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MICROPLASTICS SCIENCE

A COMPREHENSIVE META-ANALYSIS

Exposure, Human Health and Risk

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Occurrence, Exposure, Analytical Reliability, Toxicology and Risk - A Comprehensive
Critical Evidence Synthesis of 1,661 Canonical Evidence Records

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Version-controlled evidence corpus: 1,661 canonical records spanning 1968-2026

Contents

Contents	3
Abstract.....	4
1. Executive Summary	8
2. Scope and Methods.....	11
3. Definitions and Analytical Boundaries	17
4. Evidence-Quality and Appraisal Framework.....	20
5. Environmental Occurrence	22
6. Temporal Trends	24
7. Airborne Exposure	25
8. Food and Beverage Exposure	28
9. Drinking Water and Tap Water	30
10. Human Ingestion.....	33
11. Human Inhalation.....	35
12. Absorption, Clearance and Retention	38
13. Human Tissue and Biomonitoring Evidence.....	41
14. Analytical Reliability and False Positives	47
15. Animal Toxicology.....	52
16. Standardized Oral Toxicology and Highest Doses Without Detected Adverse Effects	54
17. Dose and Exposure Realism	59
18. Human Epidemiology and Clinical Associations.....	66
19. Ecotoxicology.....	70
20. Soil, Crops and Terrestrial Systems	72
21. Aquatic Systems	74
22. Wastewater and Treatment	76
23. Fragmentation, Weathering and Fate	78
24. Natural and Nonplastic Particle Comparisons	80
25. Additives, Leachates and Sorbed Chemicals	87
26. Modeling Versus Measured Evidence	90
27. Removal Versus Destruction	92
28. Risk Characterization.....	94
29. Major Evidence Gaps	101
30. Conclusions	104
31. Recommendations for Researchers, Regulators and Communicators.....	106
Appendix A. Methodology and Audit Boundaries	107
Appendix B. Conclusion Stress-Test Matrix	110
Appendix C. Source Provenance and Unresolved Records	116
Appendix D. Cited Record Bibliography	117
Appendix E. Quantitative Compatibility and Conversion Rules	124
Appendix F. Comparative Particle Context and Cumulative Weight-of-Evidence Updating	126
Appendix G. Current Agency Positions and Review Status	128
Appendix H. Cited and Conclusion-Controlling Study Summaries	130
Appendix I. Search History, Screening and Exclusion Framework.....	160
Appendix J. Structured Internal Consistency and Adversarial Audit.....	165

Abstract

This report evaluates microplastic occurrence, ordinary human exposure, analytical reliability, toxicology and risk using 1,661 canonical evidence records, including 643 independent primary-evidence records, with targeted updates through 18 August 2026. Evidence is classified by role, design, analytical validity, exposure realism, control quality, plastic specificity and risk relevance. AI-assisted tools supported structured extraction and consistency checks; the author retained responsibility for every scientific judgment. Methodologically incompatible studies were not statistically pooled.

Dust and particles are everywhere and cannot be avoided. Air, food, water, soil, household dust and human tissues contain complex mixtures of mineral, biological, cellulosic, combustion-derived, metal-containing and synthetic particles. Plastic is one small component of that normal background, so it is found widely just as other dust is found widely. Detection establishes occurrence within a defined analytical window; it does not establish absorbed dose, retained burden, injury or disease. Reported abundance remains strongly method-bounded.

A 2026 QA/QC audit evaluated 133 datasets from 101 human tissue or bodily-fluid studies. None met all 14 essential criteria for direct quantitative risk assessment. The studies remain useful for exploratory occurrence and method development, but cannot establish representative body burden, organ distribution or particle-specific dose-response. This limits one evidence stream rather than making risk unknowable: broader evaluation still draws on measured exposure, toxicology, kinetics, comparative particle evidence and epidemiology.

A global atmospheric synthesis compiled 2,782 measurements from 76 studies and reported a median of 0.03 particles/m³. Existing emission inventories overpredicted size-aligned measurements by two to four orders of magnitude, reinforcing the need to base exposure claims on measurements rather than unscaled models.

Compatible evidence places ordinary plastic-particle exposure at a small to trace level relative to the wider particulate burden. Nor et al. 2021 modeled 583 ng/day and 883 particles/day for included pathways. The Nor/EPA comparison places that mass at approximately 0.0012-0.0019% of adult soil-plus-dust ingestion alone, while same-sample studies generally find plastic to be a minority of total particles or fibres.

Like many poorly soluble dusts, plastic particles can produce inflammation or other responses when experimental systems are subjected to sufficiently high, atypical loading. This is generic high-dose particle hazard evidence, not evidence of exceptional plastic toxicity. No reliable evidence shows ordinary common-polymer dust to be more harmful than the broader low-toxicity particle mixture at compatible dose. The contrast is instructive: respirable crystalline silica or quartz, asbestos, occupational wood dust and certain toxic metal or metalloid compounds have established serious human hazards, whereas ordinary plastic dust does not. Stronger mammalian studies of selected common-polymer preparations report no adverse effects at administered doses from 25 to 5,000 mg/kg/day, orders of magnitude above modeled ordinary exposure; these values remain study-specific rather than universal thresholds.

After more than five decades and 1,661 canonical records, the central conclusion is solid rather than indeterminate: no study has established that ordinary general-population microplastic exposure causes disease in humans, and current evidence does not support classifying it as a material public-health risk. There is no scientific or logical justification for treating ordinary plastic dust as a uniquely hazardous category or for disproportionate worry about its mere presence. This conclusion remains conditional and does not cover occupational dusts, unusual hotspots, fires, damaged medical devices, intentional administration or extreme experimental doses.

Established nocebo research shows that negative expectations and threat-focused communication can increase symptoms and distress. This does not imply that any particular reported symptom is psychogenic or that genuine hazards should be minimized; it supports calm, proportionate communication when a causal hazard has not been demonstrated.

Key findings at a glance

PLAIN-LANGUAGE CONCLUSION: PLASTIC DUST IS NOT A SPECIAL HAZARD

Dust and particles are everywhere and unavoidable; plastic is only a small to trace component of this background. Like many poorly soluble dusts, plastic can produce effects at sufficiently high, atypical loading, but no reliable evidence shows ordinary plastic dust to be more harmful than the wider low-toxicity particle mixture at realistic exposure. Quartz, asbestos, occupational wood dust and certain toxic metal compounds have established serious hazards; ordinary plastic dust has not. After more than five decades and 1,661 records, there is no scientific basis for treating ordinary plastic dust as a special public-health threat.

Evidence question	Current finding	Conclusion strength	Principal boundary
Occurrence	Plastic particles are found widely because they are one component of ubiquitous environmental and household dust.	Established	Detection does not establish dominance, exceptional hazard, dose, injury or risk.
Analytical reliability	Validated spectroscopy and carefully controlled thermal methods can be informative; unconfirmed staining, optical microscopy, DLS and SEM-EDS cannot independently prove polymer identity.	Strongly supported	Performance is matrix-, polymer- and size-dependent.
Total-particle context	Compatible comparisons place plastic at a small to trace fraction of the wider particle burden; the Nor/EPA mass comparison is 0.0012-0.0019% of adult soil-plus-dust ingestion alone.	Strongly supported within stated bounds	This is a cross-domain contextual calculation using independently derived quantities, not a universal measured composition or a complete particle-exposure denominator.
Plastic specificity	High-dose effects and the principal mechanisms invoked for microplastics are not unique to plastic. No reliable evidence establishes exceptional plastic-specific hazard at ordinary exposure.	Strongly supported	Known high-hazard dusts such as quartz, asbestos, wood dust and certain metal compounds require substance-specific assessment.
Human external exposure	Ingestion and inhalation occur, but total mass and particle-number estimates remain model- and cutoff-sensitive. A 2026 global atmospheric synthesis reported a median outdoor concentration of 0.03 particles/m ³ and found that existing inventories overpredicted measured concentrations by two to four orders of magnitude.	Strongly supported	Food categories, personal breathing-zone exposure and the smallest size classes remain incompletely characterized; the atmospheric model comparison focused on 5-100 µm.
Absorption and retention	Most ingested material appears to be eliminated; the absorbed and retained fraction in humans is not reliably quantified.	Moderately supported	Direct human kinetic studies are sparse.
Human tissue evidence	An independent 2026 QA/QC audit found that none of 133 datasets from 101 human tissue or bodily-fluid studies met all 14 essential criteria for direct quantitative risk assessment. Some particle-resolved detections remain plausible as bounded occurrence evidence.	Moderately supported	The framework is deliberately strict and does not imply that every detection is false. It identifies material gaps in representativeness, blanks, recovery, particle characterization and biological plausibility.
Animal toxicology	At sufficiently high, atypical doses, plastic can produce generic particle effects, as can many other poorly soluble dusts.	Strongly supported	Many studies are high-dose proof-of-concept experiments.

Evidence question	Current finding	Conclusion strength	Principal boundary
Standardized oral toxicology	Stronger mammalian studies include formal study NOAELs of 25 mg/kg/day for weathered fragmented PP and 5,000 mg/kg/day for cellulose acetate, 1,000 mg/kg/day for PA-6, approximately 1.6-2.0 g/kg/day of each PE and PET component administered together in a 50:50 dietary mixture, and at least 2,000 mg/kg/day for fragmented PP and PTFE. Recovered historical PTFE studies provide preparation-dependent corroboration. No verified repeated-dose oral NOAEL was identified for real fragmented/weathered PS; the PVC evidence is chronic 10% control-diet context rather than a dedicated polymer-only NOAEL.	Strongly supported	The cellulose-acetate value applies to a semi-synthetic thermoplastic and a high dietary dose. Mixture component doses are not independent single-polymer NOAELs. Historical PTFE findings depend on residual dispersants. All values are polymer-, formulation-, size-, route-, sex-, duration- and endpoint-specific.
Human disease causation	No reliable causal evidence demonstrates adverse human health outcomes from ordinary general-population microplastic exposure.	Strongly supported	Human causal evidence is limited, but missing information is not positive evidence of harm; the conclusion is bounded to ordinary non-occupational exposure.
Cumulative weight of evidence	Relevant independent null, kinetic and no-adverse-effect evidence reduces the plausibility of a large hidden ordinary-exposure effect.	Strongly supported	Inference depends on quality, relevance, independence and sensitivity - not publication count. No numerical posterior is assigned.
Ecological risk	Effects can occur in selected species, laboratory systems and local hotspots; ecological management questions are separate from ordinary human oral-risk conclusions.	Moderately supported	Population relevance depends on concentration, particle form, food dilution, sediment load, species, endpoint and plastic specificity.
Risk characterization	No scientific basis exists for treating ordinary plastic dust as a unique hazard class or material public-health risk.	Strongly supported for defined ordinary exposure	The conclusion is bounded to ordinary exposure, not unusual occupational, medical, fire or hotspot scenarios.

