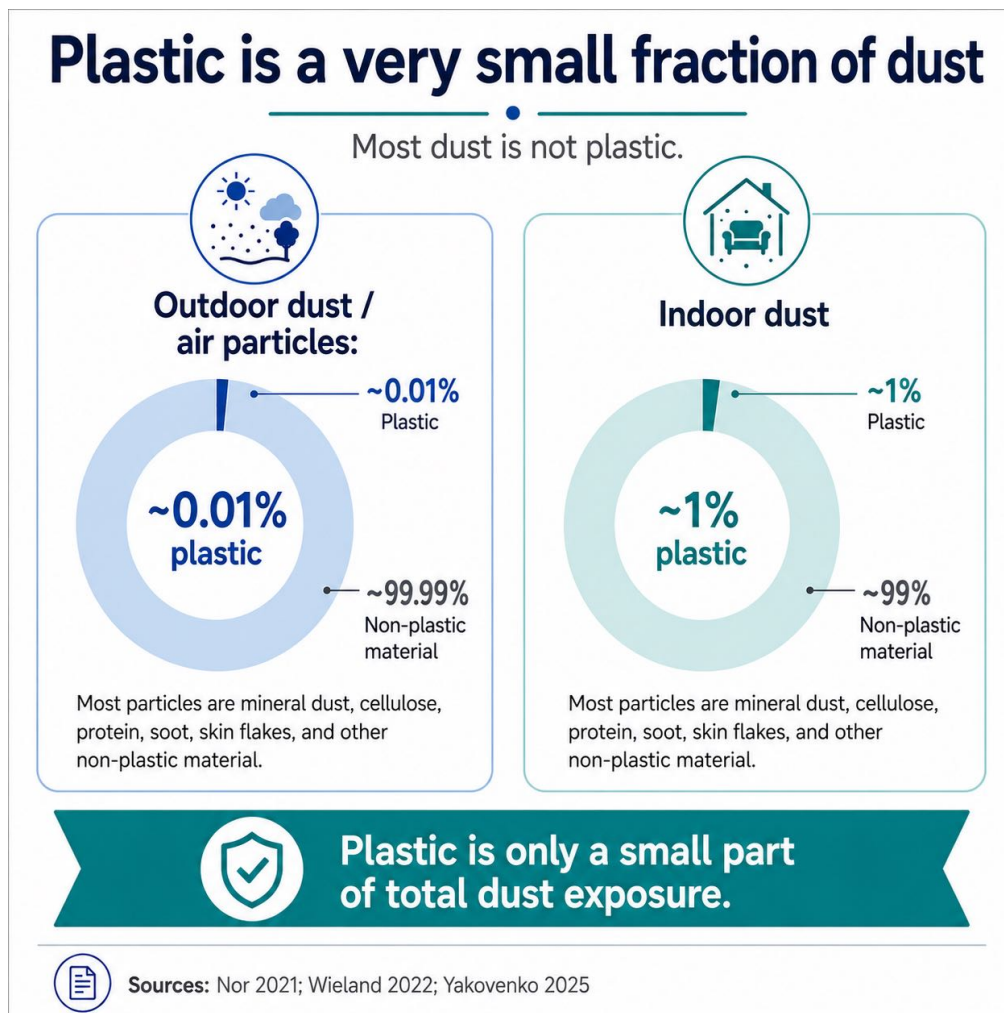
How much reported indoor and outdoor dust is plastic compared with cellulose, mineral dust, soot, skin particles, and other material?

Bottom line: Plastic appears to be a very small fraction of the total particles people breathe.

Outdoor plastic particles have been estimated to be extremely small compared with total dust exposure, around 0.01% of the total in some exposure comparisons (Mohamed Nor 2021; Eberhard 2024). Studies also show that most airborne and settled particles are not plastic (Vianello 2019; Yakovenko 2025; Wieland 2022). A fair risk comparison must include mineral dust, quartz, soot, cellulose, skin particles, natural fibers, food particles, and other debris. For indoor dust, plastics have been reported as a small fraction of total material, often around 1% or a few percent depending on the study and classification method (Vianello 2019; Khalid Ageel 2025; Yakovenko 2025). One study found plastic at about 4% of fragments and fibers, compared with about 91% proteinaceous material and about 4% cellulose (Wieland 2022).

This matters because some non-plastic particles are known hazards at sufficient exposure, while ordinary plastic dust has not been shown to cause human disease at realistic environmental levels.

Sources: Mohamed Nor 2021; IARC Monographs on crystalline silica; Ogonowski 2018; Vianello 2019; Khalid Ageel 2025; Yakovenko 2025; Wieland 2022; Tan 2016; Dris 2016; Prata 2020



How do microplastics compare with other particles people inhale or ingest?

Bottom line: Plastic is only a small fraction of the particles people encounter.

People inhale and ingest mineral dust, cellulose, cotton, wool, skin flakes, food particles, soot, pollen, pigments, and other debris. Some non-plastic particles, such as crystalline silica or wood dust, have known hazards at sufficient exposure. Normal environmental plastic-particle exposure has not been shown to cause human disease.

Sources: Vianello 2019; Wieland 2022; Salthammer 2022; Yakovenko 2025; OSHA 2023; IARC 2012

How does pollen exposure compare with airborne plastic particles?

Bottom line: Pollen is a useful real-world benchmark because it overlaps in size with many coarse airborne microplastic particles, can occur at comparable or much higher airborne concentrations, and has proven health effects in susceptible people. By contrast, no adverse human health effect has been proven from ordinary environmental exposure to plastic particles at realistic doses.

Intact pollen grains commonly fall in the broad coarse-particle range of about 10–100 μm , overlapping with many larger airborne microplastic fibers and fragments. During pollen season, airborne pollen concentrations commonly reach tens to hundreds of grains per cubic meter and can reach thousands of grains per cubic meter during peak episodes. In an Atlanta field study, average daily total pollen concentrations across three sites ranged from 281 to 561 grains/ m^3 , and peak pollen counts exceeded 5,900 to 8,800 grains/ m^3 depending on site (Jiang 2022). These concentrations are not unusual in pollen-exposure terms; some pollen-count systems classify tree pollen above about 90 grains/ m^3 as high and above about 1,500 grains/ m^3 as extremely high.



Pollen vs. Plastic Particles in Air

Exposure levels and proven health effects in the coarse airborne size range (~10–100 μm)



Feature	Pollen particles	Plastic particles / microplastics	Scientific implication
 Typical size range	Intact pollen grains are commonly about 10–100 μm , depending on species.	Airborne microplastics also occur in this broad size range, especially larger fibers and fragments.	Size overlap means being airborne or inhalable is not plastic-specific.
 Typical concentration	Often tens to hundreds of grains/ m^3 during pollen season; can rise much higher during peaks.	Reported airborne concentrations vary widely, often from single digits to hundreds of particles/ m^3 depending on method and setting.	Pollen exposure can equal or exceed plastic-particle exposure in the same size range.
 Peak measured levels	Peak pollen levels can reach thousands of grains/ m^3 . Example: <i>Quercus</i> pollen reached 3,793 grains/ m^3 in Atlanta.	Indoor studies have reported concentrations such as 58–684 particles/ m^3 , with a median of 212 particles/ m^3 in one study.	Peak pollen concentrations can be comparable to, or far above, many airborne microplastic measurements.
 Estimated daily inhalation	At 100 grains/ m^3 : ~1,000–2,000 grains/day. At 1,000 grains/ m^3 : ~10,000–20,000/day. At 3,793 grains/ m^3 : ~38,000–76,000/day.	At 10 particles/ m^3 : ~100–200/day. At 212 particles/ m^3 : ~2,120–4,240/day. At 684 particles/ m^3 : ~6,840–13,680/day.	On high pollen days, natural particle exposure can exceed comparable plastic-particle exposure by 1–3 orders of magnitude.
 Surface chemistry	Often partly hydrophobic due to sporopollenin, waxes, lipids, and pollenkitt.	Many plastic particles are also hydrophobic or partly hydrophobic.	Hydrophobicity is not unique to plastic and does not by itself imply toxicity.
 Biological activity	Biologically active; carries proteins, enzymes, lipids, and allergenic molecules.	Most common plastic polymers are comparatively inert solids, though additives and contaminants may need case-specific evaluation.	Pollen is biologically active in a way ordinary plastic dust generally is not.
 Proven health effects	Yes. Pollen causes allergic rhinitis / hay fever, allergic conjunctivitis, and can trigger asthma attacks in susceptible people.	No adverse human health effect has been proven from ordinary environmental exposure at realistic doses.	This is the key contrast: pollen is a proven health-relevant particle; plastic-particle harm at real-world exposure remains unproven.
 Best interpretation	A common natural airborne particle with proven effects in susceptible individuals.	A small subset of ordinary airborne and settled particles; detection is not proof of harm.	The correct risk question is whether realistic exposure causes reproducible biological harm.
 Key takeaway Pollen is a useful benchmark. It shares many of the same broad particle properties often attributed to microplastics — similar size range, airborne transport, inhalation, and hydrophobicity — yet pollen is a proven cause of allergy and asthma in susceptible people, whereas no adverse human health effect has been proven from ordinary environmental exposure to plastic particles at realistic doses.			



Illustrative comparison based on published exposure ranges; measured values vary by species, season, environment, and analytical method.

Airborne microplastic measurements are much more method-dependent. One indoor personal-exposure study reported average microplastic concentrations of 4.8 ± 1.6 MPs/ m^3 in houses, 4.2 ± 1.6 MPs/ m^3 in workplaces, 5.8 ± 1.9 MPs/ m^3 in subways, and 17.3 ± 2.4 MPs/ m^3 in buses, with most fibers and fragments below 100 μm (Torres-Agulló 2022). A later indoor study using Raman microspectroscopy reported higher indoor airborne microplastic concentrations of 58–684 MPs/ m^3 , with a median of 212 MPs/ m^3 , but also found that microplastics represented only 0–1.6% of non-plastic particles in the same environments (Maurizi 2024).

Using a simple adult inhalation range of 10–20 m^3 of air per day, the comparison is clear. At 100 pollen grains/ m^3 , a person may inhale roughly 1,000–2,000 pollen grains/day. At 1,000 grains/ m^3 , the estimate becomes 10,000–20,000 grains/day. At 3,793 grains/ m^3 , the estimate becomes roughly 38,000–76,000 grains/day. By comparison, airborne microplastic concentrations of 10 MPs/ m^3 correspond to about 100–200 particles/day, while 212 MPs/ m^3 corresponds to about 2,120–4,240 particles/day, and 684 MPs/ m^3 corresponds to about 6,840–13,680 particles/day. These are illustrative calculations, not universal exposure values, because both pollen and microplastic concentrations vary with location, season, weather, indoor activity, sampling method, and particle-size cutoff.

The health-effect contrast is more important than the particle count. Pollen is not merely a detectable natural particle. It is a proven biological allergen. In susceptible people, breathing pollen can cause allergic rhinitis, allergic conjunctivitis, and asthma attacks; CDC states that pollen exposure can cause hay-fever symptoms, allergic conjunctivitis, and asthma attacks when pollen is an asthma trigger. Allergic rhinitis is an IgE-mediated response to inhaled allergens and often co-occurs with asthma and conjunctivitis (Bousquet 2020).

This makes pollen a strong benchmark for interpreting microplastic claims. Pollen shares many properties often used to imply concern about microplastics: it is airborne, inhalable, environmentally widespread, partly hydrophobic, biologically interactive, and within the same broad size range as many coarse airborne particles. Yet pollen is the particle with proven health effects in susceptible people, while ordinary environmental plastic-particle exposure has not been shown to cause human disease. Therefore, the mere fact that plastic particles are detectable, airborne, hydrophobic, or inhalable is not evidence of hazard. The valid risk question is whether

realistic exposure causes reproducible biological harm beyond the much larger background of ordinary non-plastic particles.

Sources: Jiang 2022; Torres-Agulló 2022; Maurizi 2024; CDC 2024; Bousquet 2020; WHO 2022; FDA 2024; Vianello 2019; Wieland 2022; Salthammer 2022; Yakovenko 2025

Are natural particles automatically safer than plastic particles?

Bottom line: No. Natural does not automatically mean safe.

Natural does not automatically mean safe, and synthetic does not automatically mean dangerous. Respirable crystalline silica (quartz), asbestos and occupational wood dust have established carcinogenic hazards; certain metal-containing particles can have serious systemic toxicity; soot and combustion particles have well-established health effects; and pollen has proven allergic effects in susceptible people. Comparable human hazards have not been demonstrated for ordinary common-polymer plastic dust at realistic environmental exposure. Risk depends on dose and material-specific toxicity, not whether a particle is natural, synthetic or plastic.

Pollen is a clear example: it is natural, airborne, partly hydrophobic, and biologically active, and it has proven health effects in susceptible people, whereas ordinary environmental plastic-particle exposure has not been shown to cause human disease.

Sources: IARC 1995; IARC 2012; OSHA silica guidance; WHO 2022; FDA 2024; Wieland 2022

Are microplastics an indoor-air issue or an outdoor-air issue?

Bottom line: Most personal airborne-particle exposure occurs indoors because people spend most of their time indoors.

Indoor dust contains many particle types, including textile fibers, skin flakes, cellulose, mineral dust, and a small fraction of plastic. Reducing total dust is more rational than focusing only on plastic.

Sources: EPA Indoor Air Quality; Salthammer 2022; Vianello 2019; Eberhard 2024; Prata 2018

What should the public do to reduce microplastic exposure?

Bottom line: If exposure reduction is desired, reduce total dust rather than targeting the plastic fraction alone. Ordinary cleaning and ventilation can reduce mixed particulate exposure. There is no evidence-based medical need to organize everyday life around avoiding trace plastic dust specifically.

Reasonable steps include using normal ventilation and cleaning to reduce dust, but choosing water filtration only when it has validated performance. These steps should not be considered medical necessities.

Sources: WHO 2022; FDA 2024; EPA Indoor Air Quality; Salthammer 2022; Koelmans 2022

Which public exposure-reduction actions measurably reduce dose rather than merely reduce anxiety?

Bottom line: The most credible actions are the ones that reduce total dust and unnecessary abrasion.

Routine cleaning, ventilation, and sensible food-contact use are more defensible than fear-based rules such as avoiding all plastic. Many claimed exposure-reduction steps have not been shown to meaningfully reduce dose or improve health. Recommendations should be practical, measurable, and proportional to evidence.

Sources: WHO 2022; FDA 2024; EPA Indoor Air Quality; EFSA 2025; Cherian 2023

Are exposure-reduction products or detox claims legitimate?

Bottom line: Detox claims are not supported by evidence.

There is no credible "microplastic cleanse." Fear-based marketing is not science. There is no medical reason to undertake any procedure claimed to remove particles, and every medical procedure entails risk.

Sources: FDA 2024; WHO 2022; FTC consumer-health marketing principles; Koelmans 2022

6. Methods, False Positives, and Evidence Quality

Are most microplastics studies reliable?

Bottom line: Some are useful, but many contain major limitations.

Common weaknesses include contamination, poor blank correction, uncertain particle identity, unrealistic exposure, weak controls, and conclusions that go beyond the data. This is why headlines can be misleading. A weak detection study should not become a health-risk headline. Before trusting a claim, check whether the study proved plastic identity, used realistic exposure, and actually showed harm.

Sources: Gouin 2022, Koelmans 2019; Cowger 2020; Hermesen 2018

What are the biggest weaknesses in microplastics research?

Bottom line: The biggest weaknesses are false positives, contamination, unrealistic doses, and making claims unsupported by the science.

The public usually sees the headline, not the method. A study may report "plastic" when it only found a dye like Nile Red, a tentative spectral match, or a chemical signal that could come from something else.

Sources: Gouin 2022, Koelmans 2019, Lenz 2016; EFSA 2025; Cowger 2020; Hermesen 2018

Method hierarchy for microplastics evidence

Use this table to distinguish screening methods from methods that can support stronger claims about intact particles, polymer identity, dose, and risk. Because microplastic claims depend heavily on analytical method quality, the table below summarizes what common methods can and cannot prove.

Method	What it can show	Main limitation	Evidence value in this review
Visual microscopy	Particle shape, color, approximate size, and morphology.	Cannot prove that a particle is plastic; false positives are common without chemical confirmation.	Useful for screening only. Not sufficient by itself for plastic identity.
Nile Red staining	Hydrophobic particle screening and approximate particle counts.	Can stain non-plastic hydrophobic material and generate false positives.	Weak unless followed by chemical confirmation.
μFTIR / FTIR imaging	Polymer identification for particles within the method size range; can preserve particle size and shape information.	Limited for very small particles; results depend on spectral quality, library match, blanks, and contamination control.	Stronger when paired with good QA/QC, blanks, recovery checks, and transparent match criteria.
Raman spectroscopy	Polymer identification and particle morphology, often at smaller sizes than FTIR.	Fluorescence interference, spectral-match uncertainty, contamination, and declining reliability for very small particles.	Useful when method limits and match criteria are explicit; not automatically definitive for submicron particles.
LD-IR	Automated particle detection and polymer identification over defined size ranges.	Still depends on size cutoff, spectral library, sample preparation, blanks, and match thresholds.	Useful for supply-chain or survey studies when QA/QC is clear.
Pyrolysis-GC/MS	Polymer-associated chemical fragments and approximate polymer mass in a bulk sample.	Destroys the sample and does not prove intact particles, particle number, particle size, or tissue location.	Useful for mass context, but not proof of intact microplastic particles by itself.
Electron microscopy / SEM-EDS	Particle morphology, surface features, and elemental composition.	Does not identify most organic polymers without complementary spectroscopy or chemical analysis.	Useful supporting evidence, not sufficient alone for polymer identity.
Mass-based exposure modeling	Estimated exposure dose and plausibility of reported body burdens.	Depends on input data quality and assumptions; may not capture every individual scenario.	Essential for checking whether particle-count claims fit realistic dose and mass constraints.

Why can Nile Red staining give false positives?

Bottom line: Because Nile Red can stain non-plastic hydrophobic material.

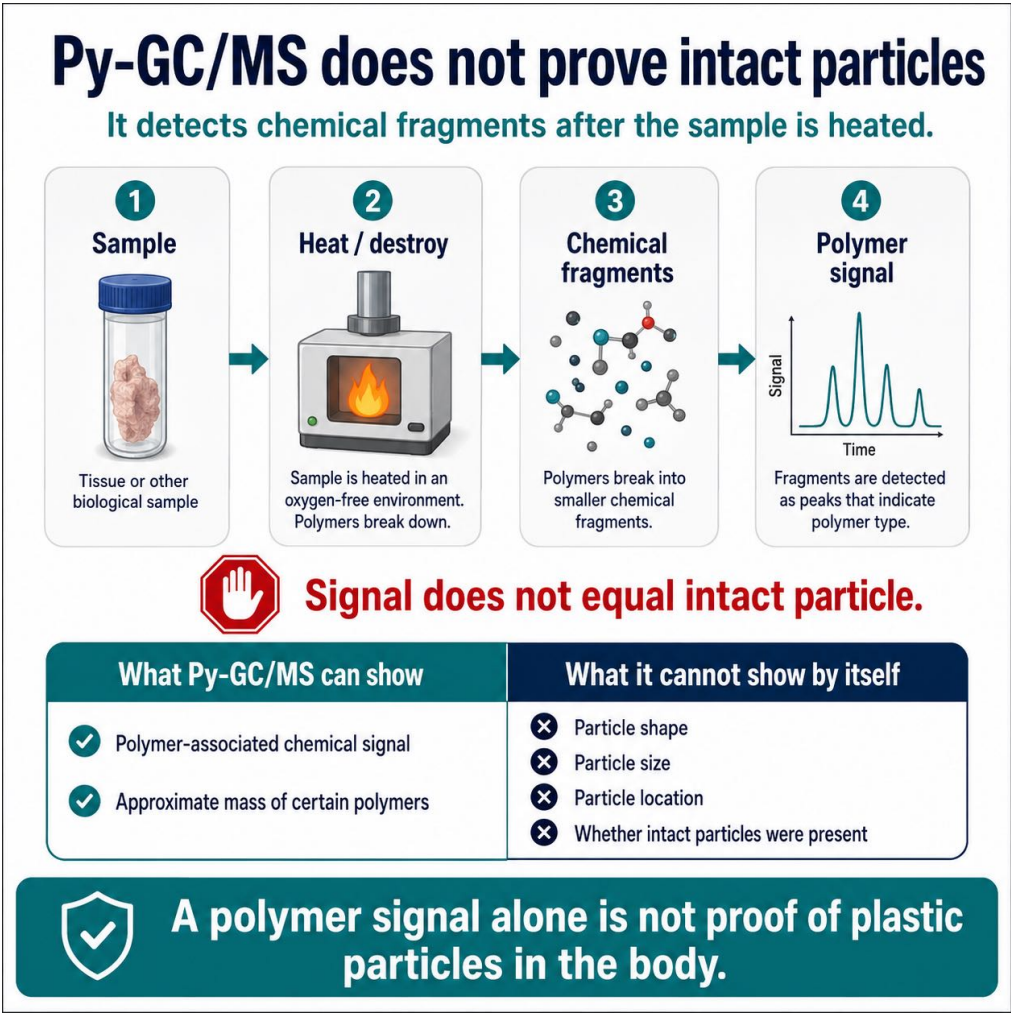
A bright stained particle is not proof of plastic. Oils, biological material, and other hydrophobic substances can give misleading results. Nile Red should not be treated as definitive identification by itself. Studies based on it have been shown to massively overestimate plastic amounts. A 2026 study found 1,891 particles/m³ using Nile Red dye but only 161 confirmed MP/m³ from μ Raman, 1 μ g/m³ from Py-GC/MS, and 0 particles using a μ FTIR workflow (Aves 2026).

Sources: Aves 2026, Michelaraki 2020, EFSA 2025, Stanton 2019; Maes 2017

Why is pyrolysis-GC/MS not proof of plastic particles?

Bottom line: Py-GC/MS does not prove intact plastic particles by itself, because it detects chemicals formed at high temperature rather than showing the original particle.

Sources: Aves 2026, Rauert 2025; Li 2024; Brits 2024; Rauert 2022; Almeida 2026



Can Raman spectroscopy identify very small particles reliably?

Bottom line: Raman becomes difficult for very small particles, especially below about 1 micrometer.

Small particles produce weak signals and are harder to distinguish from background material. Claims about nanoplastics or sub-micron particles need especially strong validation.

Sources: EFSA 2025; Li 2024; Jüngling 2026; Qian 2024; Meyns 2023

Can FTIR identify microplastics reliably?

Bottom line: FTIR can be useful for larger particles, but it has size limits and can misidentify difficult samples. Reliability depends on particle size, sample preparation, spectral quality, library matching, and operator decisions. FTIR results should be supported by good blanks and transparent quality criteria.

Sources: EFSA 2025; Jüngling 2026; Koelmans 2019; Meyns 2023

How can contamination create false detections?

Bottom line: Plastic fibers and dust are common in labs, air, clothing, equipment, and sample handling.

Without strong procedural blanks and clean handling, a study may measure contamination introduced during collection or analysis instead of particles from the original sample.

One study cast doubt on prior studies by showing that gloves used in the laboratory shed a substance (lubricant) that is then detected and mistaken for plastic particles. They listed many studies that may be invalid because of this newly identified source of contamination.

Sources: Clough 2026; Koelmans 2019; Cowger 2020; Hermesen 2018

Why can solubles or precipitates create false positives?

Bottom line: Because dissolved substances can come out of solution and form particles during processing.

Those particles may look like microplastics or interfere with staining and spectroscopy. A study must prove the final particles are plastic, not precipitated salts, proteins, fats, residues, or other non-plastic material. This form of error applies in particular to studies on hot drinks.

Sources: EFSA 2025; Rauert 2025; Ranjan 2021

Does particle charge make microplastics or nanoplastics uniquely dangerous?

Bottom line: No. Surface charge is not unique to plastic particles, and charge alone does not prove risk.

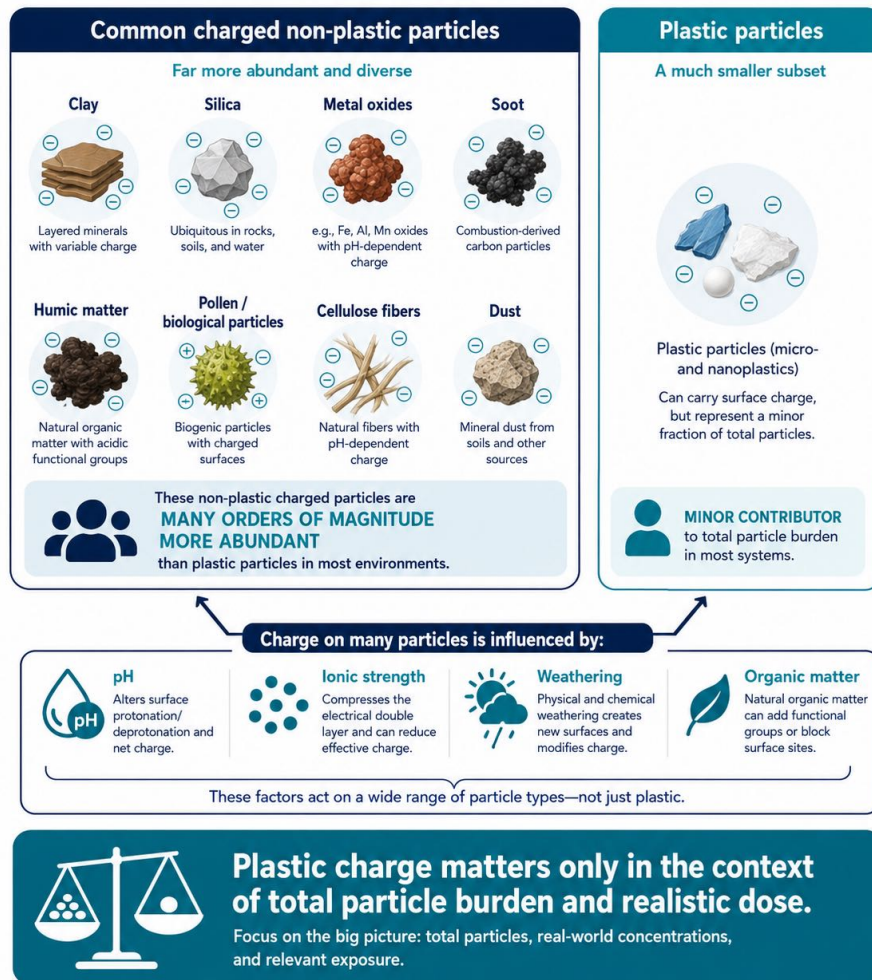
Some recent claims argue that microplastics and nanoplastics may be especially hazardous because small particles can carry surface charge, form coronas, interact electrostatically with biological surfaces, or behave as colloids. Those are real particle-science concepts, but they are not unique to plastic. Clay minerals, silica, metal oxides, soot, cellulose fibers, natural organic colloids, humic substances, biological particles, and ordinary dust particles also carry surface charge or develop charge depending on pH, ionic strength, organic matter, weathering, mineral composition, and surrounding chemistry.

The relevant question is therefore not whether plastic particles can be charged. The relevant question is whether plastic particles contribute a meaningful fraction of total charged particle surface area compared with the vastly larger background of natural and non-plastic particles. Environmental and indoor particle mixtures are dominated by mineral dust, crustal material, combustion particles, road-wear particles, soot, fibers, biological material, skin fragments, silica, calcium carbonate, organic matter, and other non-plastic particles. Plastic is typically a small subset of total particulate exposure.

Charge-based claims therefore require the same evidence standard as any other microplastics claim: confirmed plastic identity, realistic particle size, realistic surface state, realistic dose, comparison with non-plastic particles, and demonstrated harm beyond ordinary charged mineral, organic, and biological particles. Without that comparison, “plastic particles are charged” is not a risk conclusion. It is only a generic property of particulate matter.

Charge is not unique to plastic

Surface charge is a general particle property.



Surface charge, eco-corona formation, aggregation, and colloidal behavior can affect particle transport and measurement. They are legitimate reasons to improve analytical methods and reporting standards. They are not, by themselves, evidence that normal microplastic or nanoplastic exposure causes human disease.

Sources: Rahman 2024; Yang 2025; Gustafsson 2018; Mukherjee & Agrawal 2017; IARC 2012; WHO 2022

Why are particle counts often misleading compared with mass?

Bottom line: A huge number of tiny particles can still weigh almost nothing.

Public claims often sound alarming because they report counts, not mass. Risk depends on dose, and dose often requires mass, size, polymer, route of exposure, and biological effect. Counts alone do not prove danger.

Sources: EFSA 2025; Koelmans 2022; Welle 2018; Mohamed Nor 2021

What makes a microplastics study credible?

Bottom line: A credible study proves particle identity, controls contamination, uses realistic exposure, and avoids overclaiming.

Strong studies include clean sampling, procedural blanks, validated methods, clear size cutoffs, polymer confirmation, mass estimates where possible, realistic dose comparison, and conclusions that match the data.

Sources: EFSA 2025, Plastics Research Council; Koelmans 2019; Gouin 2022; Cowger 2020; Hermesen 2018

What are the strongest studies on microplastics risk?

Bottom line: The strongest studies use realistic exposure, validated identification methods, strong blanks, appropriate controls, and conclusions that match the data.

A strong study should identify the material correctly, compare the dose with real exposure, measure mass where possible, and avoid turning detection into proof of harm.

Sources: Koelmans 2022; WHO 2022; FDA 2024; SAPEA 2019; Hermesen 2018; Cowger 2020

What are the weakest types of studies?

Bottom line: The weakest studies use unrealistic doses, unrepresentative particles, weak controls, contamination-prone methods, or detection tools that cannot prove the particles are plastic.

These studies may be useful for method development or hazard screening, but they should not be used as proof of real-world human risk.

Sources: Lenz 2016; Gouin 2024; Hermesen 2018; EFSA 2025; Cowger 2020

How does this review grade microplastics studies?

Bottom line: Studies are graded by whether they prove plastic identity, avoid contamination, use realistic dose, measure mass where possible, use relevant particles, and show harm rather than merely detection. Structured computer-assisted scoring is used to improve consistency and reduce subjective variation, but it does not eliminate human judgment or potential reviewer bias.

The goal is not to dismiss studies. The goal is to separate reliable evidence from weak claims.

Sources: Koelmans 2019; Koelmans 2022; Hermesen 2018; Cowger 2020; WHO 2022

Which organ-detection studies survive strict contamination-control and intact-particle verification?

Bottom line: This must be judged study by study.

A study should not be accepted merely because it reports plastic in a human tissue. It should prove that the material was plastic, that it was an intact particle, that contamination was controlled, that the size was measurable by the method used, and that the claimed amount fits realistic exposure.

Current human biomonitoring should be interpreted claim by claim rather than as all true or all false. Lane et al. 2026 screened 133 datasets from 101 peer-reviewed human studies and found that none met all 14 essential criteria for direct quantitative risk assessment. That result limits precise estimates of representative body burden and particle-specific dose-response; it does not mean every occurrence finding is false or that overall health risk is unknowable. Lung occurrence is biologically plausible because people inhale dust, but even valid detection does not establish disease or body-wide accumulation. The strongest interpretation keeps occurrence, quantitative burden, accumulation and causation as separate questions.

The body also has normal particle-clearance systems. Inhaled particles deposited in the airways and alveoli can be trapped in mucus, cleared by the mucociliary escalator, or engulfed by macrophages. Geiser reviewed macrophage clearance of inhaled micro- and nanoparticles and noted that particles deposited in the airways and alveoli are readily taken up by resident surface macrophages. This process is part of normal lung defense. It does not mean that no particle can ever persist or cause harm, but it does mean that detection of a particle is not the same as accumulation, toxicity, or disease.

Sources: Lane 2026; Hermesen 2018; Cowger 2020; Clough 2026; Rauert 2025; Jenner 2022; Laforsch 2025; Geiser 2010

7. Disease and Health-Outcome Claims

Recent 2025–2026 human studies deserve explicit consideration rather than omission. They include reported associations involving fecal microplastics and colorectal cancer, cerebrospinal-fluid measurements and neurological or aneurysm outcomes, brain-tumour and matched-tissue studies, intestinal-tumour detections, coronary-blood detections, and reproductive-clinic samples. These studies expand the occurrence and association literature, but selected clinical populations, cross-sectional or case-control designs, uncertain historical exposure, analytical limitations, confounding and reverse causation remain major boundaries. Tran et al. 2026 systematically reviewed 25 studies in living humans; the review found variable risk of bias and did not convert association into causation. The integrated conclusion therefore remains that current human evidence does not establish disease caused by ordinary microplastic exposure.

Sources: Tran 2026; Lane 2026; Xu 2025; He 2025; Li 2026a-b; Chen 2026; Fang 2026; Lu 2026; Paolisso 2026; Kong 2025; Marfella 2024

Do microplastics cause heart attacks or strokes?

Bottom line: No study has proved that normal microplastic exposure causes heart attacks or strokes.

Marfella et al. 2024 reported an association between plastic-associated material in atherosclerotic plaque and later cardiovascular events, but association is not proof of causation. Miller et al. 2017 provides a direct causal-inference control: patients with severe pre-existing carotid atherosclerosis inhaled traceable ~5 nm gold nanoparticles only 24 hours before endarterectomy, and particulate gold was subsequently detected in the excised plaques. The plaques necessarily pre-dated the gold exposure. In a complementary atherosclerosis-prone mouse model, gold accumulated preferentially in plaque-rich vascular regions yet repeated gold administration did not increase plaque size. Thus, particles can localize in diseased vascular tissue without having created the lesion. A 2026 coronary-blood study likewise reported higher MNP detection in STEMI, but smoking was the only independent predictor of MNP presence. These findings support investigation, not a conclusion that ordinary microplastic exposure causes heart attacks or strokes.