How to read this report

1. Occurrence is expected in a particle-filled world. Detection establishes that material was present within the stated matrix, size range and analytical performance; it does not by itself establish exposure, uptake or harm.
2. All numerical results are method-bounded. Counts, masses and concentrations from different lower-size cutoffs or analytical methods should not be pooled unless compatibility is demonstrated.
3. Dose governs toxicological interpretation. Many poorly soluble particles can produce effects at sufficiently high or atypical loading; administered laboratory dose must be distinguished from ordinary environmental exposure, absorbed dose and retained burden.
4. Plastic specificity must be demonstrated. A particle-free control cannot separate a plastic-specific effect from the generic effect of adding particles, and whole-product leachates are not equivalent to intact particles.
5. The comparator must match the proposed mechanism. When a generic particle mechanism is invoked, the relevant contextual denominator includes the broader particulate mixture using an explicit route, size window and dose metric. A plastic-only count cannot establish dominance or proportional risk.
6. Cumulative weight-of-evidence updating is quality-weighted. A new study is interpreted against relevant, independent prior evidence; a weak, high-dose or method-limited result does not reset the evidence base to an uninformative state.
7. Uncertainty is not a hazard signal. It identifies what is incompletely measured; it does not supply positive evidence of harm or justify describing danger as merely undiscovered.
8. Safety is conditional. Science evaluates whether harm is demonstrated or reasonably expected under defined exposure conditions; it does not require proof that harm is impossible at every dose, route and circumstance.
9. Proportionate communication matters. When integrated evidence weighs against material risk, routine detection should not be framed as a hidden threat; negative expectations can themselves generate symptoms and distress.

1. Executive Summary

This report synthesizes a version-controlled database of 1,661 canonical evidence records spanning 1968-2026 (more than five decades): 643 independent primary-evidence records, 556 secondary scientific syntheses, 158 formal technical/context records, 71 context-only records, 232 excluded/provenance records and one unresolved source-identification record. Evidence is not counted as though every publication were equivalent. Each major conclusion is linked to analytical validity, study design, exposure realism, control quality, publication lineage, plastic specificity, the strongest contradictory evidence and explicit evidence that would change the conclusion.

The most robust cross-cutting conclusion is methodological. Microplastic measurements are highly sensitive to lower size cutoff, sample volume, blank correction, recovery, particle-identification rules and matrix preparation. Spectroscopy can identify individual particles within its detection window when spectra and quality controls are adequate. Thermal methods can quantify polymer-associated mass, but destructive analysis does not by itself establish intact-particle number, size, morphology or tissue location. Nile Red, optical microscopy, dynamic light scattering and SEM-EDS are not independently sufficient to prove polymer identity. Results obtained using incompatible constructs cannot be pooled merely because all are reported as microplastics.

Dust and particles are everywhere. Humans continuously inhale and ingest a complex mixture of mineral dust, soil, soot, pollen, fungal material, cellulose, skin and textile fibres, food particles, metal-containing particles and synthetic particles. A particle-free environment is neither realistic nor necessary for health. Plastic's detection in air, water, food, dust and selected tissues is expected for a widely used material present as a minor component of this broader background. Detection is not a special biological event and does not establish absorbed dose, injury or disease.

Atmospheric occurrence is now better constrained by a global synthesis of 2,782 concentration and deposition measurements from 76 studies. The median measured concentration was 0.03 particles/m³, with medians of 0.08 particles/m³ over land and 0.003 particles/m³ over the ocean. The arithmetic mean was 31 particles/m³ because a small number of high measurements strongly skewed the distribution. Existing top-down and bottom-up emission inventories overpredicted the measured concentrations by two to four orders of magnitude, demonstrating that typical outdoor exposure should be represented by the measured distribution rather than by unscaled model output (Evangelou et al. 2026).

Liu et al. 2026 measured strong colour-dependent light absorption in selected pigmented plastic particles and then modeled a global direct radiative forcing of 0.039 ± 0.019 W/m². The optical measurements are potentially useful, but the global estimate depended on modeled atmospheric burdens of 0.075 mg/m² for microplastics and 0.017 mg/m² for nanoplastics - together about two-thirds of the study's modeled black-carbon burden. Approximately 85% of the claimed forcing came from the least constrained nanoplastic term, and emission-rate uncertainty was not included in the reported forcing range. In light of the measurement-constrained finding that existing atmospheric inventories overpredicted observed concentrations by two to four orders of magnitude, the warming estimate is treated as a high-end model scenario rather than a validated global climate contribution.

Exposure estimates must be stratified by construct, but the compatible evidence consistently places plastic in perspective. Nor et al. 2021 provides an integrated modeled estimate of 883 particles/day and 583 ng/day for included pathways and a 1-5,000 micrometre domain. The Nor/EPA comparison to 30-50 mg/day adult soil-plus-dust ingestion yields 0.0012-0.0019% by mass, or about one to two parts per 100,000. Same-sample indoor, airborne-fibre and potable-water studies use different metrics and size windows but likewise find plastic to be a minority. The exact fraction varies by setting and method; the central point does not: ordinary plastic-particle exposure is small relative to the total dust and particle background.

Two later high-mass reports were audited because their headline values could appear to overturn the benchmark. Yadav et al. 2022 estimated 122-203 mg/day after converting visually counted objects to mass using assumed spheres, cylinders, size-bin means and density limits. Its particle count was only about 2.3 times the Nor et al. 2021 particle count, while its mass was more than 200,000 times larger; a few large objects dominate a cubic volume conversion. Song et al. 2024 reported an apparent 27.1 mg/day at the study median body weight using Py-GC/MS, but the method measured polymer-associated pyrolysis signals rather than intact particles and was vulnerable to complex-matrix interference, especially for polyethylene. These studies are retained as method-stress cases, not pooled as equal-credibility upper bounds.

Human ingestion and inhalation occur, but external intake, uptake, internal dose and long-term retention are different quantities. Recent disease-state, surgical and postmortem studies - which do not establish representative body burden, antecedent exposure or causation - report polymer-associated material or spectroscopically assigned particles in feces, plaques, coronary blood, cerebrospinal fluid, tumour and peritumoural tissue, postmortem brain, blood, bone and intervertebral discs. Most ingested material appears to be eliminated, while human uptake of the smallest particles remains incompletely quantified. Reverse causation, altered physiology, confounding, contamination and method-specific signal assignment remain conclusion-controlling limitations.

Lane et al. 2026 provides the most comprehensive independent QA/QC audit of the human biomonitoring literature identified for this update. Across 133 datasets from 101 studies, every dataset had at least one essential criterion scored zero; the mean was 5.8 zero scores and 12.8 of 28 total points. The most common gaps involved positive recovery controls, particle size and shape, clean-air conditions, sample representativeness, digestion validation and blank/LOD/LOQ reporting. The authors define this as unsuitable for direct quantitative risk assessment, not as proof that every detection is false. The result supports selective confidence: stronger particle-resolved studies can establish bounded occurrence, but current human data cannot reliably quantify population body burden or provide a particle-specific dose-response.

Tran et al. 2026 systematically reviewed 25 studies in living humans. Eleven were rated low overall risk of bias with ROBINS-E, eight had some concerns and six were high risk; the largest concerns involved confounding and exposure measurement. A low ROBINS-E rating means that a non-randomized exposure study was comparatively well conducted within that tool. It does not by itself validate microplastic identity, establish temporality, eliminate reverse causation or convert an association into a causal effect. This report therefore separates risk of bias from causal sufficiency and from confidence in the exposure analyte.

Toxicology must be interpreted by material, route, preparation and dose. Like other poorly soluble particles, plastic particles can produce biological responses when experimental systems are subjected to sufficiently high or unusual loading. Such responses establish hazard under the test conditions, not a unique plastic property or ordinary population risk. Selected stronger oral mammalian studies report no treatment-related adverse effects at study-specific doses from 25 to 5,000 mg/kg/day for defined PP, PA-6, PTFE, PE/PET and cellulose-acetate preparations. These administered doses are millions to hundreds of millions of times above the selected modeled ordinary external-mass exposure. The comparisons are diagnostic scale checks, not universal thresholds or formal margins of exposure.

Plastic-specific causation requires the correct comparator. Inhalation and ingestion, irritation or overload at high dose, inflammation, oxidative stress, sorption, biofilms, fragmentation and uptake of sufficiently small particles are not unique to plastic. No reliable evidence demonstrates that ordinary common-polymer particles are materially more hazardous than the broader low-toxicity particle mixture at compatible dose. The contrast with known hazards is clear: respirable crystalline silica or quartz has an established exposure-response relationship with lung cancer; occupational wood dust is associated with sinonasal cancer; and arsenic, cadmium, chromium, lead, mercury and related compounds have established systemic toxicity, with several species carcinogenic (Steenland et al. 2001; Demers et al. 1995; Tchounwou et al. 2012). Those specific agents deserve special treatment because their hazards are demonstrated. Ordinary plastic dust has not met that criterion.

Human-health and ecological questions are separated. The absence of demonstrated disease from ordinary human exposure does not resolve local wastewater, sediment, filter-feeder, fibre, food-dilution, entanglement or larger-debris issues. Ecological risk depends on species, concentration, particle dimensions, environmental realism, food availability and habitat. Source-specific management can therefore be justified without converting an ecological or litter-management problem into an unsupported generalized human-health claim. Total-particle context does not negate local source control, obstruction or entanglement, formulation-specific toxicity, unusual persistence, sensitive receptors or high-concentration hotspots; those must be evaluated at the relevant endpoint, receptor, material and concentration.

Official agency statements must be interpreted within the evidence base, scope and date of each agency review. None of the agency assessments summarized here evaluated the complete 1,661-record evidence corpus assembled for this report, and several relied on substantially earlier or narrower evidence sets. Consequently, statements that evidence is “insufficient” or that further research is needed should not be interpreted as a finding that the broader scientific literature is insufficient to evaluate ordinary microplastic risk. They describe the limitations of the evidence and methods considered within those particular agency assessments.

Within their respective scopes, several agencies are already broadly consistent with the central conclusion of this report. FDA states that current evidence does not demonstrate that detected microplastic levels in foods pose a human-health risk. BfR states that current scientific knowledge indicates relatively low human-health risk and that tissue detections and disease correlations do not establish causation. WHO's 2022 assessment reviewed evidence only through December 2021 and emphasized uncertainty and research needs. EFSA is preparing a new assessment for completion in 2027. EPA's inclusion of microplastics in draft CCL 6 is an information-gathering and prioritization action, not a determination that harm has been demonstrated. The UK Committee on Toxicity concluded that the available evidence within its inhalation assessment was insufficient to perform a complete quantitative risk assessment; this is a limitation of that assessment, not evidence that ordinary exposure causes harm.

These agency positions should therefore not be treated as independent evaluations of the same comprehensive evidence base analyzed here. This report evaluates a substantially broader body of evidence spanning more than five decades and integrates occurrence, exposure, analytical validity, kinetics, toxicology, comparative particle science and human evidence. On that larger evidence base, the conclusion is stronger than "we do not know": ordinary general-population microplastic exposure has not been demonstrated to cause adverse human-health effects and does not presently constitute a demonstrated material public-health risk.

The public-health conclusion should be stated directly. After more than five decades and 1,661 canonical records, no study has established a causal relationship between ordinary general-population microplastic exposure and disease in humans. Current evidence does not support classifying that exposure as a material public-health risk, and it provides no scientific or logical basis for treating ordinary plastic dust as a uniquely hazardous category. This is stronger and more informative than saying merely that risk is unknown, but narrower than claiming absolute harmlessness at every dose, route or circumstance. The conclusion would change with validated, replicated evidence of realistic exposure, materially greater internal dose and clinically adverse outcomes, or exceptional plastic-specific potency relative to matched particles.