Sources: Marfella et al., NEJM 2024; Miller 2017 and correction; Paolisso 2026; Li 2024; Brits 2024; Rauert 2025

Are microplastics in arteries causally involved in cardiovascular events?

Bottom line: No. Causation has not been proved.

A correlation between cardiovascular events and plastic-associated material in arterial plaque does not establish that the material caused the plaque or the events. Miller et al. 2017 demonstrates why localization must not be confused with causation: deliberately inhaled inert gold nanoparticles accumulated in pre-existing plaques within 24 hours, and repeated gold exposure did not enlarge plaques in mice. By extension, finding plastic or another particle within a plaque or thrombus is evidence of co-location, not proof that the particle initiated that lesion. Causation requires independent evidence of temporality, exposure, dose-response and biological effect. Human blood and vascular tissue also contain many nonplastic particles, so any particle-specific mechanism must be demonstrated rather than assumed.

Sources: Marfella et al., NEJM 2024; Miller 2017 and correction; Wu 2023; Bradford Hill 1965

Do microplastics cause cancer?

Bottom line: No credible evidence proves that normal microplastic exposure causes cancer in humans.

Recent human studies have reported associations or detections in cancer-related samples, including fecal microplastic counts in colorectal-cancer cases and microplastic-positive intestinal tumours. These findings are important but do not establish that exposure preceded disease: disease state, diet, bowel transit, inflammation, surgery and sample handling can all affect measured particles. Laboratory cellular-stress findings under artificial conditions likewise do not establish cancer in people at real exposure levels.

Sources: Xu 2025; Chen 2026; WHO 2022; FDA 2024; Koelmans 2022; SAPEA 2019; Gruber 2023; Prust 2020

Do microplastics cause dementia or Alzheimer's disease?

Bottom line: No credible evidence proves that microplastics cause dementia or Alzheimer's disease.

Recent brain and cerebrospinal-fluid studies report occurrence and associations, including Alzheimer's-related CSF findings and higher particle-associated signals in some diseased brain tissues. These studies do not establish that microplastics preceded or caused dementia: disease-related barrier changes, selected clinical sampling, postmortem differences, analytical uncertainty and reverse causation remain plausible. Claims linking microplastics to dementia or Alzheimer's disease should therefore not be presented as causal fact.

Sources: He 2025; Nihart 2025; Li 2026a; Lane 2026; Rauert 2025; WHO 2022; Prust 2020

Do microplastics cause infertility or lower testosterone?

Bottom line: No credible evidence proves that normal microplastic exposure causes infertility or lower testosterone in humans.

Recent infertility-clinic studies report plastic-associated measurements in follicular fluid, seminal plasma or related samples and associations with selected reproductive endpoints. These are selected clinical populations and generally lack validated antecedent internal-dose measurements, so confounding and reverse causation remain unresolved. Animal and cell studies also often use doses far above ordinary exposure. Human causation has not been established.

Sources: Kong 2025; Montano 2025; Koelmans 2022; WHO 2022; Zhao 2023

Do microplastics disrupt hormones?

Bottom line: Microplastic particles themselves have not been proved to disrupt hormones at realistic human exposure.

Some plastic additives can have toxicological profiles of their own, but additives are a separate question from microplastic particles. The two should not be confused.

Sources: Hahladakis 2018; EFSA 2025; WHO 2022

Do microplastics damage the gut microbiome?

Bottom line: No credible evidence proves that normal microplastic exposure damages the human gut microbiome.

Some laboratory and animal studies report microbiome changes under artificial exposures. Those findings do not prove a meaningful effect in people at real-world doses.

Sources: Koelmans 2022; WHO 2022

Do microplastics meaningfully deliver toxic chemicals to humans compared with direct chemical exposure?

Bottom line: No. Credible evidence does not show that microplastics are an important chemical-delivery pathway for humans at normal exposure levels.

Plastic particles can contain or adsorb chemicals in principle. The practical question is whether they deliver a meaningful dose compared with direct chemical exposures from food, air, dust, medicines, cosmetics, consumer products, and the environment. That has not been demonstrated for typical microplastic exposure. (chemical-vector dose context: Koelmans 2016; Koelmans 2022; Bakir 2014; Teuten 2009).

It is also important not to assign chemicals to the wrong plastics. Common packaging plastics such as polyethylene, polypropylene, polystyrene, uPVC, and PET do not require BPA or phthalates as ingredients.

Sources: FDA 2024; Plastics Research Council; Koelmans 2016; Koelmans 2022; Bakir 2014; Teuten 2009

Are plastic additives the same thing as microplastics?

Bottom line: No. Additives are chemicals. Microplastics are solid particles.

A claim about an additive should not automatically be used as a claim about plastic particles. Common packaging plastics such as polyethylene, polypropylene, PET, polystyrene, and uPVC should be judged by their actual composition and approved use, not by assumptions about unrelated additives.

As an example, media articles talk about release of BPA (an endocrine disruptor) from PET used in beverage bottles. What they fail to recognize is that PET is not made using BPA, no BPA is added and so it cannot be released. The same applies to phthalates which are a type of plasticizer not added to common plastics like PET, polyethylene, polypropylene, polystyrene or uPVC (the kind of PVC that drinking water pipes are made from). It is unscientific and incorrect to mention these additives in association with plastics and plastic particles that do not contain them.

Sources: DeArmitt 2025; Zweifel 2009

Are “plastic chemicals” uniquely dangerous?

Bottom line: No. “Plastic chemicals” is usually a misleading phrase because it lumps together polymers, additives, residual monomers, processing aids, contaminants, and unrelated chemicals as though they are one toxic category.

Most plastics people use every day — polyethylene, polypropylene, PET, polystyrene, ABS, nylon, and unplasticized PVC — do not contain BPA or phthalates. BPA is mainly associated with polycarbonate and epoxy resins, while phthalates are mainly used to soften some flexible PVC products, not most plastics.

The additives commonly used in plastics are usually stabilizers, antioxidants, processing aids, fillers, pigments, lubricants, and UV stabilizers. They are added at low levels to improve durability, safety, processing, and recyclability. Many, such as hindered phenolic antioxidants and phosphite stabilizers, are selected precisely because they have low toxicity at realistic exposure levels.

Nor are these chemicals unique to plastic. The same or similar chemicals occur in paper, cardboard, inks, coatings, adhesives, leather, rubber, textiles, cosmetics, fragrances, paints, sealants, and many other materials. Therefore, a claim about “chemicals from plastics” must show actual exposure, actual dose, actual migration, and actual harm. Simply detecting a chemical, or associating it rhetorically with plastic, is not evidence of risk.

Sources: DeArmitt 2025; Zweifel 2009

8. Microplastics evidence audit table

The table below summarizes the major disputed claims, what the cited studies actually show, what they do not prove, the main limitation, and the evidence grade. It is designed to make the review transparent for the public, journalists and policymakers.

Public claim	Best evidence	What the evidence actually shows	What it does not prove	Main limitation / context	Evidence grade
Plastic in brain / 6 g brain claim	Nihart 2025; Rauert 2025; Monikh 2025; Li 2026a; Lane 2026; Mohamed Nor 2021	Reported polymer-associated mass signals and some particle-resolved occurrence evidence in selected human brain/olfactory tissues.	Does not establish representative brain burden, progressive accumulation, neurological harm, or ~6 g of confirmed intact plastic particles.	Destructive mass methods do not preserve particle identity/size/location; lipid-rich matrix, selected samples, contamination and mass-plausibility issues remain. Exposure and burden models cover defined size domains and cannot be used as a simple retention ratio.	Selected occurrence evidence; 6 g intact-particle burden not validated
Particles or polymer signals in blood / thrombi	Wu 2023; Brits 2024; Leslie 2022; Rauert 2025; Guan 2023	Blood studies report particles or polymer-associated signals by method. Wu analyzed thrombi rather than normal circulating blood and identified 1 LDPE particle among 87 Raman-identified particles; non-plastic particles dominated.	Does not prove disease, toxicity, accumulation, normal blood composition, or that destructive polymer-mass signals represent intact circulating plastic particles.	Very small amounts; contamination risk; methods differ. Wu is a thrombus study, not a general blood-particle survey; Py-GC/MS destroys the sample.	Occurrence / association context; weak for harm
Bottled-water nanoplastics	Qian 2024; Materic 2024; Qian Reply 2024; Jüngling 2026; Ruggeri 2025	Reported high particle counts in bottled water using stimulated Raman scattering; later work addresses method challenges.	Did not prove bottled water is dangerous or causes disease; high-count result has not been established as routine exposure.	New method; blank/control debate; particle count rather than risk; very small estimated mass.	Method-limited detection; weak for risk
Credit-card-per-week exposure claim	Cox 2019; Senathirajah 2021; Welle 2018; Mohamed Nor 2021	Earlier exposure models produced public claims of large intake; later mass-based analyses give much lower context.	Does not prove people ingest grams per week; does not fit mass-based exposure or retention estimates.	Particle-to-mass assumptions and overinterpretation; conflicts with later mass-based estimates.	Unsupported / contradicted
Lifetime intake and retained burden	Mohamed Nor 2021	Nor et al. separately modeled ~583 ng/day external intake over a 1–5,000 µm domain and ~40.7 ng adult tissue burden for a 1–10 µm domain.	Does not provide a measured whole-exposure absorption or retention fraction; the two outputs should not be divided.	Different particle-size domains and model stages; useful for scale and plausibility, not a direct retention percentage.	Strong for scale context; not a direct retention fraction
High-dose laboratory harm studies	Lenz 2016; Jung 2026; Gouin 2022; Koelmans 2019; Eberhard 2024; Burns 2018	Many studies use concentrations far above measured environmental or human exposure. Jung 2026 gavaged mice with 2 mg PE/day for 8 weeks and mapped a high-dose PPARα–ANXA2 liver response.	Do not prove that normal human exposure causes harm or that the same mechanism is engaged at realistic dose.	Dose can be hundreds, thousands, or millions of times above real exposure; many studies also use artificial particles or forced exposure routes. Jung used no realistic-dose arm or dose-response series.	Useful for hazard / mechanism; weak for real-world risk
Artificial polystyrene bead studies	Lenz 2016; Hartmann 2019; Gigault 2021; Gouin 2022	Often use uniform laboratory beads because they are convenient and measurable.	Do not prove effects from real-world mixed plastic particles at realistic exposure.	Particle type, shape, size, surface, and dose often do not match environmental exposure.	Weak for human risk
Blood-brain barrier crossing claim	Kopatz 2023; Lenz 2016; Shan 2022; Semmler-Behnke 2008	Reported that lab-made polystyrene nanoparticles can cross biological barriers under experimental conditions.	Does not prove environmental nanoplastics cross the human BBB at normal exposure levels.	Artificial particles, forced exposure, and extreme dose relative to realistic exposure.	Weak for real-world human risk
Heart attacks / strokes / atheromas	Marfella 2024; Miller 2017 + correction; Paolisso 2026; Bradford Hill 1965	Reported vascular associations and particle localization. Miller showed deliberately inhaled inert gold could localize in pre-existing plaques; repeated gold exposure did not enlarge plaques in mice.	Does not prove that plastic caused plaque, thrombosis, heart attacks, strokes or death.	Association/localization is not causation; temporality, confounding, exposure measurement and reverse causation remain critical.	Association/localization only; causation not proved

Public claim	Best evidence	What the evidence actually shows	What it does not prove	Main limitation / context	Evidence grade
Placenta / fetal exposure claims	Ragusa 2021; Liu 2023; Zhu 2023; Bongaerts 2020; Clough 2026	Reported particles or plastic-associated findings in placenta or related samples.	Did not prove fetal harm, developmental disease, or reliable body-wide accumulation.	Small samples; contamination sensitivity; variable methods; particle identity and blanks often disputed.	Detection only; harm not proved
Lung tissue findings	Jenner 2022; Hayden 2023; Amato-Lourenco 2021; Wieland 2022; Vianello 2019	Plastic occurrence is plausible by inhalation. Hayden independently characterized 124,018 inorganic particles across 45 classes in 38 lung biopsies, showing the large nonplastic background against which plastic detections occur.	Does not prove disease, plastic dominance, population lung burden, or that Hayden provides a plastic/nonplastic ratio.	Methods and size windows differ; Hayden FE-SEM/EDS was qualitative and optimized for inorganic particles, while plastic identity requires polymer-specific methods.	Moderate for plastic occurrence; strong nonplastic context; weak for harm
Stool findings / elimination	Schwabl 2019; Zhang 2021; Mohamed Nor 2021	Reported particles in stool and modeled very low retained burden.	Does not prove accumulation or harm.	Stool detection mainly shows ingestion and excretion; analytical methods vary.	Moderate to strong for elimination context
Tea-bag particle release	Hernandez 2019; EFSA 2025	Reported particle release from some polymer-containing tea bags under hot-water conditions.	Did not prove harm to consumers.	Method limitations, particle-count emphasis, solubles/precipitate issues, and missing dose-risk context.	Release detection only; weak for risk
Plastic cutting boards	Yadav 2023; EFSA 2025; IARC 1995	Reported particles can be released during cutting or scraping.	Did not prove normal cutting-board use causes disease or meaningful exposure risk.	Particle count and estimated mass need context; hygiene and alternative-material risks must be compared.	Release possible; weak for health risk
Drinking-water occurrence and treatment	WHO 2019; Koelmans 2019; Pivokonsky 2018; Pivokonsky 2020; Velasco 2022; Velasco 2023; Mintenig 2019; Jüngling 2026; Yang 2024; Gambino 2022	Microplastics can be detected in some drinking water; treatment removes many particles; plastic may be a small minority of total particles.	Does not prove health risk from tap or bottled water.	Methods vary; size cutoffs differ; particle count often lacks mass and toxicity context.	Moderate for occurrence; weak for risk
Drinking-water distribution pipes are a net source of microplastics to household tap water	Sun 2024; Feld 2021	Sun found fewer microplastics in household tap water than in treated water leaving the plant, suggesting the distribution system was not a net contributor in that system. Feld found very low confirmed microplastic levels in 17 Danish tap-water samples, with no PE or PVC particles confirmed.	Does not prove that pipes never release particles. Abrasion, installation, ageing or abnormal conditions can create release; not every particle was source-traced.	Source contribution is system- and condition-specific. Sun used 1 L samples and a 20 µm cutoff; Feld was not a direct pipe-release experiment.	Moderate evidence against general net-source assumption; not evidence of zero release under all conditions
Ocean fiber composition	Suaria 2020	Global surface-water fiber analysis found about 8% synthetic fibers and about 92% natural fibers.	Does not prove all ocean particles everywhere have the same composition or that fibers are harmful.	Surface-water fiber dataset; not a complete inventory of all marine debris.	Strong for fiber-composition context
Ocean ecological risk	Beiras 2020; Jovanovic 2018; GESAMP 2016; FAO 2017	Reviewed or tested risks and found currently monitored microplastics pose negligible or limited demonstrated risk under realistic conditions.	Does not prove no possible effect under every species, particle type, or local pollution condition.	Ecological risk varies by exposure, species, particle type, and local conditions.	Moderate to strong for current monitored risk
Dust exposure context	Mohamed Nor 2021; Wieland 2022; Vianello 2019; Yakovenko 2025; Eberhard 2024	Plastic appears to be a small fraction of indoor and outdoor particles compared with mineral dust, cellulose, protein, soot, skin particles, and other material.	Does not prove every dust environment has the same composition.	Dust composition varies by setting and method; exposure is often reported by count rather than mass.	Strong for comparative context
Chemical-vector claim	Koelmans 2016; Koelmans 2022; Bakir 2014; Teuten 2009; Beiras 2018; Beiras 2019; Ziajahromi 2019	Plastics can sorb chemicals in principle; several studies show vector effects are often negligible or can reduce bioavailability.	Does not prove microplastics are an important chemical-delivery pathway for humans at normal exposure.	Chemical dose from particles is tiny compared with direct exposure pathways; context often missing.	Strong against major human vector risk

Public claim	Best evidence	What the evidence actually shows	What it does not prove	Main limitation / context	Evidence grade
Additives vs microplastic particles	Hahladakis 2018; EFSA 2025; FDA 2024; Lithner 2011	Additives, monomers, leachables, and particles are different categories that require separate evidence.	A chemical claim does not automatically prove particle toxicity.	Public and media often conflate particles with additives or unrelated chemicals.	Strong conceptual distinction
Alternatives to plastic	Meng 2024; Voulvoulis 2019; Franklin Associates 2018; Simantris 2024; Reimann 2010; Krachler 2009; Turner 2019	Many plastic replacements increase material use, emissions, weight, breakage, waste, or other impacts.	Does not prove plastic is best in every application.	LCA is application-specific; substitution must compare full systems.	Strong for substitution caution
Agency risk conclusions	FDA 2024/2026; WHO 2022; EFSA 2025; EPA 2026; BfR 2025; UK COT 2024	Agency positions differ by route, date and purpose. Several call for better methods/data; FDA and BfR communicate low or unproven current human-health risk. EPA draft CCL 6 is a prioritization step, not a harm determination.	Does not mean all agencies reviewed the same evidence base, nor that statements of uncertainty prove harm or make the broader literature incapable of risk evaluation.	Agency reviews use different evidence windows and scopes; WHO 2022 reviewed evidence through Dec 2021, EFSA is preparing a new opinion, and UK COT could not complete a full inhalation assessment from its available evidence.	Strong for policy context; not a substitute for full evidence synthesis

9. Common Claims and Public Interpretation

Do people eat a credit card of plastic every week?

Bottom line: No. The "credit card per week" claim is not credible.

A credit card weighs about 5 grams. Mohamed Nor et al. estimated adult intake at about 583 nanograms per day, or about 0.015 grams over 70 years before elimination and with only nanogram-scale retained burden (Mohamed Nor 2021). That is nowhere near 5 grams per week. The credit-card claim exaggerates exposure by enormous amounts. Re-analysis showing that the widely repeated "one credit card per week" microplastics ingestion claim is based on serious calculation and data-combination errors (Pletz 2022).

Sources: Mohamed Nor 2021; Cox 2019; Welle 2018; Plastics Research Council; Senathirajah 2021; Pletz 2022

Are there hundreds of thousands of particles in bottled water?

Bottom line: No reliable evidence shows that bottled water contains dangerous numbers of plastic particles. One study using a new method made a very high particle-count claim, but it has not been independently replicated. Even if the count were correct, particle count is not the same as danger.

A large count of extremely tiny particles can represent very little mass. A health claim also needs proof of particle identity, dose, biological uptake, and harm. Bottled-water particle counts alone do not show a health risk. Even if there were hundreds of thousands of nanoparticles in water, converting the reported particle counts and sizes into mass gives an estimated concentration around 10 ng/L, or about 0.000000001% by weight (calculated from Qian 2024). The plastic types discussed are the same food-contact polymers used in bottles and caps (Welle 2018; FDA 2024).

Sources: Qian 2024, Welle 2018; Jüngling 2026; Materic 2024; Schymanski 2018

Do microplastics carry toxic chemicals into the body?

Bottom line: No credible evidence shows that microplastics are an important toxic-chemical delivery pathway for humans at normal exposure levels.

Plastic particles can contain additives or sorb chemicals from the surrounding environment in principle. The practical question is dose: do microplastics deliver a meaningful chemical exposure compared with direct exposure from food, air, water, dust, medicines, cosmetics, consumer products, and the surrounding environment? Critical reviews and model-based analyses show that this has not been demonstrated for typical human microplastic exposure (Koelmans 2016; Bakir 2014; Teuten 2009; Gerdes 2019; Koelmans 2022).

In some experiments, plastic particles do not amplify chemical toxicity and may instead reduce short-term bioavailability by binding chemicals to the particle surface. Rehse et al. reported that microplastic particles reduced the short-term toxic effect of bisphenol A on aquatic organisms, and Kleinteich et al. found that sorption of PAHs to microplastics decreased their acute toxicity in zebrafish embryos. Other studies likewise show that the chemical-vector claim is not a simple one-way transfer from plastic to organism (Beiras 2018; Beiras 2019; Ziajahromi 2019).

These studies do not prove that microplastics are beneficial. They show that the simple claim "plastic particles carry toxic chemicals into the body" is scientifically incomplete. A valid vector claim must quantify the chemical mass on the particles, compare it with direct chemical exposure, show release under realistic biological conditions, and demonstrate harm at realistic microplastic doses. Additive questions should also be separated from particle questions: food-contact plastics and additives must comply with applicable food-contact regulations, but chemical-additive exposure is not the same question as microplastic-particle toxicity.

It is also important not to treat ordinary plastic products as if they all contain the same chemicals. BPA, phthalates, PFAS, flame retardants, pigments, stabilizers, monomers, oligomers, and leachables are different questions. Many are not present in most common plastics at all, and many are not unique to plastics.

Sources: Beiras 2018, Beiras 2019, Ziajahromi 2019, Welle 2018; Koelmans 2016; Koelmans 2022; Bakir 2014; Teuten 2009; Rehse 2018; Kleinteich 2018

Are alternatives to plastic always safer?

Bottom line: No. Alternatives are not automatically safer or better.

Glass, metal, paper, cotton, and other alternatives can increase weight, breakage, energy use, emissions, food waste, or other hazards. A fair comparison must look at total risk and life-cycle impact, not just whether an item is plastic. Copper from pipes or cookware can be toxic. There are studies showing heavy metals coming from stainless steel and glass containers and paper.

Sources: Simantris 2024; Reimann 2010; Krachler 2009; Turner 2019; Kamerud 2013; Verissimo 2005; DeArmitt 2023; Franklin Associates 2018; Li 2024; Meng 2024; Voulvoulis 2019

Replacing plastic can worsen overall impact

Material choices should be judged by total impact, not fear.



Should plastic be avoided because of microplastics?

Bottom line: No. A plastic avoidance recommendation is unjustified. People can use plastics sensibly. Avoiding all plastic is shown to increase other risks or environmental impacts. Decisions should be based on measured exposure, proven risk, and full comparisons with alternatives.

Sources: Meng 2024; Voulvoulis 2019; Li 2024; Koelmans 2022; WHO 2022; FDA 2024

10. Agency and Regulatory Context

Agency statements must be interpreted within the scope, evidence window and purpose of each review. They are not independent evaluations of one identical evidence base, and none of the agency assessments summarized here evaluated the complete evidence corpus synthesized in the current Plastics Research Council evidence program. WHO's 2022 review covered evidence through December 2021; EFSA is preparing a new opinion; EPA's draft CCL 6 listing is an information-collection and regulatory-priority step rather than a determination of harm; the UK Committee on Toxicity could not complete a full quantitative inhalation assessment from the evidence available to it; and Germany's BfR states that current knowledge indicates relatively low human-health risk and that tissue detections or correlations do not establish causation. Statements that more data are needed should therefore not be read as a finding that the broader scientific literature is incapable of evaluating ordinary microplastic risk.

Sources: FDA 2024 and 2026; WHO 2022; EFSA 2025; EPA draft CCL 6 (2026); BfR 2025; UK COT 2024

What does FDA currently say?

Bottom line: FDA says current evidence does not show that microplastics or nanoplastics in food pose a human health risk.

FDA also recognizes that analytical methods and exposure data are still developing. The practical message is clear: keep studying the issue, but current evidence does not show a food-safety risk.

Sources: FDA, Microplastics and Nanoplastics in Foods, 2024; WHO 2022; FDA 2024

What does WHO currently say?

Bottom line: WHO did not conclude that microplastics in drinking water, diet or air posed a demonstrated human-health risk in its 2022 review, but that review reflects the evidence available through December 2021 rather than the full newer literature.

WHO identified major method, exposure and toxicology uncertainties and called for additional research. That is a statement about the evidence available within that review window, not evidence that ordinary exposure has been shown to cause harm.

Sources: WHO 2019; WHO 2022; Koelmans 2022

What does EPA currently emphasize?

Bottom line: EPA emphasizes research, measurement and method development; its 2026 draft CCL 6 inclusion of microplastics is a prioritization and information-collection step, not a regulatory determination that harm has been demonstrated.

EPA's work focuses on occurrence, analytical methods, fate and potential risk. A Contaminant Candidate List entry imposes no drinking-water requirement and should not be represented as proof of toxicity or a completed health-risk determination.

Sources: EPA Microplastics Research; EPA 2024; California 2025

What does EFSA say or imply?

Bottom line: EFSA identifies major knowledge and measurement gaps and is preparing a new scientific opinion on potential health risks from microplastics in food, water and air.

EFSA's current work should not be interpreted as a finding that ordinary exposure is harmful. Its new opinion is intended to reassess the evidence using improved methods and newer data; completion is expected by the end of 2027.

Sources: EFSA 2016; EFSA 2025; SAPEA 2019

Why do agencies call for more research if there is no proven harm?

Bottom line: Agency calls for more research identify unresolved questions within particular assessment scopes; they are not evidence that harm has been demonstrated and are not proof that the full literature is insufficient to evaluate ordinary risk.

Better methods can improve precision, comparability and route-specific quantitative assessment. That is different from saying the central risk question is unknowable: exposure, toxicology, kinetics, comparative particle evidence and human studies can still be integrated to judge whether ordinary exposure constitutes a demonstrated material public-health threat.

Sources: WHO 2022; FDA 2024; EFSA 2025; EPA Microplastics Research; SAPEA 2019

Are there regulatory limits for microplastics in food or drinking water?

Bottom line: There are not yet broad health-based microplastic limits in food or drinking water. The absence of a dedicated limit should not be interpreted either as proof of safety or as evidence that harm is suspected; agencies generally require validated measurement methods, exposure data and a substance-specific risk basis before setting numerical limits.

Some jurisdictions are developing monitoring methods and requirements. Monitoring is not the same as proof of harm. Good regulation should be based on validated methods, realistic dose, and demonstrated risk.

Sources: EPA Microplastics Research; FDA 2024; WHO 2019; WHO 2022; EFSA 2025

Should microplastics be regulated?

Bottom line: Regulation should target real risk, not headlines.

Monitoring may be useful, but monitoring is not proof of harm. Poorly designed rules can push people toward heavier, higher-impact, or more hazardous substitutes and can distract from larger risks.

Sources: EPA Microplastics Research; WHO 2022; SAPEA 2019; Koelmans 2022; OECD 2021

What is the precautionary principle, and does it justify plastic bans?

Bottom line: Precaution should prevent real harm, not create worse substitutes.

If replacing plastic increases waste, emissions, breakage, food spoilage, metal exposure, or other risks, then a plastic ban may increase overall harm.

Sources: SAPEA 2019; WHO 2022; Franklin Associates 2018; Voulvoulis 2019; Meng 2024; Simantris 2024

11. How to Evaluate New Microplastics Claims

What would count as proof that microplastics harm humans?

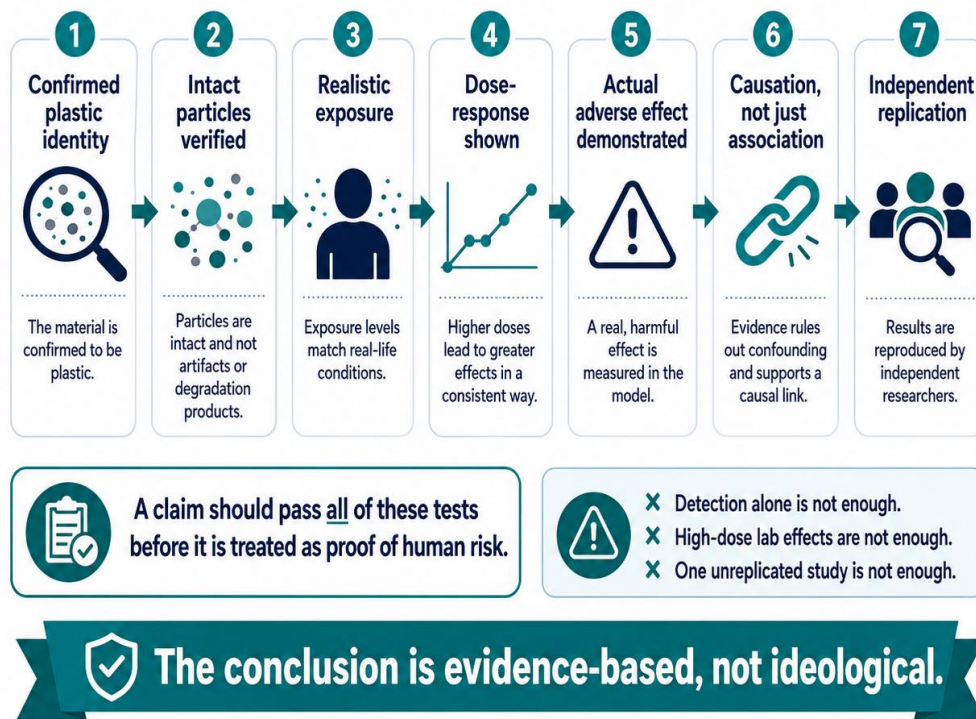
Bottom line: Proof would require confirmed particle identity, realistic exposure levels, reliable dose measurement, biological uptake at relevant sites, consistent health effects, and evidence that plastic particles caused the effect rather than merely being associated with it.

Detection alone is not enough. Association alone is not enough. High-dose laboratory effects are not enough.

Sources: Bradford Hill 1965; Koelmans 2022; WHO 2022; FDA 2024; Hermesen 2018; Cowger 2020

What would prove harm?

The conclusion can change — but only with strong evidence.



Sources: WHO 2022; EFSA 2025; FDA 2024; Lenz 2016

What would change the conclusion?

Bottom line: The conclusion would change if high-quality studies showed confirmed plastic particles at realistic exposure levels causing reproducible harm in humans or credible animal models.

Current evidence does not show that. The review should remain open to better evidence while rejecting claims that do not fit the data.

A new claim should not be accepted just because it sounds alarming. Use this checklist before treating any claim as true.

- Did the study prove the particles were plastic?
- Did it prove they were intact particles, not dissolved material, additives, or contamination?
- Were procedural blanks and contamination controls strong enough?
- Was the dose realistic?

- Was the particle type realistic?
- Was the particle size measurable by the method used?
- Did the study measure mass, or only particle count?
- Did it prove harm, or only detection?
- Did it prove causation, or only association?
- Does the claimed amount fit known exposure estimates?

Sources: Bradford Hill 1965; Koelmans 2022; WHO 2022; FDA 2024; Ioannidis 2005; Goodman 1999

12. Conclusion

The conclusion of this review is clear: dust and particles are everywhere, plastic is only one small component of that background, and normal real-world microplastic exposure has not been shown to cause human disease. There is no scientific or logical reason to treat ordinary plastic dust as a special public-health threat simply because it is plastic.

This is not because the science is missing. The relevant literature spans more than five decades across polymer toxicology, particle toxicology, occupational dust, exposure science, food-contact materials, medical polymers, environmental science and human biomonitoring. The 2026 comprehensive Plastics Research Council synthesis catalogued 1,661 canonical evidence records. The central conclusion is therefore solid rather than indeterminate: ordinary plastic-particle exposure has not been shown to cause human disease.

Dust and particles are not unusual; they are unavoidable at some level. Soil, sand, clay, concrete, paper, wood, cotton, wool, skin cells, pollen, spores, soot, metals, rust, glass, ceramics, paint, road dust, food particles and textile fibers all contribute to the particles people inhale and ingest every day. Plastic is one small part of this total particle exposure. Its detection is therefore expected and, by itself, says nothing about exceptional hazard.

Ordinary common-polymer plastic particles behave in many important respects like other low-toxicity particles. At sufficiently high, atypical particle loading they can irritate tissues, trigger inflammation or impair clearance, just as many other poorly soluble dusts can. Those high-dose effects demonstrate generic particle biology and the importance of dose; they do not establish a special plastic hazard at normal environmental exposure.

The comparative evidence is important. Several non-plastic particle classes have established serious human hazards that ordinary plastic dust has not demonstrated: respirable crystalline silica and occupational wood dust are established carcinogenic hazards, asbestos is a well-established fibrous carcinogen, and certain toxic metal-containing particles can cause systemic toxicity. No reliable evidence shows ordinary common-polymer plastic dust to be more hazardous than the broader low-toxicity dust background at realistic exposure. On that evidence, singling out plastic while ignoring the larger and sometimes more hazardous particle mixture is scientifically incomplete.

The important question is dose. Real-world exposure to microplastics is extremely small by mass. Best available estimates place normal intake approximately 3 million to 600 million times below the principal higher-confidence study-specific doses that produced no detected adverse effects in animal studies. The exact benchmark differs by polymer and study, so the values are not one universal safety limit for every plastic. Normal inhalation exposure is also far below occupational limits for low-toxicity dust. Even if exposure estimates were wrong by large factors, they would still remain far below those selected no-adverse-effect dose scales.

This matters because many alarming studies do not use realistic conditions. They often expose cells, animals, or aquatic organisms to concentrated doses hundreds, thousands, or millions of times above real-world exposure. They often use perfect laboratory polystyrene beads rather than the mixed, irregular, weathered particles actually found in the environment. They often report stress markers, associations, or particle detections rather than disease. Those studies may be useful for method development or hazard screening, but they do not prove that normal human exposure is harmful.

Detection is not danger. Finding a particle, a plastic-like signal, or a polymer-associated chemical fragment does not prove toxicity, accumulation, disease, or causation. A valid health-risk claim must prove that the material was plastic, that intact particles were present, that contamination was controlled, that the dose was realistic, that the

particles reached a relevant biological site, and that harm occurred because of the plastic particles. Most alarming claims do not meet that standard.

Accumulation has also not been demonstrated. Most ingested particles pass through the digestive tract. The body has normal particle-clearance systems, including gut elimination, mucociliary clearance, macrophage uptake, lymphatic handling, and renal or biliary clearance for sufficiently small or soluble material. Finding particles in stool is therefore evidence of elimination, not evidence of danger. Claims of large accumulation in organs are not credible unless they fit measured intake, realistic retention, and basic mass balance.