Risk communication is part of risk management. Established nocebo research shows that negative expectations and threat-focused messaging can increase symptoms, distress and avoidable health concern. This does not imply that any particular reported symptom is psychogenic and does not justify withholding uncertainty; it requires presenting uncertainty in proportion to the evidence. Repeatedly framing ordinary plastic-particle detection as a hidden threat, when the integrated evidence weighs against material risk, can create a real burden without a demonstrated exposure benefit (Colloca & Barsky 2020).

Author, Affiliations and Independence

Dr. Chris DeArmitt is a polymer scientist and President of Phantom Plastics LLC, an independent technical consultancy specializing in polymer formulation, materials selection, product development, failure analysis and plastics science. Phantom Plastics does not manufacture, market or sell plastics or plastic products.

Dr. DeArmitt is also Founder of the Plastics Research Council, a nonprofit organization established to review and communicate scientific evidence concerning plastics, microplastics, materials and environmental claims. Plastics Research Council works without pay or compensation of any kind to share peer-reviewed science.

This report was prepared as unpaid work for the Plastics Research Council. Neither Dr. DeArmitt nor Phantom Plastics received payment, research funding, sponsorship or other financial support for preparing or publishing the report. No client, plastics manufacturer, trade association, advocacy organization, donor or government agency commissioned, directed, reviewed or controlled its evidence selection, scientific interpretation, wording, conclusions or publication.

Competing interests

The author declares no competing financial or non-financial interests in relation to this report.

Professional expertise in polymer science is disclosed as relevant author background and qualification. It should not be characterized as a competing interest merely because the subject of the report includes plastics. The review is designed to be transparent and checkable: major claims are linked to identified sources; evidence is graded

using explicit criteria; contrary and null findings are retained; and detection, association, hazard and causal-risk conclusions are separated.

AI assistance and author responsibility

AI-assisted tools, including OpenAI ChatGPT, supported structured extraction, consistency checking, source triage, calculation checking and adversarial review. They were not treated as scientific authorities or independent reviewers. The author retained responsibility for source selection, evidence appraisal, interpretation, wording and every final conclusion.

2. Scope and Methods

The version-controlled evidence-database baseline contains 1,661 unique canonical identifiers. This public-release manuscript does not alter the database: all canonical identifiers, frozen order, workbook structure and the exact 158-address formula map remain unchanged. Sources identified after the database freeze are discussed as supplementary sources outside the frozen baseline and are not included in the 1,661-record count unless separately adjudicated in a later database version.

The evidence synthesis preserves the database hierarchy. Independent primary records are distinguished from reviews, technical context, commentary, media, supporting information and duplicates. Reviews are not counted as new empirical datasets. Preprints, final publications, corrections, supplements and derivative communications are treated as one publication lineage where identified.

Evidence was interpreted using four linked questions: What was measured? How reliably was it measured? Under what exposure conditions was it observed? What inference is justified? The appraisal separates occurrence, exposure, uptake, retention, hazard, adverse outcome and population risk. It also separates intact particles from additives, leachates, sorbed chemicals and destructive analytical marker signals.

This integrated synthesis uses the structured database fields, quality grades, risk-relevance statements, limitations and source-level quantitative extraction layer. Cross-study comparisons are included only where original units, size windows, methods and assumptions remain explicit; incompatible datasets are not pooled.

Integrated evidence synthesis

The review was designed as an evidence system rather than a conventional narrative search. Canonical Study IDs preserve publication lineage and prevent a review, preprint, final article, supplement, correction, dataset or media derivative from being counted as independent confirmation. Records excluded from the scientific-source count remain visible so that selection decisions can be audited. This is important in a fast-growing field where a single underlying experiment may generate several publications and public claims.

Primary evidence was appraised on separate dimensions. Method validity addresses whether the analytical approach can identify the claimed material in the stated matrix and size range. Exposure realism addresses whether the concentration, route, duration and particle preparation resemble plausible environmental contact. Control quality addresses blanks, recovery, comparator design and the ability to distinguish particles from chemicals or handling artefacts. Plastic specificity addresses whether the observed result is attributable to plastic rather than to particles generally, additives, leachates or sorbed chemicals. These dimensions are related but not interchangeable; a study may be analytically strong yet toxicologically unrealistic, or exposure-relevant yet unable to assign polymer identity confidently.

The quantitative synthesis retained original units and created conversions only when the formula and assumptions could be stated explicitly. Particle number, mass, surface area and volume were not treated as mutually convertible without measured size distributions, density, shape and recovery. Data were grouped by compatibility before comparison. Where compatibility was incomplete, the report uses scale comparisons or diagnostic ratios with a clear warning rather than pooled estimates or formal margins of exposure.

The stopping rule is conclusion-based. Additional occurrence papers are logged for later updates unless they materially change exposure, analytical validity, a toxicity threshold, a causal conclusion, or the interpretation of a major study through correction or retraction. This prevents an effectively unlimited literature search from displacing synthesis while retaining a mechanism for conclusion-changing evidence.

Targeted currency update through 29 July 2026

A targeted current-evidence review assessed conclusion-relevant human evidence, exposure syntheses, analytical-method studies, atmospheric measurements and models, and toxicology sources available through 29 July 2026. Searches used title, DOI and topic combinations in PubMed, publisher platforms and official agency sites. Newly added or materially reappraised sources include Tran et al. 2026, Xu et al. 2025, Nihart et al. 2025 and its correction, the Monikh et al. 2025 critique and Campen et al. 2025 reply, He et al. 2025, Li et al. 2026 brain-tumour and matched-tissue studies, Chen et al. 2026, Fang et al. 2026, Lu et al. 2026, Paolisso et al. 2026, Heo et al. 2024, Aves et al. 2026, Jüngling et al. 2026, Jamison Hart and Lenhart 2026, Clough et al. 2026, Evangelou et

al. 2026, Liu et al. 2026, Lane et al. 2026 and the German BfR assessment of the Nihart study. Sources identified after the database freeze are labelled as supplementary sources outside the frozen baseline until separately adjudicated into a later database version.

Targeted thermoplastics oral-toxicology and source-recovery supplement through 27 July 2026

A focused follow-up reviewed newly supplied and newly located evidence on polyethylene (PE), polypropylene (PP), polyvinyl chloride (PVC), polystyrene (PS), polyethylene terephthalate (PET), polytetrafluoroethylene (PTFE) and cellulose acetate. The 26 July layer added the secondary-cited 90-day PE dietary record, the Feron et al. 1981 lifespan 10% VCM-free PVC control diet, newer PET studies and the PP/PS source-level correction. The 27 July source-recovery layer added the Thomas et al. 1991 direct cellulose-acetate study, Haskell Laboratory PTFE Reports 56-61 and 224-68, and Otaka et al. 1978. Thomas et al. supports a formal subchronic cellulose-acetate NOAEL of 5,000 mg/kg/day. The Haskell reports show that high-diet PTFE findings were preparation-dependent and that residual dispersants, rather than PTFE alone, produced selected liver and body-weight effects. Otaka et al. is reclassified as PET chloroform-extract toxicology rather than direct PET-particle or PET-polymer toxicology. The recovered evidence remains supplementary outside the frozen 1,661-record database baseline.

Focused human-biomonitoring QA/QC supplement through 29 July 2026

A focused update incorporated Lane et al. 2026 and its supplementary scoring files. The study applied a 14-criterion 0-1-2 framework to 133 datasets from 101 peer-reviewed studies of human organs, tissues and bodily fluids. The three domains were study and sample description, contamination mitigation and particle characterization. A zero on any essential criterion rendered a dataset unsuitable for direct quantitative risk assessment within that framework; the authors explicitly distinguished this from scientific worth or publication quality. Exact study-level scores were used as an external cross-check and did not overwrite the version-controlled database grades.

Focused retained-lung inorganic-particle context supplement through 13 August 2026

A focused update incorporated the supplied peer-reviewed Hayden et al. 2023 full text as a supplementary primary context source. Using automated in-situ FE-SEM/EDS, the authors characterized 124,018 inorganic particles in 38 surgical lung-biopsy specimens and assigned them to 45 particle or mineral classes and 12 broader groups. More than 90% of analyzed particles had a greatest dimension below 2.5 micrometres. The source is used here to strengthen route-matched nonplastic lung-particle context and to clarify what FE-SEM/EDS can and cannot establish. It is not added to the frozen 1,661-record database, is not used to estimate a population lung burden or a plastic fraction, and does not change the report's human-health conclusion.

Focused inhaled nanoparticle translocation supplement through 13 August 2026

A focused update reviewed the supplied Miller et al. 2017 ACS Nano article, its supporting information and the published correction as one publication lineage. Healthy volunteers deliberately inhaled well-characterized gold nanoparticles, allowing high-sensitivity HR-ICPMS and Raman tracking of systemic appearance and tissue localization. The correction changed the authors' illustrative 24-hour translocation estimate from approximately 0.2% to approximately 0.02% after correcting concentration units and the inhaled-dose calculation. The evidence is used here to establish biological plausibility and strong size dependence for translocation of ultrasmall inhaled nanoparticles, not as a microplastic absorption fraction and not as evidence of cardiovascular injury.

Focused nonplastic tire-rubber leachate boundary supplement through 13 August 2026

A focused boundary update reviewed the peer-reviewed primary studies of Tian et al. 2021 and Tian et al. 2022. Tire rubber is an elastomeric material, not a plastic, so tire-wear particles and tire-derived chemicals are not classified as microplastics in this report and these sources are not added to the frozen evidence corpus. They are retained solely to document a proven source-specific leachate hazard and to prevent a category error in which toxicity of 6PPD-quinone, a tire-rubber additive transformation product, is attributed to plastic particles.

Focused comparative dust-hazard and risk-communication context supplement through 18 August 2026

A focused context update added five peer-reviewed sources concerning generic high-dose overload responses of poorly soluble low-toxicity particles, established human hazards from respirable crystalline silica, occupational wood dust and toxic metals or metalloids, and clinically documented nocebo effects. These sources are supplementary context outside the frozen 1,661-record baseline. They do not add plastic-specific toxicity records or alter database counts. Their purpose is to sharpen the public-health interpretation: plastic is a small component of ubiquitous dust; high-dose particle effects are not unique to plastic; some nonplastic dust components have established human hazards; and communication should be proportionate to demonstrated risk.