Claims about plastic in blood, brain, placenta, arteries, reproductive tissues, and other organs must be interpreted with particular caution. These studies are technically difficult, contamination is easy, and some methods detect chemical signals rather than intact particles. Lane et al. 2026 found that none of 133 human tissue or bodily-fluid datasets met all 14 criteria for direct quantitative risk assessment. Hayden et al. 2023 shows that lung tissue contains a vast and chemically diverse nonplastic particle background, while Miller et al. 2017 demonstrates that traceable nanoparticles can accumulate at pre-existing diseased vascular sites without having caused those lesions. Even when particles or polymer-associated signals are genuinely present, localization is not the same as accumulation, toxicity or causation.

Microplastics are also not a proven toxic-chemical delivery pathway for humans at normal exposure. Plastic particles can contain additives or sorb chemicals in principle, but the practical question is dose. The chemical exposure carried by tiny amounts of microplastic must be compared with direct exposure from food, water, air, dust, medicines, cosmetics, consumer products, and the surrounding environment. Current evidence does not show that microplastics deliver a meaningful toxic dose to humans under normal conditions.

The same principle applies to environmental claims. Microplastics can be detected in water, soil, sediment, air, dust, wildlife, and food. But detection in the environment is not proof of ecological harm, human-health risk, or a worsening crisis. The strongest evidence shows that risk depends on actual concentration, dose, particle type, exposure route, retention, and demonstrated biological effect. In many cases, plastic is a small minority of total particles, while natural fibers, mineral particles, cellulose, soot, pigments, and other non-plastic materials dominate.

The public has often been misled because headlines treat detection as if it were harm, particle counts as if they were dose, and high-dose laboratory effects as if they represented real life. That is not sound science. A million tiny particles can still represent a negligible mass. A laboratory effect at an extreme dose does not prove everyday risk. A correlation does not prove causation. A plastic-associated chemical signal does not prove intact particles. A study headline does not override decades of toxicology and exposure evidence.

The evidence does not support panic, plastic-specific avoidance, detox products or fear-based policy. Disproportionate focus on plastic dust is particularly difficult to justify when the much larger particulate background is ignored and some of its components have established hazards that plastic does not. Unnecessary fear is not a neutral outcome: sustained stress and anxiety can affect wellbeing and decision-making. This does not mean that any particular symptom is imaginary or psychogenic; it means public-health messaging should be proportional to demonstrated risk. Material decisions should be based on total evidence and full-system comparison, not fear of one small class of particles.

None of this means litter should be ignored. Unnecessary release of particles should be reduced where practical. Waste should be managed responsibly. Large plastic debris, lost fishing gear, and poor waste management are

real environmental issues. But those are not evidence that normal microplastic exposure causes human disease. Macroplastic litter and microplastic health fear are different questions and should not be confused.

The scientific conclusion is therefore straightforward:

- Dust and particles are ubiquitous and unavoidable at some level; detection is normal and does not equal harm.
- Plastic is only a small part of the much larger particulate background in many measured settings.
- No reliable evidence shows ordinary common-polymer plastic dust to be more hazardous than the broader low-toxicity dust background at realistic exposure.
- Respirable crystalline silica, asbestos, occupational wood dust and certain toxic metal-containing particles have established serious hazards that ordinary plastic dust has not demonstrated.
- At high, atypical particle loading, plastic can produce generic particle effects just as other poorly soluble dusts can; this does not make ordinary plastic exposure a special hazard.
- Normal real-world exposure is approximately 3 million to 600 million times below the principal higher-confidence study-specific doses that caused no detected adverse effects in animal studies.
- After decades of evidence and a comprehensive 1,661-record synthesis, there is no credible evidence that normal microplastic exposure causes human disease and no scientific basis for treating plastic dust as a special public-health threat.

Public concern should be proportional to evidence. The evidence shows that ordinary microplastic exposure is a low-dose component of a much larger particle background, not a demonstrated public-health crisis. The rational response is accurate measurement, realistic dose comparison, sensible control of total dust where appropriate, attention to particle hazards that are actually demonstrated, and scientifically honest communication that does not create unnecessary fear.

The burden of proof now lies with anyone claiming that normal microplastic exposure is harmful. To overturn the current conclusion, a study would need to show confirmed intact plastic particles, realistic exposure, strong contamination control, meaningful retained dose, reproducible harm, and causation rather than mere detection or association.

The logical conclusion is clear: dust cannot be eliminated from life, plastic is only a small fraction of that background, and there is no scientific evidence that ordinary plastic dust warrants special concern over the many other particles people encounter every day. Normal real-world microplastic exposure has not been shown to cause human disease.

Appendix A. Review Methods and Corpus Construction

Review type and scope

This document is a critical evidence review supported by a controlled living evidence corpus. It is not presented as a prospectively registered PRISMA systematic review. The public Q&A review directly evaluated more than 700 microplastics studies over seven years and was cross-checked against the broader 2026 Plastics Research Council comprehensive synthesis.

Canonical evidence records

The certified 2026 baseline contains 1,661 unique canonical evidence records after record-identity and publication-lineage resolution. In this program, a canonical record is the single identity-resolved evidence record used as one independent unit after duplicate versions and publication-lineage items are reconciled. Supporting information, drafts, press material, corrections or retractions, and other non-independent or administrative records are linked or excluded from independent scientific-source counts as appropriate.

Corpus sources and evidence acquisition

The corpus was assembled from full texts and abstracts collected by the review program; title, DOI, and source discovery; cited references; publisher pages; DOI records; repositories; authoritative records; correction and retraction records; and controlled database fields. The evidence base spans occurrence, exposure, analytical reliability, kinetics, toxicology, comparative particle science, human evidence, environmental effects, and agency or regulatory context.

Screening, adjudication, and database control

Retention was controlled through source verification, publication-lineage control, scientific appraisal, residual-priority closure, and separately adjudicated insertions. The database tracks canonical study IDs, former IDs, duplicate consolidation, metadata correction, evidence role, source type, appraisal or priority, provenance, exclusions, corrections, and adjudication rationale. Structured computer-assisted scoring is used to improve consistency; final scientific interpretation remains a human judgment.

Search-history limitation

Historical raw search strings, search dates, result totals, pre-canonical deduplication counts, and a complete PRISMA identification flow were not retained for the legacy corpus. The corpus should therefore be understood as a controlled living evidence program rather than a reconstructable prospectively registered systematic-search workflow. This limitation is stated explicitly so that the 1,661-record count is not mistaken for a raw database-search yield.

Evidence cutoff and updating

This public release incorporates literature and agency material identified by the review team through 19 August 2026. New evidence after that date is outside the release window and will be evaluated in subsequent updates. The evidence-quality criteria used for study appraisal are described in “How We Grade the Evidence,” and the review remains open to correction when stronger contrary evidence is identified.

Sources: Plastics Research Council 2026 comprehensive synthesis and controlled corpus documentation.

Selected Sources

- R. Ahmad et al., Occupational Cement Dust Exposure and Inflammatory Nemesis: Bangladesh Relevance, *Journal of Inflammation Research*, 14, 2425-2444, 2021
- Akbulut et al., Microplastic release from single-use plastic beverage cups, *Foods*, 13 (10), 1564, 2024. DOI: 10.3390/foods13101564
- F. N. Almeida et al., Py-GC-MS/MS quantification of microplastics in vertebrate tissues: addressing false positives of polyethylene, *Journal of Hazardous Materials*, 501, 140658, 2026. DOI: 10.1016/j.jhazmat.2025.140658
- C. Alomar et al., Microplastic ingestion in reared aquaculture fish: Biological responses to low-density polyethylene controlled diets in *Sparus aurata*, *Environmental Pollution*, 280, 116960, 2021. DOI: 10.1016/j.envpol.2021.116960
- L. F. Amato-Lourenco et al., Presence of airborne microplastics in human lung tissue, *Journal of Hazardous Materials*, 416, 126124, 2021
- A. L. Andrady, Microplastics in the marine environment, *Marine Pollution Bulletin*, 62, pp. 1596-1605, 2011
- S. N. Athey & L. M. Erdle, Are we underestimating anthropogenic microfiber pollution? A critical review of occurrence, methods, and reporting, *Environmental Toxicology and Chemistry*, 41, pp. 822-837, 2022
- A. Aves et al., Comparative assessment of five analytical methods for airborne microplastics highlights importance of identifying sub-10 µm methods, *Scientific Reports*, 2026. DOI: 10.1038/s41598-026-49405-2
- S. Ayoub Meo et al., Effect of Duration of Exposure to Cement Dust on Respiratory Function of Non-Smoking Cement Mill Workers, *Int. J. Environ. Res. Public Health*, 10, 390-398, 2013
- B. Baensch-Baltruschat et al., Tyre and road wear particles: A calculation of generation, transport and release to water and soil with special regard to German roads, *Science of the Total Environment*, 752, 141939, 2020
- A. Bakir, S. J. Rowland & R. C. Thompson, Enhanced desorption of persistent organic pollutants from microplastics under simulated physiological conditions, *Environmental Pollution*, 185, pp. 16-23, 2014
- L. G. A. Barboza, L. R. Vieira, V. Branco, C. Carvalho & L. Guilhermino, Microplastics increase mercury bioconcentration in gills and bioaccumulation in the liver, and cause oxidative stress and damage in *Dicentrarchus labrax* juveniles, *Scientific Reports*, 8, 15655, 2018. DOI: 10.1038/s41598-018-34125-z
- L. Baumann et al., Comment on "Uptake and accumulation of polystyrene microplastics in zebrafish (*Danio rerio*) and toxic effects in liver", *Environmental Science & Technology*, 50 (22), pp. 12521-12522, 2016. DOI: 10.1021/acs.est.6b04193
- T. Bayes and R. Price, An essay towards solving a problem in the doctrine of chances, *Philosophical Transactions of the Royal Society of London*, 53, pp. 370-418, 1763
- S. Beer et al., No increase in marine microplastic concentration over the last three decades - A case study from the Baltic Sea, *Science of the Total Environment*, 621, pp. 1272-1279, 2018
- R. Beiras & A. M. Schönemann, Currently monitored microplastics pose negligible ecological risk to the global ocean, *Scientific Reports*, 10, 22281, 2020. DOI: 10.1038/s41598-020-79304-z
- R. Beiras et al., Ingestion and contact with polyethylene microplastics does not cause acute toxicity on marine zooplankton, *Journal of Hazardous Materials*, 360, pp. 452-460, 2018
- R. Beiras et al., Polyethylene microplastics do not increase bioaccumulation or toxicity of nonylphenol and 4-MBC to marine zooplankton, *Science of the Total Environment*, 629, pp. 1-9, 2019
- J. Berg, Addendum to "Editorial retraction of the report 'Environmentally relevant concentrations of microplastic particles influence larval fish ecology,' by O. M. Lönnstedt and P. Eklöv", *Science*, 358 (6370), p. 1549, 2017. DOI: 10.1126/science.aar776
- T. Berg et al., The release of nickel and other trace elements from electric kettles and coffee machines, *Food Additives & Contaminants*, 17 (3), pp. 189-196, 2000
- M. Bergmann, L. Gutow, M. Klages (Eds.), *Marine Anthropogenic Litter*, Chapter 4, p 105, Springer 2015 Citing Van Franeker & Meijboom, 2002
- A. L. Bond, J. F. Provencher, R. D. Elliot, P. C. Ryan, S. Rowe, I. L. Jones, G. J. Robertson & S. I. Wilhelm, Ingestion of plastic marine debris by Common and Thick-billed Murres in the northwestern Atlantic from 1985 to 2012, *Marine Pollution Bulletin*, 77, pp. 192-195, 2013. DOI: 10.1016/j.marpolbul.2013.10.005

- E. Bongaerts et al., Translocation of (ultra)fine particles and nanoparticles across the placenta; a systematic review on the evidence of in vitro, ex vivo, and in vivo studies, *Particle & Fibre Toxicology*, 17 (56), 142235, 2020
- J. Boucher & D. Friot, Primary Microplastics in the Oceans: A Global Evaluation of Sources, International Union for Conservation of Nature (IUCN), Gland, Switzerland, 2017. DOI: 10.2305/IUCN.CH.2017.01.en
- J. Bousquet et al., Allergic rhinitis, *Nature Reviews Disease Primers*, 6, 95, 2020. DOI: 10.1038/s41572-020-00227-0
- H. Bové et al., Ambient black carbon particles reach the fetal side of human placenta, *Nature Communications*, 10, 3866, 2019
- A. B. Bradford Hill, The environment and disease: association or causation? *Proceedings of the Royal Society of Medicine*, 58 (5), pp. 295-300, 1965
- E. Brancaleone et al., Microplastic in Drinking Water: A Pilot Study, *Microplastics*, 3 (1), pp 31-45, 2024
- J. A. Brandon et al., Multidecadal increase in plastic particles in coastal ocean sediments, *Science Advances*, 5, eaax0587, 2019
- M. Brits et al., Quantitation of micro and nanoplastics in human blood by pyrolysis-gas chromatography-mass spectrometry, *Microplastics & Nanoplastics*, 4 (12), 2024
- T. Buerki-Thurnherr, U. von Mandach & P. Wick, Knocking at the door of the unborn child: engineered nanoparticles at the human placental barrier, *Swiss Medical Weekly*, 142, w13559, 2012. DOI: 10.4414/smw.2012.13559
- R. Buesen, S. Vogel, T. Thoma, V. Strauss, S. Groters, K. Becker, F. Muller, T. Grundling, P. Pfohl, A. Vogel, A. Haase & W. Wohlleben, Oral toxicity of small microplastic of polyamide assessed by a standardized guideline study, *Microplastics and Nanoplastics*, 5, 31, 2025. DOI: 10.1186/s43591-025-00137-6
- E. E. Burns & A. B. A. Boxall, Microplastics in the aquatic environment: Evidence for or against adverse impacts and major knowledge gaps, *Environmental Toxicology and Chemistry*, 37, pp. 2776-2796, 2018
- California State Water Resources Control Board, *Microplastics in Drinking Water: monitoring methods and policy materials*, 2025
- A. R. Carraretto et al., Glass Ampoules: Risks and Benefits, *Revista Brasileira de Anestesiologia*, 61 (4), pp. 513-521, 2011
- A. I. Catarino et al., Low levels of microplastics in wild mussels indicate that microplastic ingestion by humans is minimal compared to exposure via household fibres fallout during a meal, *Environmental Pollution*, 237, pp. 675-684, 2018
- CDC, *Pollen and Your Health*, Centers for Disease Control and Prevention, 2024
- C. M. Celano et al., Anxiety disorders and cardiovascular disease, *Current Psychiatry Reports*, 18, 101, 2016
- H. Chen, Z. Liu, W. Qiu, et al., Tumour recurrence, gastrointestinal symptoms and inflammation associated with microplastic-positive intestinal tumours, *Nature Health*, 2026
- S. P. Cetintepe et al., Is exposure to cement dust and heavy metals associated with reduced pulmonary function? A cross-sectional study among cement factory workers in Türkiye, *BMJ Open*, 15, e102214, 2025. DOI: 10.1136/bmjopen-2025-102214
- A. Chamas et al., Degradation rates of plastics in the environment, *ACS Sustainable Chemistry & Engineering*, 8, pp. 3494-3511, 2020
- I. Chaïb et al., Microplastic contaminations in a set of beverages sold in France, *Journal of Food Composition and Analysis*, 144, 107719, 2025
- J. Cherian et al., Microplastic removal from drinking water using point-of-use devices, 2023. URL: <https://pmc.ncbi.nlm.nih.gov/articles/PMC10054062/>
- N. Chiannilkuchai & S. Kejkornkaew, Safety concerns with glass particle contamination: improving the standard guidelines for preparing medication injections, *International Journal for Quality in Health Care*, 33 (2), pp. 1-6, 2021
- H.-C. Chuang, Y.-T. Yang, H.-C. Chen, Y.-H. Hwang, K.-Y. Wu, T.-F. Chen, C.-L. Chen, M.-K. Jhan & T.-J. Cheng, Acute effects of pulmonary exposure to zinc oxide nanoparticles on the brain in vivo, *Aerosol and Air Quality Research*, 20 (7), pp. 1651-1664, 2020. DOI: 10.4209/aaqr.2019.10.0523
- M. E. Clough et al., Avoiding and reducing microplastic false positives from dry glove contact, *Analytical Methods*, 18, pp. 2914-2926, 2026. DOI: 10.1039/D5AY01801C
- V. T. Covello, Risk communication: an emerging area of health communication research, *Annals of the International Communication Association*, 15 (1), pp. 359-373, 1992
- W. Cowger et al., Reporting guidelines to increase the reproducibility and comparability of research on microplastics, *Applied Spectroscopy*, 74 (9), pp. 1066-1077, 2020

K. D. Cox et al., Human consumption of microplastics, *Environmental Science & Technology*, 53 (12), pp. 7068-7074, 2019. DOI: 10.1021/acs.est.9b01517

A. Cozar et al., Plastic debris in the open ocean, *Proceedings of the National Academy of Sciences*, 111 (28), pp. 10239-10244, 2014