Methods transparency and audit status

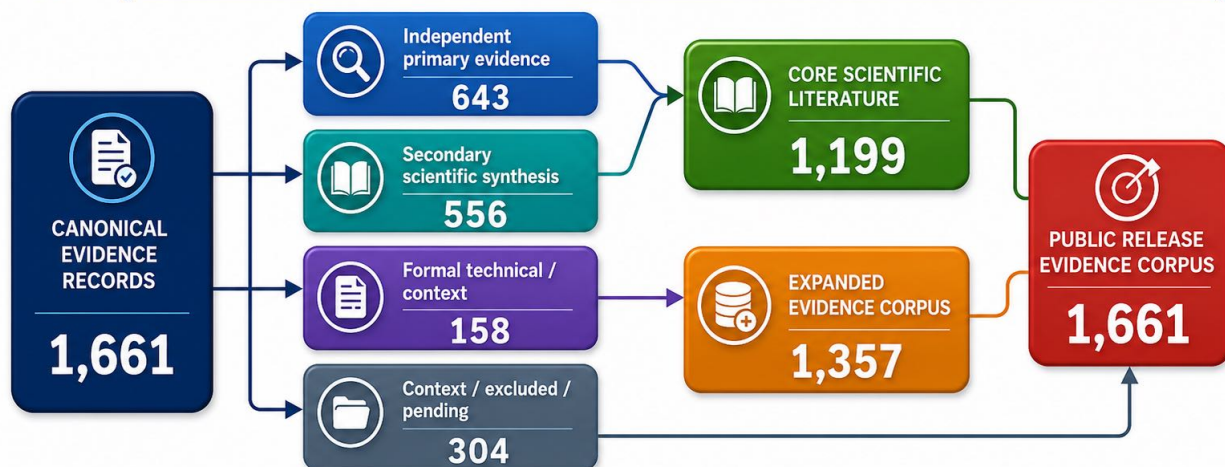
This public-release edition publishes the search and screening information that is actually recoverable. The earliest corpus-building searches were not retained as one prospectively registered verbatim query log with raw-result and deduplication counts; the report therefore does not invent a PRISMA identification flow. Appendix I records exact canonical-corpus accounting, documented acquisition and adjudication episodes, official-agency refresh queries, prospective Boolean concept blocks, publication-lineage rules, inclusion and exclusion criteria, and the version-controlled process for sources identified after the database freeze.

Metric	Count	Interpretation
Canonical records	1,661	Version-controlled record count and frozen order unchanged
Independent primary evidence	643	Internally reconciled after the STUDY-01235 correction; canonical order unchanged
Secondary scientific synthesis	556	Reviews and syntheses; not new primary datasets
Formal technical/context	158	Internally reconciled after moving STUDY-01235 to independent primary engineering evidence
Primary quality	11 High; 297 Moderate; 295 Low; 40 Very low	Evidence must be weighted, not counted equally
Human observational/clinical quality	2 Moderate; 50 Low; 11 Very low	No High-quality human causal record



Evidence-source flow: version-controlled 2026 synthesis profile

Records are not equal-weight studies; reviews and supporting files are not double-counted.



Core = primary + secondary. Expanded = core + formal technical/context.
The corpus retains all provenance categories.



Version-controlled

643
primary

+

556
secondary

+

158
formal/context

+

304 context/excluded/
pending

=

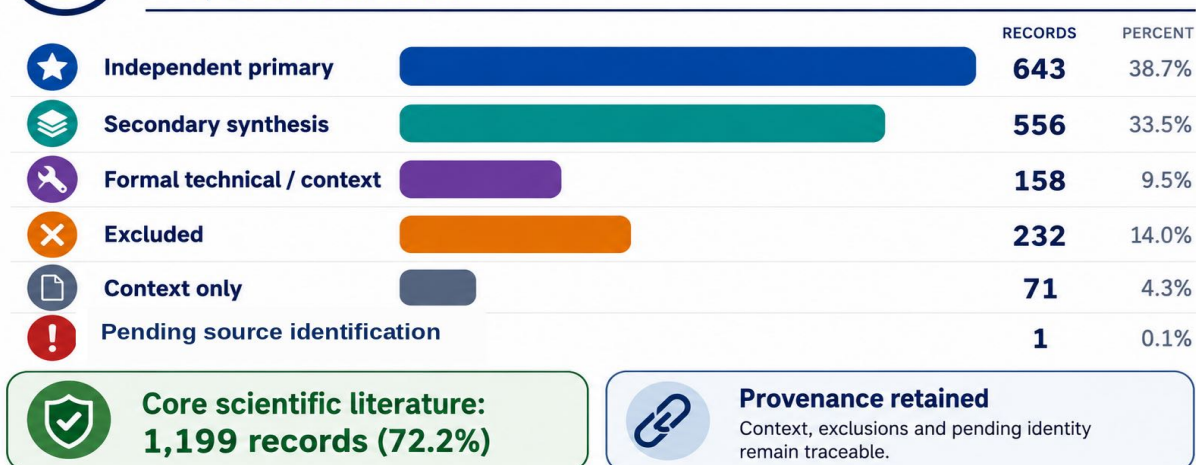
1,661

Figure 1. Evidence-source flow for the version-controlled 2026 synthesis profile.



Evidence roles in the controlled synthesis corpus

n = 1,661 canonical records



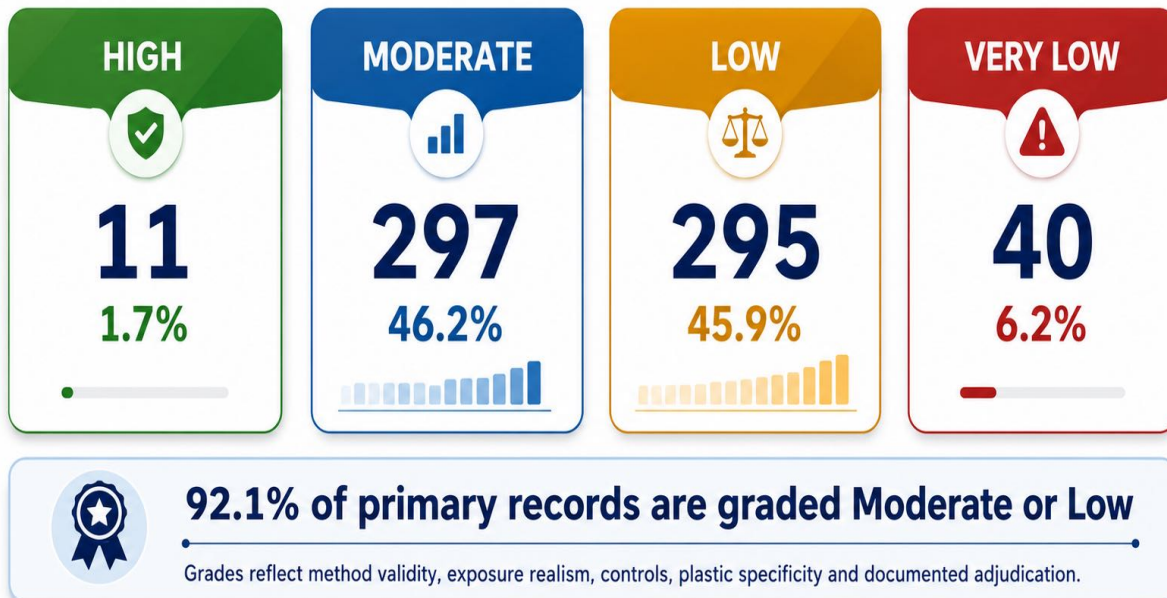
One low-priority source-identification record remains in quarantine (STUDY-01095).

Figure 2. Evidence-role distribution after internal database reconciliation. The remaining unresolved source-identification record is STUDY-01095; the Cardona Orozco thesis is classified as independent primary engineering evidence.



Primary-evidence quality distribution

643 independent primary-evidence records



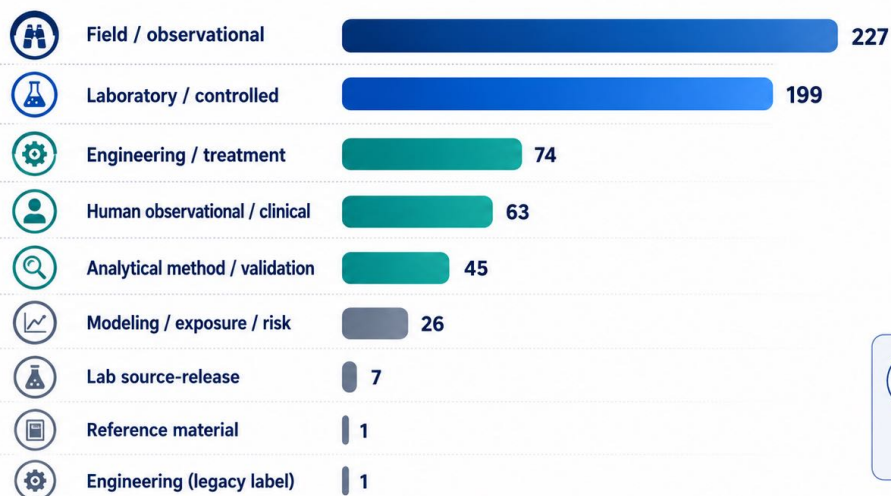
A grade defines evidentiary reliability and permitted inference; it does not encode whether a result was positive, null or adverse.

Figure 3. Primary-evidence quality distribution.



Primary study-design distribution

Different designs answer different scientific questions.



426
records

66.3%

field/observational +
laboratory/controlled



Design mix matters

Counts do not imply equal
causal reach, method validity
or risk relevance.



Design categories are mutually exclusive controlled labels in the certified evidence database.

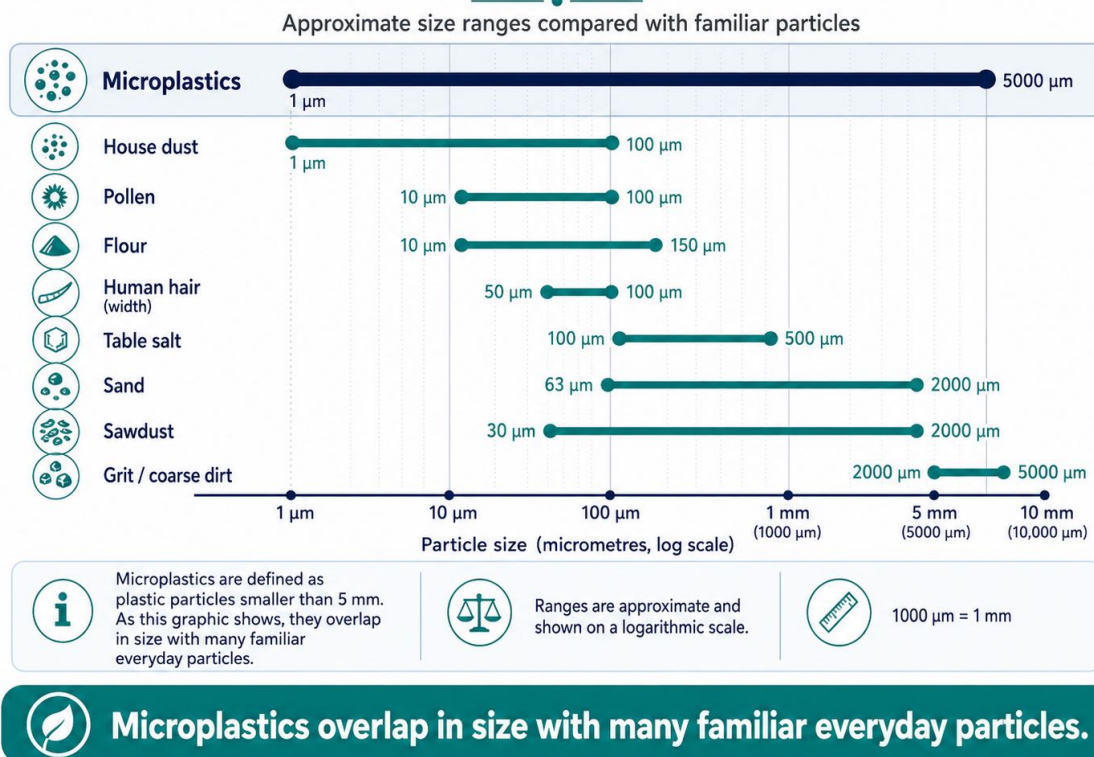
Figure 4. Primary study-design distribution.

3. Definitions and Analytical Boundaries

Microplastic is used here as a size-based descriptor for plastic particles, generally below 5 mm, while nanoplastic denotes smaller particles for which operational definitions and measurement capabilities remain inconsistent. The report does not assume that a thermal degradation product, polymer-associated mass signal, stain-positive object, carbon-rich morphology or hydrodynamic-size peak is necessarily an intact plastic particle.

Scope boundary: tire rubber is an elastomer, not a plastic; tire-wear particles and tire-derived rubber chemicals are excluded from the microplastic category used in this report.

How Big Are Microplastics?



External exposure means material present in air, food, water or another contact medium. Administered dose is the amount supplied in an experiment. Internal dose is the amount absorbed into the body. Retained dose is the amount remaining after clearance. A detected signal is not automatically an internal or retained particle burden.

Hazard is the capacity to cause an effect under specified conditions. Risk combines hazard with the magnitude, route, frequency and duration of exposure, plus susceptibility and uncertainty. A statistically significant biomarker change is not automatically an adverse outcome, and an adverse outcome in a high-dose model is not automatically a population risk.

What 'safe' means in science and regulation

Science cannot establish absolute zero risk for any substance because no experiment can test every dose, duration, route, formulation, population and endpoint. That limitation applies to substances routinely and correctly described as safe. Water can cause life-threatening hyponatremia when consumed in extreme excess, and oxygen can cause toxicity at supraphysiologic exposure. Their hazards at extreme conditions do not make ordinary exposure unsafe.

Regulatory safety is therefore conditional and evidence-based. The U.S. FDA uses a standard of reasonable certainty of no harm under intended conditions and explicitly states that science can never be absolutely certain of the absence of any risk from any substance. This report applies the same logic: it evaluates ordinary exposure, not every theoretically possible condition.

A positive claim that ordinary microplastic exposure causes disease requires positive evidence: valid exposure measurement, temporal sequence, dose-response, a clinically adverse outcome, control of confounding and independent replication. The logical possibility of unmeasured harm does not provide that evidence. Uncertainty alone cannot carry the burden of a causal claim.

Null evidence has weight when it is relevant and sufficiently sensitive. One underpowered negative study proves little. Repeated independent studies that could detect a material effect at realistic exposure, but do not, progressively constrain the plausible size and generality of any hidden effect. This is evidence against a material effect, even though it is not proof that harm is impossible under every condition.

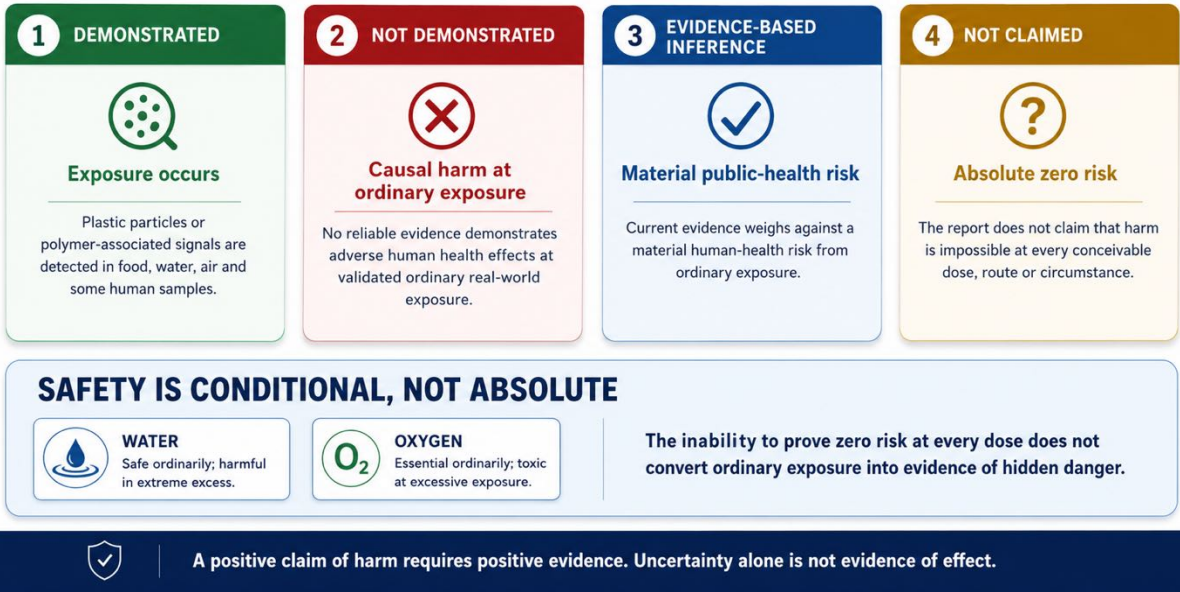
Table 1. Operational definitions for the central human-health conclusion

Term	Operational meaning in this report	What it excludes
Ordinary general-population exposure	Routine exposure through food, drinking water, ambient/indoor air and settled dust outside occupational, medical, experimental or unusual source-hotspot settings.	Occupational dusts, intentional administration, damaged-device exposure, fires, unusual spills and highly contaminated local sources.
Reliable causal evidence	Validated exposure or internal-dose measurement, temporality, an adverse endpoint, adequate confounder control, coherent dose-response and independent replication.	Detection alone, cross-sectional disease-state sampling, unvalidated markers and isolated transient biomarkers.
Adverse health outcome	A clinically, functionally or pathologically meaningful impairment, not merely any statistically significant biological change.	Adaptive responses, uncertain-significance molecular shifts and analytical signals without demonstrated injury.
Material public-health risk	A causal effect at ordinary exposure large and probable enough to meaningfully alter population disease burden or justify population-wide health intervention.	A theoretical possibility, a very small undetectable effect, or a hazard shown only at remote experimental dose.
Conditional safety conclusion	The current evidence weighs against material risk under the defined exposure conditions and remains revisable if high-information contrary evidence appears.	A claim of absolute harmlessness for every polymer, formulation, route, population or dose.

These definitions prevent two opposite errors: treating every unmeasured possibility as a present danger, and extending a bounded ordinary-exposure conclusion to occupational, source-specific or extreme-dose circumstances.

WHAT THE EVIDENCE ALLOWS US TO SAY

Ordinary human exposure to microplastics



Public communication framework. Particles are ubiquitous, and plastic is one small component of the unavoidable dust mixture. Like many poorly soluble dusts, plastic particles can cause effects at sufficiently high, atypical doses; this does not establish unique hazard at ordinary exposure. The evidence supports a direct conditional conclusion: no reliable evidence demonstrates adverse health effects from ordinary exposure, and current evidence provides no basis for treating plastic dust as a special public-health threat. Absolute harmlessness at every conceivable dose is neither claimed nor required.

Comparative particle context and cumulative-evidence terminology

Comparative particle context means placing plastic within the broader particulate mixture appropriate to the route and proposed mechanism rather than comparing it with an imaginary particle-free world. The denominator may be mass, count, surface area or deposited dose, but the route, metric and size window must be explicit. Using primary exposure estimates, total modeled adult microplastic intake is approximately 0.0012-0.0019% of adult soil-plus-dust ingestion alone by mass (Nor et al. 2021; U.S. EPA 2017). This is a bounded cross-domain context, not a universal matrix composition. Same-sample indoor particle ratios and fibre-composition studies independently show that exact fractions are setting- and method-dependent while plastic remains a minority component (Dris et al. 2017; Dris et al. 2016).

Plastic-specificity means that an effect is demonstrably attributable to the plastic material, morphology or formulation rather than to adding any poorly soluble particle. High-dose inflammation and particle overload are established generic phenomena for poorly soluble low-toxicity particles (Borm & Driscoll 2019). No commonly invoked hazard-relevant physical behaviour - including small size, charge, sorption, biofilm formation, inhalation, ingestion, translocation, persistence or overload inflammation - is unique to plastic. A special regulatory or public-health category is justified only by demonstrated exceptional exposure or potency. That evidence exists for specific dust constituents such as respirable quartz, occupational wood dust and certain toxic metal compounds; it has not been demonstrated for ordinary common-polymer particles at ordinary exposure.

Cumulative weight-of-evidence updating is used qualitatively in this report to describe how new evidence changes an integrated conclusion. Existing evidence from polymer toxicology, occupational particle science, measured exposure and previous null studies provides the context for each new result. A weak or unrealistic study changes the overall conclusion little; a replicated realistic-dose causal study would change it substantially. The report does not assign a numerical probability because no formal quantitative evidence-to-probability model has been specified.

Integrated evidence synthesis

The term particle is reserved for an object for which physical particulate form is demonstrated. A polymer-associated pyrolysis product or other destructive chemical marker can support the presence of polymer-derived mass, but it does not by itself establish the number, morphology, size or location of intact particles before analysis. Similarly, a hydrophobic fluorescent object, a carbon-rich SEM-EDS feature or a DLS scattering peak is not automatically a plastic particle. These distinctions are especially consequential in tissue, blood and lipid-rich matrices.

The exposure chain is divided into external concentration, contact, intake, deposited or administered dose, absorbed dose, internal distribution, retained burden and biological response. Each transition requires evidence. A concentration in bottled water does not equal ingestion unless consumption is known; ingestion does not equal absorption; a tissue-associated signal does not establish long-term retention; and retention does not establish injury. The same chain applies to inhalation, with additional dependence on aerodynamic diameter, fibre geometry, breathing pattern, airway deposition and mucociliary clearance.

Material identity is also separated from formulation and associated chemistry. A polymer may be biologically inert under one condition while a formulation, residual monomer, additive, degradation product or sorbed contaminant produces an effect under another. Conversely, toxicity from a whole-product leachate cannot be assigned to intact particles without fractionation or causal testing. This report therefore avoids using plastic, microplastic, additive and leachate as interchangeable exposure categories.

4. Evidence-Quality and Appraisal Framework

Primary evidence was graded using method validity, exposure realism, control quality, plastic specificity and expert override. High-quality classification was rare: 11 of 643 primary records. Moderate and Low records dominated. This distribution requires conclusion weighting rather than vote counting.

Analytical validity considers polymer specificity, matrix validation, procedural blanks, recovery, spectral criteria, lower size limit and orthogonal confirmation. Exposure realism distinguishes measured occurrence and exposure-relevant conditions from scenario models and high-dose proof-of-concept experiments. Control quality gives particular weight to matched natural or alternative-particle comparators and to appropriate contamination controls.