F. De Falco et al., Microfiber release to water, via laundering, and to air, via everyday use: a comparison between polyester clothing with differing textile parameters, *Environmental Science & Technology*, 54 (6), pp. 3288-3296, 2020

DeArmitt, C., Polypropylene Resin & Mesh Design. Phantom Plastics LLC. Unpublished expert report / litigation evidence report prepared for Plaintiffs' counsel, Mostyn Law, 2017

C. DeArmitt, *The Plastics Paradox: Facts for a Brighter Future*, Phantom Plastics LLC, 2020

C. DeArmitt, *Poison in the Pipes: Is Copper the New Lead?* Phantom Plastics LLC, 2023

C. DeArmitt, *Shattering the Plastics Illusion: Exposing Environmental Myths*, Phantom Plastics LLC, 2025

C. Dibke, M. Fischer & B. M. Scholz-Böttcher, Microplastic mass concentrations and distribution in German Bight waters by pyrolysis-gas chromatography-mass spectrometry/thermochemolysis reveal potential impact of marine coatings: Do ships leave skid marks?, *Environmental Science & Technology*, 55 (4), pp. 2285-2295, 2021. DOI: 10.1021/acs.est.0c04522

R. Dris et al., Synthetic fibers in atmospheric fallout: A source of microplastics in the environment? *Marine Pollution Bulletin*, 104, pp. 290-293, 2016

K. E. Driscoll and P. J. A. Borm, Expert workshop on the hazards and risks of poorly soluble low toxicity particles, *Inhalation Toxicology*, 32, 53–62, 2020. DOI: 10.1080/08958378.2020.1735581

K. Duis & A. Coors, Microplastics in the aquatic and terrestrial environment: sources (with a specific focus on personal care products), fate and effects, *Environmental Sciences Europe*, 28 (2), 2016

T. Eberhard et al., Systematic review of microplastics and nanoplastics in indoor and outdoor air: identifying a framework and data needs for quantifying human inhalation exposures, *Journal of Exposure Science & Environmental Epidemiology*, 34, pp. 185-196, 2024

ECETOC, Poorly Soluble Particles / Lung Overload, Technical Report No. 122, European Centre for Ecotoxicology and Toxicology of Chemicals, Brussels, 2013

EFSA CONTAM Panel, Presence of microplastics and nanoplastics in food, *EFSA Journal*, 14 (6), 4501, 2016. DOI: 10.2903/j.efsa.2016.4501

EFSA, Literature review on micro- and nanoplastic release from food contact materials, *EFSA Supporting Publications*, EN-9733, 2025. DOI: 10.2903/sp.efsa.2025.EN-9733

A. Elder et al., Translocation of inhaled ultrafine manganese oxide particles to the central nervous system, *Environmental Health Perspectives*, 114 (8), pp. 1172-1178, 2006

M. Eriksen et al., Plastic pollution in the world's oceans: more than 5 trillion plastic pieces weighing over 250,000 tons afloat at sea, *PLoS ONE*, 9 (12), e111913, 2014

G. Eul Joo et al., The effect of different methods of intravenous injection on glass particle contamination from ampules, *SpringerPlus*, 5, 15, 2016

FAO, Microplastics in fisheries and aquaculture: status of knowledge on their occurrence and implications for aquatic organisms and food safety, *FAO Fisheries and Aquaculture Technical Paper No. 615*, Rome, 2017

F. Fayet-Moore et al., An Analysis of the Mineral Composition of Pink Salt Available in Australia, *Foods*, 9, 1490, 2020

Z. Fang, X. Wang, F. Wu, et al., A case-control study linking concentrations of microplastics in human cerebrospinal fluid to intracranial aneurysm risk, *Environmental Pollution*, 395, 127788, 2026

L. Feld et al., A study of microplastic particles in Danish tap water, *Water*, 13 (15), 2097, 2021. DOI: 10.3390/w13152097

V. J. Feron, C. F. M. Hendriksen, A. J. Speek, H. P. Til & B. J. Spit, Lifespan oral toxicity study of vinyl chloride in rats, *Food and Cosmetics Toxicology*, 19, pp. 317-333, 1981

Food Safety and Standards Authority of India, Micro- and Nano-Plastics as Emerging Food Contaminants: Establishing Validated Methodologies and Understanding the Prevalence in Different Food Matrices, project launch, 2024

- Franklin Associates, Life Cycle Impacts of Plastic Packaging Compared to Substitutes in the United States and Canada, prepared for the American Chemistry Council, 2018
- I. Gambino, et al., Occurrence of microplastics in tap and bottled water: Current knowledge, *International Journal of Environmental Research and Public Health*, 19(9), 5283, 2022. DOI: 10.3390/ijerph19095283
- C. C. Gaylarde, J. A. Baptista Neto & E. M. da Fonseca, Paint fragments as polluting microplastics: a brief review, *Marine Pollution Bulletin*, 162, 111847, 2021. DOI: 10.1016/j.marpolbul.2020.111847
- M. Geiser, Update on macrophage clearance of inhaled micro- and nanoparticles, *Journal of Aerosol Medicine and Pulmonary Drug Delivery*, 23 (4), pp. 207-217, 2010. DOI: 10.1089/jamp.2009.0797
- GESAMP, Sources, fate and effects of microplastics in the marine environment: part two of a global assessment, *GESAMP Reports and Studies No. 93*, 2016
- J. Gigault et al., Nanoplastics are neither microplastics nor engineered nanoparticles, *Nature Nanotechnology*, 16, pp. 501-507, 2021
- German Federal Institute for Risk Assessment (BfR), Microplastics - What do we know today? 11 November 2025; and Microplastics: Discrepancy between scientific knowledge and public perception, 2 December 2025
- M. Gilbert (Ed.), *Brydson's Plastics Materials*, 8th ed., Butterworth-Heinemann/Elsevier, Amsterdam, 2017
- P. P. González, C. Bautista-Capetillo, A. Ruiz-Canales, J. González-Trinidad, H. E. Júnez-Ferreira, A. R. C. Rodríguez & C. O. R. Rovelo, Characterization of Scale Deposits in a Drinking Water Network in a Semi-Arid Region, *International Journal of Environmental Research and Public Health*, 19 (6), pp. 3257, 2022. DOI: 10.3390/ijerph19063257
- S. N. Goodman, Toward evidence-based medical statistics. 1: The P value fallacy, *Annals of Internal Medicine*, 130 (12), pp. 995-1004, 1999
- T. Gouin et al., Screening and prioritization of nano- and microplastic particle toxicity studies for evaluating human health risks - development and application of a toxicity study assessment tool, *Microplastics and Nanoplastics*, 2, 2, 2022
- T. Gouin et al., Addressing the relevance of polystyrene nano- and microplastic particles used to support exposure, toxicity and risk assessment: implications and recommendations, *Particle and Fibre Toxicology*, 21, 39, 2024. DOI: 10.1186/s12989-024-00599-1
- E. S. Gruber et al., To waste or not to waste: questioning potential health risks of micro- and nanoplastics with a focus on their ingestion and potential carcinogenicity, *Exposure and Health*, 15, pp. 33-51, 2023
- Q. Guan et al., The landscape of micron-scale particles including microplastics in human enclosed body fluids, *Journal of Hazardous Materials*, 442, 130138, 2023
- Å. Gustafsson, A. M. Kraus, A. Gorzsás, T. Lundh & P. Gerde, Isolation and characterization of a respirable particle fraction from residential house-dust, *Environmental Research*, 161, pp. 284-290, 2018. DOI: 10.1016/j.envres.2017.10.049
- L. Hayden, S. Strausborger & M. Lewin-Smith, Automated Particle Analysis Using Field-Emission Scanning Electron Microscopy (FE-SEM) and Energy Dispersive X-Ray Spectroscopy (EDS) to Characterize Inhaled Particulate Matter (PM) in Biopsied Lung Tissue, *Microscopy and Microanalysis*, 29(1), pp. 235-243, 2023. DOI: 10.1093/micmic/ozac015
- J. N. Hahladakis et al., An overview of chemical additives present in plastics: migration, release, fate and environmental impact during their use, disposal and recycling, *Journal of Hazardous Materials*, 344, pp. 179-199, 2018
- I. Hantoro et al., Microplastics in coastal areas and seafood: implications for food safety, *Food Additives & Contaminants: Part A*, 36, pp. 674-711, 2019
- N. B. Hartmann et al., Are we speaking the same language? Recommendations for a definition and categorization framework for plastic debris, *Environmental Science & Technology*, 53 (3), pp. 1039-1047, 2019
- N. B. Hartmann, S. Rist, J. Bodin, L. H. S. Jensen, S. N. Schmidt, P. Mayer, A. Meibom and A. Baun, Microplastics as vectors for environmental contaminants: Exploring sorption, desorption, and transfer to biota, *Integrated Environmental Assessment and Management*, 13, 488-493, 2017. DOI: 10.1002/ieam.1904
- Haskell Laboratory, Report No. 56-61, Medical Research Project No. 1080, three-week dietary study of polytetrafluoroethylene preparations in rats, 1961
- Haskell Laboratory, Three-Week Feeding Study in Rats with Polytetrafluoroethylene Resins (Teflon), Medical Research Project No. 1080, Report No. 224-68, 1968

- R. C. Heindel et al., Atmospheric Dust Deposition Varies by Season and Elevation in the Colorado Front Range, USA, *JGR Earth Surface*, 125 (5), April, 2020
- G. Herbst, *Das Lymphgefäßsystem und seine Verrichtung*, Vandenhoeck und Ruprecht, Göttingen, pp. 333-337, 1844
- E. Hermesen et al., Quality criteria for the analysis of microplastic in biota samples: a critical review, *Environmental Science & Technology*, 52 (18), pp. 10230-10240, 2018
- L. M. Hernandez et al., Plastic teabags release billions of microparticles and nanoparticles into tea, *Environmental Science & Technology*, 53 (21), pp. 12300-12310, 2019. DOI: 10.1021/acs.est.9b02540
- L. E. Hopkins et al., Repeated iron-soot exposure and nose-to-brain transport of inhaled ultrafine particles, *Toxicologic Pathology*, 46 (1), pp. 75-84, 2018
- A. Horenstein & R. G. Heimberg, Anxiety disorders and healthcare utilization: a systematic review, *Clinical Psychology Review*, 81, 101894, 2020
- P. He, F. Wang, G. Xi, et al., Association of microplastics in human cerebrospinal fluid with Alzheimer's disease-related changes, *Journal of Hazardous Materials*, 494, 138748, 2025
- IARC Monographs. Silica Dust, Crystalline, in the Form of Quartz or Cristobalite. <https://monographs.iarc.who.int/wp-content/uploads/2018/06/mono100C-14.pdf>
- IARC, Arsenic, Metals, Fibres and Dusts, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 100C, International Agency for Research on Cancer, Lyon, 2012
- IARC, Wood Dust and Formaldehyde, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 62, International Agency for Research on Cancer, Lyon, 1995
- S. Inoue et al., Near-infrared (NIR-II) fluorescent poly(ethylene terephthalate) nano-microplastics for in vivo tracking, *Journal of Nanoparticle Research*, 28, article 5, 2026. DOI: 10.1007/s11051-025-06526-5
- International Organization for Standardization, ISO/TR 21960:2020, Plastics - Environmental aspects - State of knowledge and methodologies, ISO, 2020
- J. P. A. Ioannidis, Why most published research findings are false, *PLOS Medicine*, 2 (8), e124, 2005
- L. C. Jenner et al., Detection of microplastics in human lung tissue using micro-FTIR spectroscopy, *Science of the Total Environment*, 831, 154907, 2022
- J.-M. Jeon et al., Biodegradation of low-density polyethylene, polystyrene, polyvinyl chloride, and urea formaldehyde resin buried under soil for over 32 years, *Journal of Applied Polymer Science*, 56, pp. 1789-1796, 1995
- C. Jiang, W. Wang, L. Du, G. Huang, C. McConaghy, S. Fineman & Y. Liu, Field Evaluation of an Automated Pollen Sensor, *International Journal of Environmental Research and Public Health*, 19 (11), 6444, 2022. DOI: 10.3390/ijerph19116444
- B. Jovanovic, Virgin microplastics are not causing imminent harm to fish after dietary exposure, *Marine Pollution Bulletin*, 130, pp. 123-131, 2018
- W. Jung, H. Abushukair, N. Y. Patil, et al., Spatial transcriptome mapping identifies Ppara-Anxa2 cross-talk in microplastic-induced hepatotoxicity, *Science Advances*, 12, eaec8681, 2026. DOI: 10.1126/sciadv.aec8681
- I. S. Jüngling et al., Challenges and solutions in the analysis of micro- and nanoplastics down to 500 nm with automated Raman microspectroscopy: suitable filters, accuracy in the detection, identification, and quantification, *Analytical and Bioanalytical Chemistry*, 2026. DOI: 10.1007/s00216-026-06567-2
- K. L. Kamerud et al., Stainless steel leaches nickel and chromium into foods during cooking, *Journal of Agricultural and Food Chemistry*, 61 (39), pp. 9495-9501, 2013
- A. Karami et al., The presence of microplastics in commercial salts from different countries, *Scientific Reports*, 7, 46173, 2017
- H. Khalid Ageel et al., Microplastics in settled indoor dust: Implications for human exposure, *Emerging Contaminants*, 11, 100506, 2025
- D. Kim, D. Kim, H.-K. Kim, et al., Organ-specific accumulation and toxicity analysis of orally administered polyethylene terephthalate microplastics, *Scientific Reports*, 15, 6616, 2025. DOI: 10.1038/s41598-025-91170-1
- J. S. Kim, Acute and subacute repeated oral toxicity study of fragmented microplastics in Sprague-Dawley rats, *Ecotoxicology and Environmental Safety*, 228, 112964, 2021. DOI: 10.1016/j.ecoenv.2021.112964

- J. Kleinteich et al., Microplastics Reduce Short-Term Effects of Environmental Contaminants Part II: Polyethylene Particles Decrease the Effect of Polycyclic Aromatic Hydrocarbons on Microorganisms, *International Journal of Environmental Research & Public Health*, 15 (2), 287, 2018
- A. A. Koelmans et al., Microplastic as a vector for chemicals in the aquatic environment: critical review and model-supported reinterpretation of empirical studies, *Environmental Science & Technology*, 50 (7), pp. 3315-3326, 2016
- A. A. Koelmans et al., Microplastics in freshwaters and drinking water: critical review and assessment of data quality, *Water Research*, 155, pp. 410-422, 2019. DOI: 10.1016/j.watres.2019.02.054
- A. A. Koelmans et al., Risk assessment of microplastic particles, *Nature Reviews Materials*, 7 (2), pp. 138-152, 2022. DOI: 10.1038/s41578-021-00411-y
- P. J. Kole et al., Wear and tear of tyres: a stealthy source of microplastics in the environment, *International Journal of Environmental Research and Public Health*, 14 (10), 1265, 2017
- V. Kopatz et al., Micro- and nanoplastics breach the blood-brain barrier (BBB): biomolecular corona's role revealed, *Nanomaterials*, 13, 1404, 2023
- M. Krachler & W. Shotyk, Trace and ultratrace metals in bottled waters: Survey of sources worldwide and comparison with refillable metal bottles, *Science of the Total Environment*, 407 (3), pp. 1089-1096, 2009
- J. Kuang et al., Brominated flame retardants in black plastic kitchen utensils: concentrations and human exposure implications, *Science of the Total Environment*, 610-611, pp. 1138-1146, 2018. DOI: 10.1016/j.scitotenv.2017.08.173
- C. Laforsch et al., Environmental Fate and Effects of Microplastics on Organisms, *Springer Handbook of Circular Plastics Economy*, Springer NY, p. 797, 2025
- T. Lane, V. N. de Ruijter, I. Wardani, M. Barchiesi & A. A. Koelmans, A QA/QC screening framework to assess studies reporting microplastic presence in human organs, tissues, and bodily fluids, *Environment International*, 214, 110429, 2026. DOI: 10.1016/j.envint.2026.110429
- M. E. LeFevre, R. Olivo, J. W. Vanderhoff & D. D. Joel, Accumulation of latex in Peyer's patches and its subsequent appearance in villi and mesenteric lymph nodes, *Proceedings of the Society for Experimental Biology and Medicine*, 159 (2), pp. 298-302, 1978
- M. E. LeFevre et al., Intestinal barrier to large particulates in mice, *Journal of Toxicology and Environmental Health*, 6 (4), pp. 691-704, 1980
- R. Lenz, K. Enders & T. G. Nielsen, Microplastic exposure studies should be environmentally realistic, *Proceedings of the National Academy of Sciences*, 113 (29), E4121-E4122, 2016. DOI: 10.1073/pnas.1606615113
- S. Lee, K.-K. Kang, S.-E. Sung, et al., In Vivo Toxicity and Pharmacokinetics of Polytetrafluoroethylene Microplastics in ICR Mice, *Polymers*, 14 (11), 2220, 2022. DOI: 10.3390/polym14112220
- H. A. Leslie et al., Discovery and quantification of plastic particle pollution in human blood, *Environment International*, 163, 107199, 2022
- Y. Li et al., Identification and quantification of nanoplastics (20-1000 nm) in a drinking water treatment plant using AFM-IR and Py-GC/MS, *Journal of Hazardous Materials*, 463, 132933, 2024
- D. Li et al., Microplastic release from the degradation of polypropylene feeding bottles during infant formula preparation, *Nature Food*, 1, pp. 746-754, 2020
- D. Licina et al., Clothing-mediated exposures to chemicals and particles, *Environmental Science & Technology*, 53 (10), pp. 5559-5575, 2019. DOI: 10.1021/acs.est.9b00272
- T.C. Liebert et al., Subcutaneous Implants of Polypropylene Filaments, *Journal of Biomedical Materials Research*, 10 (6), pp 939-951, 1976
- R. Li, F. Lin, X. Zhao, et al., Microplastics and nanoplastics in brain tumours and the healthy human brain, *Nature Health*, 1, pp. 633-646, 2026
- X. Li, Y. Wang, Z. Feng, et al., Microplastics and nanoplastics in matched human blood, bone, and intervertebral discs: Accumulation patterns and risks, *npj Emerging Contaminants*, 2, 22, 2026
- D. Lithner, A. Larsson & G. Dave, Environmental and health hazard ranking and assessment of plastic polymers based on chemical composition, *Science of the Total Environment*, 409, pp. 3309-3324, 2011