Conclusion strength is assigned only after identifying the strongest supporting evidence, strongest contrary evidence, highest-quality contradiction, necessary analytical or causal assumptions, residual uncertainty and evidence capable of changing the conclusion.

For human tissue and bodily-fluid measurements, Lane et al. 2026 is used as a supplementary fit-for-risk-assessment cross-check. Its framework evaluates sample information, representativeness, donor metadata, collection and digestion; laboratory preparation, clean-air conditions, blanks and LOD/LOQ, blank correction and positive recovery controls; and particle size, count, polymer type and shape. The framework's any-zero rule is stricter and narrower than this report's overall quality grade: it asks whether a dataset can be used quantitatively in risk assessment, not whether the study is scientifically valuable or whether every detection is false.

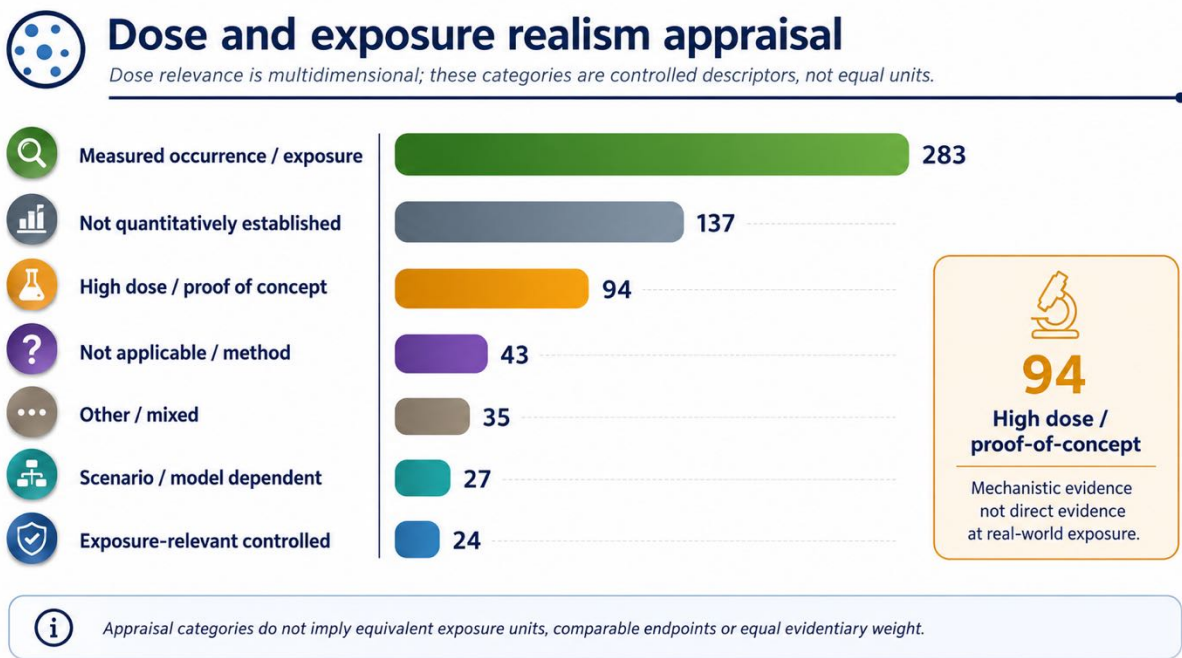


Figure 5. Primary-record dose and exposure realism appraisal.

Cumulative weight-of-evidence updating

Evidence appraisal is cumulative rather than headline-based. The version-controlled corpus contains more than 1,000 scientific evidence sources and spans 1973-2026, more than five decades of polymer and particle research. When relevant realistic-dose studies do not detect adverse health outcomes and stronger studies report no adverse effects at much higher administered doses, the range of plausible large ordinary-exposure effects becomes progressively narrower.

This cumulative-evidence logic does not convert absence of proof into absolute proof of absence. It does, however, distinguish an untested possibility from evidence. A positive claim that ordinary exposure causes disease requires positive evidence. Repeated relevant null findings, realistic-dose no-adverse-effect evidence and low measured exposure constrain the magnitude and plausibility of a hidden effect. A conclusion-changing study would need valid particle identity, realistic exposure, measured internal dose, appropriate nonplastic comparators, an adverse outcome rather than a transient marker, causal design and independent replication.

Table 2. When a null or no-adverse-effect result is informative

Criterion	Why it matters	Present interpretation
Exposure relevance or bounding dose	A study must overlap credible exposure or use a higher dose capable of bounding potency.	Realistic-dose studies test direct relevance; well-designed high-dose studies constrain claims of exceptional potency.
Validated identity and dose	A null result is uninformative if the test material, dispersion or delivered dose is uncertain.	Greater weight is assigned where material, size, preparation and administration are documented.
Adverse endpoint sensitivity	A study must measure outcomes capable of detecting meaningful injury.	Apical pathology, function, clinical chemistry and growth carry more adversity information than isolated biomarkers.
Adequate design and statistical sensitivity	Small groups, short duration or noisy endpoints may miss effects.	No single negative study is treated as decisive; convergence across sufficiently sensitive designs matters.
Independence and replication	Repeated analysis of one dataset is not independent confirmation.	Publication lineage is controlled and independent evidence streams are weighted more heavily.
Cross-stream coherence	Exposure, kinetics, toxicology and human evidence should be mutually compatible.	The present convergence supports evidence against a large material ordinary-exposure effect, not proof against every subtle effect.

Integrated evidence synthesis

Quality grades summarize evidentiary reliability, not whether a result is positive or negative. A well-controlled null study can be High or Moderate quality, while a positive study with contamination, unverified particle identity or severe dose mismatch can be Low or Very low quality. This prevents directional selection in which adverse findings receive greater weight merely because they are adverse. The same appraisal criteria are applied to null, beneficial and harmful outcomes.

No single method automatically confers high confidence. Spectroscopy requires acceptable spectra, appropriate libraries, explicit match criteria, contamination controls and a defined lower size limit. Thermal methods require validated marker specificity, matrix cleanup, recovery and blank correction. Toxicology requires dose characterization, stable dispersions, controls for vehicles and leachates, and evidence that labelled particles did not release or exchange the label. Epidemiology additionally requires valid exposure measurement, temporality and control of confounding.

Major conclusions were stress-tested against the strongest opposing evidence and the highest-quality contradictory study. Grades describe the conclusion, not the average quality of every included source. A conclusion may be Strongly supported because several independent evidence streams converge even when many individual papers are weak. Conversely, a large number of low-quality papers cannot establish a causal conclusion that lacks a valid exposure measure or temporality.

5. Environmental Occurrence

Environmental occurrence is strongly supported across marine, freshwater, atmospheric, soil and sediment matrices. Field studies show that distribution is spatially heterogeneous and shaped by buoyancy, aggregation, deposition, biological transport, hydrodynamics and sampling window. Surface observations alone do not represent total environmental inventory. Deep-sea and water-column studies support transport away from the surface, while some surveys report unexpectedly low floating abundance relative to source expectations.

The evidentiary boundary is critical: detection establishes presence within the analytical window, not total mass, temporal increase, bioavailability or harm. Studies using different mesh sizes, filters, density-separation steps and spectroscopy thresholds can report very different particle counts from the same underlying material. Key records include field evidence on North Pacific surface scarcity, vertical transport, deep-sea occurrence, atmospheric deposition and multiyear ocean surveys. (Egger et al. 2020); (Zhao 2023); (Woodall et al. 2014); (Brahney et al. 2020); (Law et al. 2014)

Integrated evidence synthesis

Environmental occurrence is best interpreted as a matrix-specific measurement problem. Air, surface water, sediment, soil, wastewater and biota differ in sampling efficiency, particle transport, organic content and contamination risk. A method validated for clean water may fail in tissue or soil, and a result reported as particles per litre cannot be compared directly with particles per kilogram, deposition flux or polymer-marker mass. The presence of plastic in one compartment also does not establish the total environmental inventory because particles move among compartments and through analytical size windows.

Spatial heterogeneity is expected. Local concentrations can be elevated near wastewater discharges, industrial handling, road runoff, textile use, agricultural films, heavily used shorelines or poorly managed waste. Remote detections demonstrate transport, not necessarily high exposure or ecological injury. Deposition and redistribution can create apparent sinks, while resuspension, fragmentation and biofouling can change where particles are sampled. The strongest occurrence studies therefore define the sampled fraction and avoid extrapolating a local count to a global mass without a defensible transport model (Egger et al. 2020; Zhao 2023; Woodall et al. 2014).

Source apportionment is often weaker than detection. Polymer identity may narrow possible sources, but common polymers have many uses and environmental weathering can obscure morphology. Fibres are particularly difficult because natural, regenerated-cellulose and synthetic fibres coexist, and airborne contamination can enter at every stage. Claims that a matrix is dominated by plastic require a denominator that includes the broader particulate burden, not only the subset selected by staining or visual pre-screening.

The occurrence evidence therefore supports monitoring and source-specific control where releases are measurable, but it does not by itself establish population risk. Risk interpretation requires concentration, bioavailability, exposure duration, organism sensitivity and an adverse-effect threshold in compatible units.

Evidence-weighted interpretation

The principal occurrence records are mutually informative rather than mutually exclusive. Surface-net surveys demonstrate widespread but spatially variable floating material, water-column work demonstrates redistribution below the surface, and deep-sea observations demonstrate transfer to benthic compartments (Egger et al. 2020; Zhao 2023; Woodall et al. 2014). A low surface count can therefore coexist with a larger vertically distributed inventory. It cannot, by itself, establish disappearance, degradation or the absence of particles elsewhere in the system.

Atmospheric deposition adds another transport pathway between urban, remote, terrestrial and aquatic compartments. The protected-area study confirms long-range transport within its sampled size and identification window, but its regional mass estimate is an extrapolation from a limited monitoring network rather than a direct inventory (Brahney et al. 2020). The same distinction applies to ocean-wide and watershed-wide estimates: measured local concentration, modeled regional flux and global stock are different quantities.

A 2026 global synthesis compiled 2,782 atmospheric measurements from 76 studies and 283 locations. Median concentration was 0.03 particles/m³ (interquartile range 0.002-0.1), with 0.08 over land and 0.003 over the ocean. Existing inventories overpredicted size-aligned measurements by two to four orders of magnitude. The 5-100 µm

comparison remains limited by heterogeneous methods, sparse ocean data, incomplete sources and assumed size distributions (Evangelou et al. 2026).

For cross-study interpretation, every occurrence result should retain five descriptors: sampled compartment, lower and upper size limits, sampling volume or mass, polymer-identification rule, and blank/recovery treatment. Without those descriptors, apparent geographic or temporal differences may reflect analytical coverage rather than environmental concentration. Occurrence is established; a method-independent global abundance distribution is not.