- S. Liu et al., Detection of various microplastics in placentas, meconium, infant feces, breastmilk and infant formula: a pilot prospective study, *Science of the Total Environment*, 854, 2023
- N.M. Liu et al., Evidence for the presence of air pollution nanoparticles in placental tissue cells, *Science of the Total Environment*, 751, 142235, 2021
- M. Liu et al., From e-waste to living space: Flame retardants contaminating household items add to concern about plastic recycling, *Chemosphere*, 365, 143319, 2024. DOI: 10.1016/j.chemosphere.2024.143319
- X. Liu et al., Size- and shape-dependent effects of titanium dioxide nanoparticles on the permeabilization of the blood-brain barrier, *Journal of Materials Chemistry B*, 5, pp. 1437-1446, 2017
- J. Lofty, D. Valero, C. A. M. E. Wilson, M. J. Franca and P. Ouro, Microplastic and natural sediment in bed load saltation: Material does not dictate the fate, *Water Research*, 243, 120329, 2023. DOI: 10.1016/j.watres.2023.120329
- O. M. Lönnstedt & P. Eklöv, Environmentally relevant concentrations of microplastic particles influence larval fish ecology, *Science*, 352 (6290), pp. 1213-1216, 2016. Retracted. DOI: 10.1126/science.aad8828
- Y. Lu et al., Uptake and accumulation of polystyrene microplastics in zebrafish (*Danio rerio*) and toxic effects in liver, *Environmental Science & Technology*, 50 (7), pp. 4054-4060, 2016. DOI: 10.1021/acs.est.6b00183
- X. Lu et al., Chronic exposure to high-density polyethylene microplastic through feeding alters the nutrient metabolism of juvenile yellow perch (*Perca flavescens*), *Animal Nutrition* 9, pp. 143-158, 2022
- W. Lu, X. Xu, L. Fan, et al., Association between exposure to microplastics and lipid disorders: A case-control study, *Journal of Advanced Research*, 2026
- T. Maes et al., A rapid-screening approach to detect and quantify microplastics based on fluorescent tagging with Nile Red, *Scientific Reports*, 7, 44501, 2017
- K. C. Makris, S. S. Andra & G. Botsaris, Pipe scales and biofilms in drinking-water distribution systems: undermining finished water quality, *Critical Reviews in Environmental Science and Technology*, 44 (13), pp. 1477-1523, 2014. DOI: 10.1080/10643389.2013.790746
- K. V. Malafeev et al., Understanding the Impact of Biodegradable Microplastics on Living Organisms Entering the Food Chain: A Review, *Polymers*, 15 (18), 3680, 2023. DOI: 10.3390/polym15183680
- B. Manjunatha et al., Developmental toxicity induced by particulate matter (PM 2.5) in zebrafish (*Danio rerio*) model, *Aquatic Toxicology*, 238, 105928, 2021
- R. Marfella et al., Microplastics and nanoplastics in atheromas and cardiovascular events, *New England Journal of Medicine*, 390, pp. 900-910, 2024. DOI: 10.1056/NEJMoa2309822
- D. Materic, Nanoplastics measurements must have appropriate blanks, *Proceedings of the National Academy of Sciences*, 2024
- K. Mattsson, M. T. Ekvall, L.-A. Hansson, S. Linse, A. Malmendal & T. Cedervall, Altered behavior, physiology, and metabolism in fish exposed to polystyrene nanoparticles, *Environmental Science & Technology*, 49 (1), pp. 553-561, 2015. DOI: 10.1021/es5053655
- L. Maurizi, L. S. Sanchez, A. Vianello, A. H. Nielsen & J. Vollertsen, Every breath you take: High concentration of breathable microplastics in indoor environments, *Chemosphere*, 361, 142553, 2024. DOI: 10.1016/j.chemosphere.2024.142553
- B. S. McEwen, Physiology and neurobiology of stress and adaptation: central role of the brain, *Physiological Reviews*, 87, pp. 873-904, 2007
- B. S. McEwen, Stress, adaptation, and disease: allostasis and allostatic load, *Annals of the New York Academy of Sciences*, 840, pp. 33-44, 1998
- J. K. McIntyre et al., "Tire Wear Particle Leachate Recreates an Urban Runoff Mortality Syndrome in Coho but Not Chum Salmon." *Environmental Science & Technology*, 55, pp. 11767-11774, 2021 DOI: 10.1021/acs.est.1c03569
- F. Meng et al., Replacing plastics with alternatives is worse for greenhouse gas emissions in most cases, *Environmental Science & Technology*, 58, pp. 2716-2727, 2024. DOI: 10.1021/acs.est.3c05191
- J. A. Merski et al., Oral toxicity and bacterial mutagenicity studies with a spunbond polyethylene and polyethylene terephthalate polymer fabric, *International Journal of Toxicology*, 27 (5), pp. 387-395, 2008. DOI: 10.1080/10915810802408729
- M. Meyns et al., Multi-feature round silicon membrane filters enable fractionation and analysis of small micro- and nanoplastics with Raman spectroscopy and nano-FTIR, *Analytical Methods*, 15, pp. 606-617, 2023

- M. Michelaraki, Potential for Nile red dye-based analysis of microplastics from oceanic samples, In Proceedings of the 2020 Global OCEANS: Singapore - U.S. Gulf Coast conference, 2020
- M. R. Miller, J. B. Raftis, J. P. Langrish, et al., Inhaled Nanoparticles Accumulate at Sites of Vascular Disease, *ACS Nano*, 11(5), pp. 4542-4552, 2017. DOI: 10.1021/acsnano.6b08551. Correction: *ACS Nano*, 11, pp. 10623-10624. DOI: 10.1021/acsnano.7b06327
- S. M. Mintenig et al., Low numbers of microplastics detected in drinking water from ground water sources, *Science of the Total Environment*, 648, pp. 631-635, 2019
- N. H. Mohamed Nor, M. Kooi, N. J. Diepens & A. A. Koelmans, Lifetime accumulation of microplastic in children and adults, *Environmental Science & Technology*, 55 (8), pp. 5084-5096, 2021. DOI: 10.1021/acs.est.0c07384
- S. Mohammadi et al., Toxic metal contamination in edible salts and its attributed human health risks: a systematic review and meta-analysis, *Environmental Science and Pollution Research*, 32, pp. 4313-4324, 2025
- M. Mohan et al., Screening for microplastics in drinking water and its toxicity profiling in zebrafish, *Chemosphere*, 341, 139882, 2023
- N. Mohanan et al., Microbial and Enzymatic Degradation of Synthetic Plastics, *Frontiers in Microbiology*, 11, 580709, 2020
- F. A. Monikh, D. Materić, E. Valsami-Jones, H.-P. Grossart, K. Altmann, R. Holzinger, I. Lynch, J. Stubenrauch & W. Peijnenburg, Challenges in studying microplastics in human brain, *Nature Medicine*, 31 (12), pp. 4034-4035, 2025. DOI: 10.1038/s41591-025-04045-3
- L. Montano et al., Microplastics in human ovarian follicular fluid : An emerging threat to female fertility, *Ecotoxicology and Environmental Safety*, 291, 117868, 2025
- R. Morris et al., Repeated Exposure to Urban Particulate Matter (UPM) Induces Oxidative Stress and Impairs Development in Zebrafish Embryos, *IBRO Neuroscience Reports* 15, S67-S466, 2023
- M. L. Moser & D. S. Lee, A fourteen-year survey of plastic ingestion by western North Atlantic seabirds, *Colonial Waterbirds*, 15 (1), pp. 83-94, 1992. DOI: 10.2307/1521357
- N. Mrosovsky, G. D. Ryan & M. C. James, Leatherback turtles: the menace of plastic, *Marine Pollution Bulletin*, 58 (2), pp. 287-289, 2009. DOI: 10.1016/j.marpolbul.2008.10.018
- A. Mukherjee & M. Agrawal, World air particulate matter: sources, distribution and health effects, *Environmental Chemistry Letters*, 15, pp 283-309, 2017
- F. D. Müller et al., Metal release from coffee machines and electric kettles, *Food Additives & Contaminants: Part A*, 32 (11), pp. 1959-1964, 2015
- R. Naftalovich et al., Polytetrafluoroethylene Ingestion as a Way to Increase Food Volume and Hence Satiety Without Increasing Calorie Content, *Journal of Diabetes Science & Technology*, 10 (4), pp. 971-976, 2016
- I. E. Napper & R. C. Thompson, Release of synthetic microplastic plastic fibres from domestic washing machines: effects of fabric type and washing conditions, *Marine Pollution Bulletin*, 112 (1-2), pp. 39-45, 2016
- A. Nemmar et al., Ultrafine particles affect experimental thrombosis in an in vivo hamster model, *American Journal of Respiratory and Critical Care Medicine*, 166, pp. 998-1004, 2002
- A. J. Nihart et al., Bioaccumulation of microplastics in decedent human brains, *Nature Medicine*, 2025. DOI: 10.1038/s41591-024-03453-1
- NIOSH. NIOSH Pocket Guide to Chemical Hazards: Particulates not otherwise regulated, Centers for Disease Control and Prevention / National Institute for Occupational Safety and Health. The entry reports OSHA PELs and directs users to Appendix D for NIOSH REL status. <https://www.cdc.gov/niosh/npg/npgd0480.html>
- NIOSH. Appendix D - Substances with No Established RELs, Centers for Disease Control and Prevention / National Institute for Occupational Safety and Health. <https://www.cdc.gov/niosh/npg/nengapdx.html>
- K-C. Nordby et al., Exposure to thoracic dust, airway symptoms and lung function in cement production workers, *Eur Respir J*, 38, pp. 1278-1286, 2011
- G. Oberdörster, Z. Sharp, V. Atudorei, A. Elder, R. Gelein, W. Kreyling & C. Cox, Extrapulmonary translocation of ultrafine carbon particles following whole-body inhalation exposure of rats, *Journal of Toxicology and Environmental Health, Part A*, 65 (20), pp. 1531-1543, 2002

- G. Oberdörster, Z. Sharp, V. Atudorei, A. Elder, R. Gelein, A. Lunts, W. Kreyling & C. Cox, Translocation of inhaled ultrafine particles to the brain, *Inhalation Toxicology*, 16 (6-7), pp. 437-445, 2004
- G. Oberdörster, E. Oberdörster & J. Oberdörster, Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles, *Environmental Health Perspectives*, 113 (7), pp. 823-839, 2005
- OECD, Global Plastics Outlook: Economic Drivers, Environmental Impacts and Policy Options, OECD Publishing, Paris, 2022
- OECD, Policies to Reduce Microplastics Pollution in Water: Focus on Textiles and Tyres, OECD Publishing, Paris, 2021. DOI: 10.1787/7ec7e5ef-en
- M. Ogonowski, Z. Gerdes & E. Gorokhova, What we know and what we think we know about microplastic effects: A critical perspective, *Current Opinion in Environmental Science & Health*, 1, pp. 41-46, 2018. DOI: 10.1016/j.coesh.2017.09.001
- A. Omidianidost et al., Occupational Exposure to Respirable Dust, Crystalline Silica and Its Pulmonary Effects among Workers of a Cement Factory in Kermanshah, Iran, *National Research Institute of Tuberculosis and Lung Disease, Iran*, 18 (2), 157-162, 2019
- OSHA. Particulates not otherwise regulated, total and respirable dust (PNOR), OSHA Chemical Data, Occupational Safety and Health Administration, 2023. <https://www.osha.gov/chemicaldata/801>
- OSHA. Permissible Exposure Limits – Annotated Table Z-1, Occupational Safety and Health Administration. <https://www.osha.gov/annotated-pels/table-z-1>
- C. Ostle et al., The rise in ocean plastics evidenced from a 60-year time series, *Nature Communications*, 10, 1622, 2019
- P. Paolisso, L. Scisciola, M. Belmonte, et al., Micro- and nano-plastics in the coronary circulation and air pollution exposure in ischaemic heart disease presentation, *European Heart Journal*, ehag447, 2026
- M. Perez et al., Particulate Matter in Injectable Drugs: Evaluation of Risks to Patients, *Pharmaceutical Technology in Hospital Pharmacy*, 1 (2), 2016
- Z. Piao, A. A. A. Boakye & Y. Yao, Environmental impacts of biodegradable microplastics, *Nature Chemical Engineering*, 1, pp. 661-669, 2024. DOI: 10.1038/s44286-024-00127-0
- M. Pivokonsky et al., Microplastics in drinking water treatment: current knowledge and research needs, *Science of the Total Environment*, 667, pp. 730-740, 2020
- M. Pivokonsky et al., Occurrence of microplastics in raw and treated drinking water, *Science of the Total Environment*, 643, pp. 1644-1651, 2018
- Plastics Research Council. Microplastics toxicity, exposure, and accumulation science. <https://plasticsresearchcouncil.com/microplastics-toxicity-exposure-and-accumulation-science/>
- Plastics Research Council. Plastics are usually the lowest-impact option; alternatives are often worse. <https://plasticsresearchcouncil.com/plastics-are-usually-the-lowest-impact-option-alternatives-are-actually-worse/>
- Plastics Research Council. Summary of comprehensive review of the science. <https://plasticsresearchcouncil.com/summary-of-comprehensive-review-of-the-science/>
- Plastics Research Council, Microplastics Science 2026: Occurrence, Exposure, Analytical Reliability, Toxicology and Risk - A Comprehensive Critical Evidence Synthesis of 1,661 Canonical Evidence Records, Public Release Version 1.0, August 2026
- M. Pletz, Ingested microplastics: Do humans eat one credit card per week? *Journal of Hazardous Materials Letters*, 3, 100071, 2022. DOI: 10.1016/j.hazl.2022.100071
- J. J. Powell et al., Origin and fate of dietary nanoparticles and microparticles in the gastrointestinal tract, *Journal of Autoimmunity*, 34 (3), pp. 226-233, 2010. DOI: 10.1016/j.jaut.2009.11.006
- J. C. Prata et al., Airborne microplastics: consequences to human health? *Environmental Pollution*, 234, pp. 115-126, 2018
- J. C. Prata et al., Microplastics in Internal Tissues of Companion Animals from Urban Environments, *Animals*, 12 (15), p 1979, 2022
- M. Prust, J. Meijer & R. H. S. Westerink, The plastic brain: neurotoxicity of micro- and nanoplastics, *Particle and Fibre Toxicology*, 17, 24, 2020
- N. Qian et al., Rapid single-particle chemical imaging of nanoplastics by SRS microscopy, *Proceedings of the National Academy of Sciences*, 121, e2300582121, 2024. DOI: 10.1073/pnas.2300582121
- A. Ragusa et al., Plasticenta: first evidence of microplastics in human placenta, *Environment International*, 146, 106274, 2021