Table 3. Cross-matrix occurrence evidence and inference boundaries

Evidence compartment	Database observation	Inference supported	Principal boundary
Surface ocean	Widespread but highly variable floating abundance (Egger et al. 2020; Law et al. 2014)	Surface occurrence within trawl window	Not total water-column inventory; mesh and weather dependent
Water column	Depth-related particles and composition (Zhao 2023)	Vertical redistribution is plausible	Transects and size window limit basin extrapolation
Deep sea	Fibres reported in sediments and corals (Woodall et al. 2014)	Transport to benthic compartments	Small heterogeneous sample; fibre-contamination risk
Atmosphere	Global median 0.03 particles/m ³ ; land 0.08, ocean 0.003; inventories two to four orders high (Brahney et al. 2020; Evangelou et al. 2026)	Low, right-skewed outdoor occurrence; land dominates particle-number emissions	5-100 µm model comparison; heterogeneous methods, sparse ocean data and incomplete sources
Temporal monitoring	Local increases and null trends both reported (Brandon et al. 2019; Beer et al. 2018)	Trends can be matrix-specific	Method and archive comparability govern trend validity
Interlaboratory comparison	Reproducibility and method-dependent variation for distributed samples	Limited to participating laboratories, matrices and test materials	Blind common samples, harmonized reporting and proficiency criteria
O-PTIR / photothermal spectroscopy	Particle-resolved spectral assignment above validated limit	Library quality, matrix residue and size performance remain method-specific	Blanks, reference spectra, recovery and orthogonal confirmation where needed

Selected evidence records

- **(Moderate)**. Egger, M., Nijhof, R., Quiros, L., et al. (2020). A spatially variable scarcity of floating microplastics in the eastern North Pacific Ocean. *Environmental Research Letters*, 15, 114056. <https://doi.org/10.1088/1748-9326/abbb4f>
- **(Moderate)**. Shiye Zhao. 2023. Pelagic microplastics in the North Pacific Subtropical Gyre: A prevalent anthropogenic component of the particulate organic carbon pool.
- **(Moderate)**. Woodall, L. C., Sanchez-Vidal, A., Canals, M., Paterson, G. L. J., Coppock, R., Sleight, V., Calafat, A., Rogers, A. D., Narayanaswamy, B. E., & Thompson, R. C. (2014). The deep sea is a major sink for microplastic debris. *Royal Society Open Science*, 1, 140317. <https://doi.org/10.1098/rsos.140317>
- **(Moderate)**. Brahney, J., Hallerud, M., Heim, E., Hahnenberger, M., & Sukumaran, S. (2020). Plastic rain in protected areas of the United States. *Science*, 368, 1257–1260. <https://doi.org/10.1126/science.aaz5819>
- **(Moderate)**. Law, K. L., et al. (2014). Distribution of Surface Plastic Debris in the Eastern Pacific Ocean from an 11-Year Data Set. *Environmental Science & Technology*. <https://doi.org/10.1021/es4053076>

6. Temporal Trends

Temporal-trend evidence is mixed. Some repeated surveys, archived samples and sediment records report increases over time, whereas other field records find no increase or show that redistribution, sampling location and method change can dominate apparent trends. Trend inference requires a stable sampling frame, comparable analytical method, consistent lower size limit and correction for sediment mixing or particle migration.

A universal statement that environmental microplastic concentrations are increasing everywhere is not justified by the version-controlled database. Local and matrix-specific increases may be credible, but they should not be generalized to the global environment without harmonized monitoring. Representative records include an 11-year eastern Pacific dataset, a multidecadal coastal record, a no-increase fish study, an urban-lake sediment record and historical beach monitoring. (Law et al. 2014); (Brandon et al. 2019); (Beer et al. 2018); (Turner et al. 2019); (Ryan et al. 1990)

Integrated evidence synthesis

A credible temporal trend requires measurements that are comparable across time. Changes in filter size, digestion, polymer libraries, contamination control, sample preservation or the inclusion of smaller particles can create an apparent increase even when the underlying environmental concentration is unchanged. Historical samples are valuable only when storage contamination, degradation and retrospective analytical recovery are addressed.

Some sentinel and archive studies report increases in specific debris categories or biological burdens, while others find stable or non-increasing concentrations within their sampled matrix and size range (Beer et al. 2018; Ryan et al. 1990). These findings are not necessarily contradictory because they concern different compartments, particle sizes, geographies and source histories. Macro-debris on beaches, floating trawl particles, seabird stomach contents and small particles in archived water answer different questions.

Global production growth is not a direct measurement of environmental concentration. Emissions, collection, treatment, degradation, transport and compartment transfer intervene between production and a sampled concentration. Models may project increasing inputs, but a projection should remain labelled as a scenario until tested against stable monitoring data. The current evidence supports local and matrix-specific trends; it does not support a universal statement that all environmental microplastic concentrations are rising everywhere.

Selected evidence records

- **(Moderate)**. Law, K. L., et al. (2014). Distribution of Surface Plastic Debris in the Eastern Pacific Ocean from an 11-Year Data Set. *Environmental Science & Technology*. <https://doi.org/10.1021/es4053076>
- **(Moderate)**. Brandon, J. A., Jones, W., & Ohman, M. D. (2019). Multidecadal increase in plastic particles in coastal ocean sediments. *Science Advances*, 5(9), eaax0587. <https://doi.org/10.1126/sciadv.aax0587>
- **(Moderate)**. Beer, S., Garm, A., Huwer, B., Dierking, J., & Nielsen, T. G. (2018). No increase in marine microplastic concentration over the last three decades – A case study from the Baltic Sea. *Science of the Total Environment*, 621, 1272–1279. <https://doi.org/10.1016/j.scitotenv.2017.10.101>
- **(Moderate)**. Simon Turner, A. A. Horton, Neil L. Rose & Claire Hall. 2019. A temporal sediment record of microplastics in an urban lake, London, UK. *Journal of Paleolimnology* 61:449-462.
- **(Low)**. Ryan, P. G., & Moloney, C. L. (1990). Plastic and other artefacts on South African beaches: Temporal trends in abundance and composition. *South African Journal of Science*, 86, 450–452.

7. Airborne Exposure

Airborne particles and fibres are a normal, unavoidable component of indoor and outdoor air. Mineral dust, soot, pollen, fungal fragments, cellulose, skin and textile fibres, road and brake wear, metals and plastics occur together. Atmospheric-deposition studies support transport over regional and long distances, but measured abundance depends on sampler location, exposure time, blank correction and particle-identification criteria. Many airborne fibres are natural or semi-synthetic rather than plastic, and the relative composition of the total inhalable particle background is frequently underreported.

Airborne occurrence is relevant to external exposure, but inhaled dose requires breathing-rate assumptions, size-resolved deposition and clearance. Atmospheric counts should not be equated with retained lung burden. Key evidence includes protected-area deposition, urban fallout, indoor-air measurements and textile fibre release. (Brahney et al. 2020); (Wright et al. 2020); (Dris et al. 2016); (Dris et al. 2017); (Liu et al. 2026)

The largest global atmospheric compilation identified in this review includes 925 concentration measurements and 1,857 deposition measurements collected in 76 studies. Its measured concentration median was 0.03 particles/m³, with the land median 27 times the ocean median. Existing inventory-based simulations overpredicted the measurements by two to four orders of magnitude. These values are occurrence and external-concentration evidence, not inhaled or retained dose, and the transport-model comparison focused on 5-100 µm particles (Evangelou et al. 2026).

Integrated evidence synthesis

Airborne exposure comprises indoor suspension, outdoor transport, deposition and resuspension. Indoor sources can include textiles, furnishings, floor dust, occupant activity and outdoor infiltration. Outdoor fibres and fragments may arise from road traffic, construction, waste handling, textiles and atmospheric transport. Source assignment from morphology alone is unreliable because many natural and regenerated fibres resemble synthetic material under optical microscopy.

Reported airborne counts depend strongly on sampler design, sampling duration, flow rate, deposition versus active collection, lower size cutoff and fibre definition. Longer and larger fibres may be efficiently captured yet have different aerodynamic behaviour from compact fragments of the same physical dimension. A deposition flux is not the same quantity as a breathing-zone concentration, and neither directly equals deposited lung dose (Brahney et al. 2020; Wright et al. 2020; Dris et al. 2016).

Remote or protected-area deposition demonstrates atmospheric transport but should not be interpreted as personal inhalation dose. Regional mass extrapolations are sensitive to particle density, assumed shape, unmeasured small particles and site representativeness. At the other extreme, occupational textile or flock environments may produce much higher fibre concentrations than ordinary residences and should be evaluated as distinct exposure scenarios rather than used as proxies for the general population.

Airborne risk assessment requires chemically confirmed size-resolved concentration, aerodynamic characterization, time-activity information and respiratory deposition or clearance data. The present database supports inhalation as a real route but does not provide a single robust global daily inhaled mass.

Evidence-weighted interpretation

Atmospheric deposition, suspended-air concentration, personal breathing-zone exposure and deposited respiratory dose are sequential but distinct endpoints. A passive collector measures fallout to a surface; it does not directly measure what a person inhales. A room sampler measures air at a location; it does not capture activity-specific breathing-zone concentration. Converting either result to lung dose additionally requires breathing rate, aerodynamic behavior, fibre geometry, regional deposition and clearance (Wright et al. 2020; Islam et al. 2023).

The airborne-fibre literature also demonstrates why the denominator matters. In the Paris indoor/outdoor survey, roughly two-thirds of classified fibres were natural or cellulosic and about one-third were petrochemical (Dris et al. 2017). The earlier atmospheric-fallout study likewise found that only a minority of characterized fibres were petrochemical (Dris et al. 2016). These results do not negate synthetic-fibre exposure; they show that plastic is one component of a much larger airborne fibre and particle mixture and that particle-free controls cannot establish plastic-specific toxicity.

Mechanistic airway evidence must separate the intact fibre from associated chemistry. In the textile-fibre airway study, nylon impaired organoid development more than polyester, but nylon leachate reproduced much of the response (Song et al. 2024). The appropriate inference is formulation- and leachate-specific hazard under the tested conditions, not a general conclusion that all intact textile microfibres have the same intrinsic toxicity.

Aves et al. (2026) analyzed aliquots from the same remote New Zealand aerosol sample set using micro-FTIR, micro-Raman, Nile Red fluorescence microscopy, pyrolysis-GC/MS and thermal-desorption proton-transfer-reaction mass spectrometry. The five methods reported different concentrations or polymer profiles, and 98% of particle-resolved detections were below 10 micrometres. This directly demonstrates that airborne results are method-defined and that datasets with different size capability or measurands should not be pooled.

Evangelou et al. 2026 also clarifies how atmospheric distributions should be reported. The global arithmetic mean concentration of 31 particles/m³ was roughly 1,000 times the median of 0.03 particles/m³ because a small number of extreme measurements dominated the mean. Median, percentile and full-distribution reporting are therefore more representative of typical measured outdoor concentrations than an unqualified mean or isolated maximum.

The Liu et al. climate-forcing paper is best separated into two evidentiary layers. Its particle-level optical measurements show that selected coloured and pigmented plastic fragments can absorb substantially more light than unpigmented material, with colour and particle size more influential than base-polymer chemistry. That laboratory result is useful for defining optical properties of the tested formulations; it does not establish how much similarly pigmented plastic mass is present in the global atmosphere.