- A. Ragusa et al., Raman microspectroscopy detection and characterisation of microplastics in human breastmilk, *Polymers*, 14, 2700, 2022
- A. M. N. A. A. Rahman, A. Rusli, M. K. Abdullah et al., A review of microplastic surface interactions in water and potential capturing methods, *Water Science and Engineering*, 17 (4), pp. 361-370, 2024. DOI: 10.1016/j.wse.2023.11.008
- A. H. Ramani et al., Effect of Exposure to Cement Dust among the Workers: An Evaluation of Health Related Complications, *J Med Sci*, 6(6), 1159-1162, 2018
- V. P. Ranjan et al., Microplastics and other harmful substances released from disposable paper cups into hot water, *Journal of Hazardous Materials*, 404, 124118, 2021
- C. Rauert et al., Assessing the efficacy of pyrolysis-gas chromatography-mass spectrometry for nanoplastic and microplastic analysis in human blood, *Environmental Science & Technology*, 59 (4), pp. 1984-1994, 2025. DOI: 10.1021/acs.est.4c12599
- C. Rauert et al., Extraction and pyrolysis-GC-MS analysis of polyethylene in samples with medium to high lipid content, *Journal of Environmental Exposure Assessment*, 2022
- S. Rehse et al., Microplastics Reduce Short-Term Effects of Environmental Contaminants Part I: Effects of Bisphenol A on Freshwater Zooplankton Are Lower in Presence of Polyamide Particles, *International Journal of Environmental Research & Public Health*, 15 (2), 280, 2018
- C. Reimann et al., Bottled drinking water: water contamination from bottle materials (glass, hard PET, soft PET), the influence of colour and acidification, *Applied Geochemistry*, 25, pp. 1030-1046, 2010
- J.-M. Restrepo-Flórez et al., Microbial degradation & deterioration of polyethylene - A review, *International Biodeterioration & Biodegradation*, 88, pp 83-90, 2014
- M. D. Robards, J. F. Piatt & K. D. Wohl, Increasing frequency of plastic particles ingested by seabirds in the subarctic North Pacific, *Marine Pollution Bulletin*, 30 (2), pp. 151-157, 1995. DOI: 10.1016/0025-326X(94)00121-0
- J. M. Rotchell et al., Detection of microplastics in human saphenous vein tissue using micro-FTIR: a pilot study, *PLOS ONE*, 18, e0280594, 2023
- F. S. Ruggeri et al., Single nanoplastics detection and nano-chemical analysis of surface degradation in commercial bottled water, *Research Square preprint*, 2025. DOI: 10.21203/rs.3.rs-6719488/v1
- G. Ryan & C. L. Moloney, Plastic and other artefacts on South African beaches: temporal trends in abundance and composition, *South African Journal of Science*, 86, pp. 450-452, 1990
- T. Salthammer, Microplastics and their additives in the indoor environment, *Angewandte Chemie International Edition*, 61, e202205713, 2022. DOI: 10.1002/anie.202205713
- SAPEA, Science Advice for Policy by European Academies, A Scientific Perspective on Microplastics in Nature and Society, SAPEA, Berlin, 2019. DOI: 10.26356/microplastics
- L. Schöpfer et al., Microplastics persist in an arable soil but do not affect soil microbial biomass, enzyme activities, and crop yield, *Journal of Plant Nutrition & Soil Science*, 185 (6), pp. 836-849, 2022
- P. Schwabl et al., Detection of various microplastics in human stool: a prospective case series, *Annals of Internal Medicine*, 171 (7), pp. 453-457, 2019
- D. Schymanski et al., Analysis of microplastics in water by micro-Raman spectroscopy: release of plastic particles from different packaging into mineral water, *Water Research*, 129, pp. 154-162, 2018
- Science Media Centre, Expert reaction to a study investigating the accumulation of microplastics in human organs, Feb. 3rd 2025 www.sciencemediacentre.org/expert-reaction-to-a-study-investigating-the-accumulation-of-microplastics-in-human-organs/
- D. A. Seli et al., High NaCl Concentrations in Water Are Associated with Developmental Abnormalities and Altered Gene Expression in Zebrafish, *International Journal of Molecular Sciences*, 25 (7), 4104, 2024
- M. Semmler-Behnke et al., Biodistribution of 1.4- and 18-nm gold particles in rats, *Small*, 4, pp. 2108-2111, 2008
- K. Senathirajah et al., Estimation of the mass of microplastics ingested - A pivotal first step towards human health risk assessment, *Journal of Hazardous Materials*, 404, 124004, 2021
- S. Shan et al., Polystyrene nanoplastics penetrate across the blood-brain barrier and induce activation of microglia in the brain of mice, *Chemosphere*, 298, 134261, 2022

- S. A. Shansal et al., Consequences of cement dust exposure on pulmonary function in cement factory workers, *Am J Ind Med.*, 64, pp. 192–197, 2021
- S. Sharma, J. Mohler, S. D. Mahajan, S. A. Schwartz, L. Bruggemann and R. Aalinkel, Microbial biofilm: A review on formation, infection, antibiotic resistance, control measures, and innovative treatment, *Microorganisms*, 11, 1614, 2023. DOI: 10.3390/microorganisms11061614
- A. Shimada, T. Kawamura, H. Mutoh, H. Hirose, A. Morita & T. Tsubota, Translocation pathway of the intratracheally instilled ultrafine particles from the lung into the blood circulation in the mouse, *Toxicologic Pathology*, 34 (7), pp. 949-957, 2006. DOI: 10.1080/01926230601080502
- N. Simantris, Single-use plastic or paper products? A dilemma that requires societal change, *Cleaner Waste Systems*, 7, 100128, 2024
- S. Singh et al., Microplastic exposure disparities in California communities through bottled water consumption, *Microplastics & Nanoplastics*, 5 (43), 2025
- M. Smith et al., Microplastics in seafood and the implications for human health, *Current Environmental Health Reports*, 5, pp. 375-386, 2018
- Y. K. Song et al., Rapid production of micro- and nanoplastics by fragmentation of expanded polystyrene exposed to sunlight, *Environmental Science & Technology*, 54 (18), pp. 11191–11200, 2020. DOI: 10.1021/acs.est.0c02288
- L. B. Spear, D. G. Ainley & C. A. Ribic, Incidence of plastic in seabirds from the tropical Pacific, 1984–1991: relation with distribution of species, sex, age, season, year and body weight, *Marine Environmental Research*, 40 (2), pp. 123–146, 1995. DOI: 10.1016/0141-1136(94)00140-K
- T. Stanton et al., Exploring the efficacy of Nile Red in microplastic quantification: a costaining approach, *Environmental Science & Technology Letters*, 6 (10), pp. 606-611, 2019
- X. Sun et al., Microplastic transportation in a typical drinking water supply: from raw water to household water, *Water*, 16 (11), 1567, 2024. DOI: 10.3390/w16111567
- V. Stock et al., Uptake and cellular effects of PE, PP, PET and PVC microplastic particles, *Toxicology in Vitro*, 70, 105021, 2021. DOI: 10.1016/j.tiv.2020.105021
- J. A. Styles & J. Wilson, Comparison between in vitro toxicity of polymer and mineral dusts and their fibrogenicity, *The Annals of Occupational Hygiene*, November, 16 (3), pp 241-250, 1973
- G. Suaria et al., Microfibers in oceanic surface waters: a global characterization, *Science Advances*, 6 (23), eaay8493, 2020
- S. F. Suglia, A. Gryparis, R. O. Wright, J. Schwartz & R. J. Wright, Association of black carbon with cognition among children in a prospective birth cohort study, *American Journal of Epidemiology*, 167 (3), pp. 280-286, 2008. DOI: 10.1093/aje/kwm308
- R. Sussarellu et al., Oyster reproduction is affected by exposure to polystyrene microplastics, *Proceedings of the National Academy of Sciences*, 113 (9), pp. 2430-2435, 2016. DOI: 10.1073/pnas.1519019113
- S. Y. Tan et al., A review of heavy metals in indoor dust and its human health-risk implications, *Reviews on Environmental Health*, 31 (4), pp. 447-456, 2016
- E. L. Teuten et al., Transport and release of chemicals from plastics to the environment and to wildlife, *Philosophical Transactions of the Royal Society B*, 364, pp. 2027-2045, 2009
- UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment, Statement on the potential risks from inhalation exposure to microplastics, 2024
- W. C. Thomas, L. F. McGrath, K. A. Baarson, C. S. Auletta, I. W. Daly & R. F. McConnell, Subchronic oral toxicity of cellulose acetate in rats, *Food and Chemical Toxicology*, 29 (7), pp. 453-458, 1991. DOI: 10.1016/0278-6915(91)90090-T
- Z. Tian et al., A ubiquitous tire rubber-derived chemical induces acute mortality in coho salmon, *Science*, 371, pp. 185-189, 2021
- Z. Tian, M. Gonzalez, C. A. Rideout, et al., 6PPD-Quinone: Revised Toxicity Assessment and Quantification with a Commercial Standard, *Environmental Science & Technology Letters*, 9(2), pp. 140-146, 2022. DOI: 10.1021/acs.estlett.1c00910
- A. Torres-Agulló, A. Karanasiou, T. Moreno & S. Lacorte, Airborne microplastic particle concentrations and characterization in indoor urban microenvironments, *Environmental Pollution*, 308, 119707, 2022
- B. Toussaint et al., Review of micro- and nanoplastic contamination in the food chain, *Food Additives & Contaminants: Part A*, 36, pp. 639-673, 2019

H. A. A. Tran, J. J. Ng, D. H. Tang, et al., Health impacts of micro- and nanoplastics in humans: systematic review of in vivo evidence, *Environmental Health*, 25, 26, 2026

A. Turner, Heavy metals in the glass and enamels of consumer container bottles, *Environmental Science & Technology*, 53, pp. 8398-8404, 2019

A. Turner, Paint particles in the marine environment: an overlooked component of microplastics, *Water Research X*, 12, 100110, 2021. DOI: 10.1016/j.wroa.2021.100110

U.S. Environmental Protection Agency, Indoor Air Quality, EPA Report on the Environment. URL: <https://www.epa.gov/report-environment/indoor-air-quality>

U.S. Environmental Protection Agency, Microplastics Research, EPA, 2024/2025/2026. URL: <https://www.epa.gov/water-research/microplastics-research>

U.S. Environmental Protection Agency, Draft Contaminant Candidate List 6 (CCL 6), 2026. Inclusion on the CCL is an information-collection and regulatory-prioritization step and is not a regulatory determination of harm

U.S. Food and Drug Administration, Microplastics and Nanoplastics in Foods, FDA, 2024. URL: <https://www.fda.gov/food/environmental-contaminants-food/microplastics-and-nanoplastics-foods>

U.S. Food and Drug Administration, Human Foods Program 2026 Priority Deliverables, 10 February 2026. FDA identifies research to develop accurate, reproducible methods to detect, quantify and characterize microplastics in human food; this is a method-development priority rather than a finding that current food levels cause harm

UNEP, From Pollution to Solution: A Global Assessment of Marine Litter and Plastic Pollution, United Nations Environment Programme, Nairobi, 2021

A. Vaksmaa et al., Biodegradation of polyethylene by the marine fungus *Parengyodontium album*, *Science of the Total Environment*, 934, 172819, 2024

J. A. van Franeker et al., Monitoring plastic ingestion by the northern fulmar *Fulmarus glacialis* in the North Sea, *Environmental Pollution*, 159 (10), pp. 2609–2615, 2011. DOI: 10.1016/j.envpol.2011.06.008

J. A. van Franeker & A. Meijboom, Litter NSV: Marine litter monitoring by Northern Fulmars: A pilot study, Alterra-Rapport 401, Alterra, Wageningen, 2002. DOI: 10.18174/45695

A. Velasco et al., Contamination and removal efficiency of microplastics and synthetic fibres in a conventional drinking water treatment plant in Geneva, Switzerland, *Science of the Total Environment*, 880, 2023

M.I.S. Verissimo et al., The evaluation of copper contamination of food cooked in copper pans using a piezoelectric quartz crystal resonator, *Sensors and Actuators B* 111–112, pp. 587–591 2005

A. Vianello et al., Simulating human exposure to indoor airborne microplastics using a Breathing Thermal Manikin, *Scientific Reports*, 9, 8670, 2019

N. Voulvoulis et al., Examining Material Evidence – The Carbon Footprint, Centre for Environmental Policy, Imperial College London & Veolia UK, ACC, 2019

C. Völker, J. Kramm & M. Wagner, On the creation of risk: framing of microplastics risks in science and media, *Global Challenges*, 4 (6), 1900010, 2020

G. Volkheimer & F. H. Schulz, The phenomenon of persorption, *Digestion*, 1 (4), pp. 213-218, 1968. DOI: 10.1159/000196856

G. Volkheimer, Passage of particles through the wall of the gastrointestinal tract, *Environmental Health Perspectives*, 9, pp. 215-225, 1974. DOI: 10.1289/ehp.749215

S. Wagner et al., Tire wear particles in the aquatic environment - A review on generation, analysis, occurrence, fate and effects, *Water Research*, 139, pp. 83-100, 2018

F. Wang, C. S. Wong, D. Chen, X. Lu, F. Wang and E. Y. Zeng, Interaction of toxic chemicals with microplastics: A critical review, *Water Research*, 139, 208–219, 2018. DOI: 10.1016/j.watres.2018.04.003

P. Wardrop, J. Shimeta, D. Nugegoda, P. D. Morrison, A. Miranda, M. Tang & B. O. Clarke, Chemical pollutants sorbed to ingested microbeads from personal care products accumulate in fish, *Environmental Science & Technology*, 50 (7), pp. 4037-4044, 2016. DOI: 10.1021/acs.est.5b06280

- An Introduction to Materials in Medicine, 4th ed., Edited by William R. Wagner, Shelly E. Sakiyama-Elbert, Guigen Zhang, Michael J. Yaszemski, and Buddy D. Ratner. Academic Press / Elsevier, 2020
- J. E. Weinstein et al., From Macroplastic to Microplastic: Degradation of High-Density Polyethylene, Polypropylene, and Polystyrene in Salt Marsh Habitat, *Environmental Toxicology & Chemistry*, 35 (7), pp. 1632-1640, 2016
- F. Welle & R. Franz, Microplastic in bottled natural mineral water - literature review and considerations on exposure and risk assessment, *Food Additives & Contaminants: Part A*, 35 (12), pp. 2482-2492, 2018
- World Health Organization, Dietary and Inhalation Exposure to Nano- and Microplastic Particles and Potential Implications for Human Health, World Health Organization, Geneva, 2022. ISBN 978-92-4-005460-8. URL: [WHO 2022 publication page](#)
- World Health Organization, Microplastics in Drinking-Water, World Health Organization, Geneva, 2019. ISBN 978-92-4-151619-8. URL: [WHO 2019 publication page](#)
- S. Wieland et al., From properties to toxicity: comparing microplastics to other airborne microparticles, *Journal of Hazardous Materials*, 428, 128151, 2022
- P. Wiercik et al., Characterisation of Scale Deposits in Drinking Water Pipes by FTIR and ICP-OES, *Materials*, 19, 1223, 2026
- D. Wu et al., Pigment microparticles and microplastics found in human thrombi based on Raman spectral evidence, *Journal of Advanced Research*, 49, pp 141–150, 2023
- F. Wu, et al., Microplastic contamination in the dominant crabs at the intertidal zone of Chongming Island, Yangtze Estuary, *Science of the Total Environment*, 896, 165258, 2023
- J. Xu, J. Qu, H. Jin & W. Mao, Associations between microplastics in human feces and colorectal cancer risk, *Journal of Hazardous Materials*, 495, 139099, 2025
- H. Yadav et al., Cutting boards: an overlooked source of microplastics in human food?, *Environmental Science & Technology*, 57 (22), pp. 8225-8235, 2023. DOI: 10.1021/acs.est.3c00924
- N. Yakovenko et al., Human exposure to PM10 microplastics in indoor air, *PLOS ONE*, 20 (7), e0328011, 2025
- H. Yang, Z. Chen, L. Kong, H. Xing, Q. Yang & J. Wu, A review of eco-corona formation on micro/nanoplastics and its effects on stability, bioavailability, and toxicity, *Water*, 17, 1124, 2025. DOI: 10.3390/w17081124
- L. Yang, et al., Microplastics in drinking water: A review on methods, occurrence, sources, and potential risks assessment. *Environmental Pollution*, 347, 123857, 2024. DOI: 10.1016/j.envpol.2024.123857
- H. Yariibeygi et al., The impact of stress on body function: a review, *EXCLI Journal*, 16, pp. 1057-1072, 2017
- K. Yorioka et al., Glass Ampoules: Risks and Benefits, *Journal of Food and Drug Analysis*, 17 (3), pp. 225-228, 2009
- Z. Yu et al., Drinking boiled tap water reduces human intake of nanoplastics and microplastics, *Environmental Science & Technology Letters*, 11, pp. 273-279, 2024. DOI: 10.1021/acs.estlett.4c00081
- N. Zhang, Y. B. Li, H. R. He, J. F. Zhang & G. S. Ma, You are what you eat: Microplastics in the feces of young men living in Beijing, *Science of the Total Environment*, 767, 144345, 2021. DOI: 10.1016/j.scitotenv.2020.144345
- A. Zhao, J. Sun and Y. Liu, Understanding bacterial biofilms: From definition to treatment strategies, *Frontiers in Cellular and Infection Microbiology*, 13, 1137947, 2023. DOI: 10.3389/fcimb.2023.1137947
- Q. Zhao et al., Detection and characterization of microplastics in the human testis and semen, *Science of the Total Environment*, 877, 162713, 2023
- L. Zhu et al., Identification of microplastics in human placenta using laser direct infrared spectroscopy, *Science of the Total Environment*, 856, 159060, 2023
- S. Ziajahromi et al., Effects of polyethylene microplastics on the acute toxicity of a synthetic pyrethroid to midge larvae (*Chironomus tepperi*) in synthetic and river water, *Science of the Total Environment*, 671, pp 971-975, 2019



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