The global forcing estimate is much less secure. The model assumed a combined microplastic-plus-nanoplastic atmospheric burden of 0.092 mg/m², approximately 66% of its modeled black-carbon burden of 0.14 mg/m². Nanoplastics supplied 0.033 of the total 0.039 W/m² forcing, about 85%, despite being extrapolated from assumed size distributions and a prescribed 28-day lifetime. The reported ±0.019 W/m² range did not include uncertainty in MNP emission rates. Evangelou et al. subsequently found that existing atmospheric inventories overpredicted size-aligned measurements by two to four orders of magnitude and that land, not the ocean, dominated particle-number emissions. The Liu forcing magnitude and North Pacific hotspot are therefore retained as model-dependent high-end scenarios rather than established climate effects.

Table 4. Airborne evidence chain from occurrence to respiratory risk

Evidence stage	What is measured	Representative evidence	What remains unmeasured
Atmospheric deposition	Fallout to collectors	Urban/remote deposition (Wright et al. 2020; Brahney et al. 2020)	Personal breathing-zone concentration
Global atmospheric synthesis	Concentration and deposition distributions	Median concentration 0.03 particles/m ³ ; 0.08 land; 0.003 ocean; inventories two to four orders high (Evangelou et al. 2026)	Complete <5 µm range, personal breathing-zone dose, full source inventory and harmonized methods
Suspended indoor/outdoor air	Fibres per cubic metre	1-60 indoors; 0.3-1.5 outdoors (Dris et al. 2017)	Mass, full small-particle range and individual intake
Airway deposition model	Predicted regional deposition	Shape- and flow-dependent trajectories (Islam et al. 2023)	Validated population deposition and clearance
Human lung occurrence	Particles per tissue mass	Blank-adjusted 0.69 particles/g (Jenner et al. 2022)	Inhaled dose, residence time and disease causation
Mechanistic airway response	Organoid/animal response	Nylon and leachate effects (Song et al. 2024)	Realistic dose and intact-fibre specificity

Selected evidence records

- **(Moderate)**. Brahney, J., Hallerud, M., Heim, E., Hahnenberger, M., & Sukumaran, S. (2020). Plastic rain in protected areas of the United States. *Science*, 368, 1257–1260. <https://doi.org/10.1126/science.aaz5819>
- **(Moderate)**. Stephanie L. Wright, Julia Ulke, Anais Font, Kelly L. A. Chan & Frank J. Kelly. 2020. Atmospheric microplastic deposition in an urban environment and an evaluation of transport. *Environment International* 136:105411.
- **(Moderate)**. Rachid Dris, Johnny Gasperi, Mohammed Saad, Cécile Mirande & Bruno Tassin. 2016. Synthetic fibers in atmospheric fallout: A source of microplastics in the environment? *Marine Pollution Bulletin* 104:290-293.
- **(Moderate)**. Dris, R., Gasperi, J., Mirande, C., Mandin, C., Guerrouache, M., Langlois, V., & Tassin, B. (2017). A first overview of textile fibers, including microplastics, in indoor and outdoor environments. *Environmental Pollution*, 221, 453–458. <https://doi.org/10.1016/j.envpol.2016.12.013>
- **(Moderate)**. Liu, Y., Fu, H., Zhang, H., et al. (2026). Atmospheric Warming Contributions from Airborne Microplastics and Nanoplastics. *Nature Climate Change*. <https://doi.org/10.1038/s41558-026-02620-1>

8. Food and Beverage Exposure

Microplastic and other anthropogenic particles have been reported in seafood, packaged foods, beverages and food-contact systems. These studies establish potential ingestion pathways but usually quantify particles in the product, not the absorbed or retained dose. Concentrations vary with processing, packaging, preparation temperature, storage, digestion method, lower size cutoff and confirmation rate.

Seafood and protein surveys are useful for exposure characterization but often include fibres and particles of mixed origin. Benchmark extraction and identification studies show why recovery and contamination controls are essential. Product-use studies, such as disposable cups or heated contact materials, can generate high counts under selected conditions; those counts require explicit size and mass context. (Milne 2024); (Traylor et al. 2024); (Dehaut et al. 2016); (Zhou 2023); (Qajarloo et al. 2026)

Selected quantitative examples illustrate the incompatibility of direct pooling: protein foods were reported at a mean 0.3 particles/g for particles at least 45 micrometres (Milne 2024), whereas Spanish bottled water had a median 0.73 particles/L above 15 micrometres (Gálvez-Blanca et al. 2024). Matrix, denominator, lower-size cutoff, recovery and confirmation rules differ, so these values describe separate source pathways rather than a common exposure distribution.

Integrated evidence synthesis

Food and beverage measurements face demanding blank-control and matrix-recovery problems. Digestion, filtration and transfer steps can introduce fibres, while fats, proteins, salts and pigments can interfere with spectroscopy or staining. Studies that report anthropogenic particles rather than confirmed polymers should not be recoded as microplastic measurements. A spectroscopically confirmed subset can inform composition, but extrapolating that subset to every visually selected object adds uncertainty (Traylor et al. 2024).

Concentrations vary by food type, processing, preparation, packaging, geography and analytical cutoff. Seafood gut-content studies demonstrate ingestion by organisms but do not automatically quantify human intake because uneaten organs may contain most particles. Edible-tissue studies are more directly relevant, yet recovery and contamination remain important, and some well-controlled investigations report negligible occurrence above their size threshold (Qin et al. 2026).

Packaging can be a source in some systems, but packaging is not the only source and an association with packaged products does not prove migration from the package. Processing equipment, air, water, ingredients and laboratory handling may contribute. Source attribution requires matched controls, product-contact experiments or chemical and morphological evidence consistent with the suspected material.

Dietary exposure estimates should preserve the distinction between measured concentration and modeled intake. Consumption rates, serving size, preparation and the number of unmeasured food categories can dominate uncertainty. The evidence confirms that some foods and beverages contain plastic or anthropogenic particles; it does not yet support a precise universal dietary dose.

Evidence-weighted interpretation

Food and beverage studies encompass several different exposure questions: particles within edible tissue, particles deposited during processing or preparation, and particles released from packaging or utensils. These pathways require different controls. Tissue digestion must remove biological material without destroying polymers, while packaging-release experiments must distinguish the tested article from ingredients, background air, processing equipment and pre-existing particles. The seafood benchmark study shows that digestion chemistry can materially change polymer recovery and therefore reported abundance (Dehaut et al. 2016).

Edible-tissue findings must also preserve the analytical classification. The U.S. West Coast seafood study reported anthropogenic particles, a category broader than confirmed plastic (Traylor et al. 2024). A Malaysian fish study reported negligible muscle occurrence within its greater-than-100-micrometre window, but that result does not address smaller particles (Qin et al. 2026). These studies support bounded external dietary occurrence, not universal tissue concentrations or absorbed human dose.

The Spanish bottled-water study provides an unusually useful denominator by reporting both microplastics and artificial cellulosic particles. Median concentrations were approximately 0.73 microplastic particles per litre and 1.70

artificial cellulosic particles per litre; estimated microplastic mass was approximately 1.61 micrograms per litre and intake was 4-18 ng/kg body weight/day (Gálvez-Blanca et al. 2024). The mass was calculated from particle geometry and density, and particles below 15 micrometres were outside routine resolution, so these values should remain study-specific rather than being pooled with studies using different cutoffs.

The selected protein-food survey reported approximately 0.3 particles per gram and modeled about 11,000 particles per person per year from the included protein categories, equivalent to roughly 30 particles per day (Milne 2024). Only a subset of objects was chemically analyzed, the lower size cutoff was approximately 45 micrometres, and the selected foods were not a complete diet. The result is therefore a category-specific external-intake estimate, not a total dietary exposure or internal dose.

Table 5. Food and beverage evidence hierarchy

Evidence type	Representative finding	What it supports	Critical limitation
Measured protein foods	>45-micrometre particles in selected foods (Milne 2024)	Category-specific external intake	Incomplete diet; subset confirmation
Edible seafood tissue	Anthropogenic particles in seafood (Traylor et al. 2024)	Potential dietary occurrence	Anthropogenic is broader than confirmed plastic
Fish tissue size window	Muscle occurrence negligible above 100 micrometres (Qin et al. 2026)	Bounded result for larger particles	Does not address smaller particles
Bottled water	0.73 MPs/L; 1.70 artificial cellulosic particles/L (Gálvez-Blanca et al. 2024)	Study-specific count and mass estimate	Below 15 micrometres excluded; mass modeled
Packaging release	Temperature/time-dependent release reports (Zhou 2023; Qajarloo et al. 2026)	Possible product-use pathway	Background and processing attribution

Selected evidence records

- **(Low).** Madeleine H. Milne. 2024. Exposure of U.S. adults to microplastics from commonly-consumed proteins. *Environmental Pollution*, 343 (2024) 123233.
- **(Moderate).** Traylor, S. D., Granek, E. F., Duncan, M., & Brander, S. M. (2024). From the ocean to our kitchen table: anthropogenic particles in the edible tissue of U.S. West Coast seafood species. *Frontiers in Toxicology*, 6, 1469995. <https://doi.org/10.3389/ftox.2024.1469995>
- **(Moderate).** Dehaut, A., Cassone, A.-L., Frère, L., Hermabessiere, L., Himber, C., Rinnert, E., Rivière, G., Lambert, C., Soudant, P., Huvet, A., Duflos, G., & Paul-Pont, I. (2016). Microplastics in seafood: Benchmark protocol for their extraction and characterization. *Environmental Pollution*, 215, 223–233. <https://doi.org/10.1016/j.envpol.2016.05.018>
- **(Low).** Guanyu Zhou. 2023. **How many microplastics do we ingest when using disposable drink cups?** *Journal of Hazardous Materials*, 441 (2023) 129982.
- **(Low).** Asadi Qajarloo, Z., Arfaeinia, H., Jahanara, M., et al. (2026). Influence of temperature and storage time on microplastic release from packaged sausages. *Scientific Reports*. <https://doi.org/10.1038/s41598-026-53153-8>

9. Drinking Water and Tap Water

Drinking-water evidence is highly method-dependent. A well-controlled Danish survey using large volumes and micro-FTIR found very few confirmed particles, with results near method-background and detection-limit considerations. Other source-to-tap studies report higher counts and show that treatment and distribution networks can remove, retain, introduce or remobilize particles.

Pipe material is only one possible source. Raw-water loading, treatment media, reservoirs, household plumbing, storage, sampling contamination and analytical cutoff all contribute. A rise from treatment plant to tap does not by itself identify the pipe as the source, while a decline does not prove destruction. The strongest interpretation is system-specific fate rather than a universal material ranking. (Feld et al. 2021); (Adediran et al. 2024); (Sun et al. 2024); (Mukotaka 2021); (Radityaningrum et al. 2023)

The controlled quantitative layer retains study-specific water denominators rather than a pooled concentration. Examples include 0.2 particles/L in a Danish 10-100 micrometre subset (Feld et al. 2021), 0.64-1.58 particles/L among Spanish bottled-water brands above 15 micrometres (Gálvez-Blanca et al. 2024), source-to-tap means of 0.6, 5.2 and 1.2 particles/L in Haikou (Li et al. 2024), and higher product- or matrix-specific values in selected bottled water, beverages, container-release tests and snow meltwater. These data illustrate method heterogeneity, not a universal drinking-water concentration range.

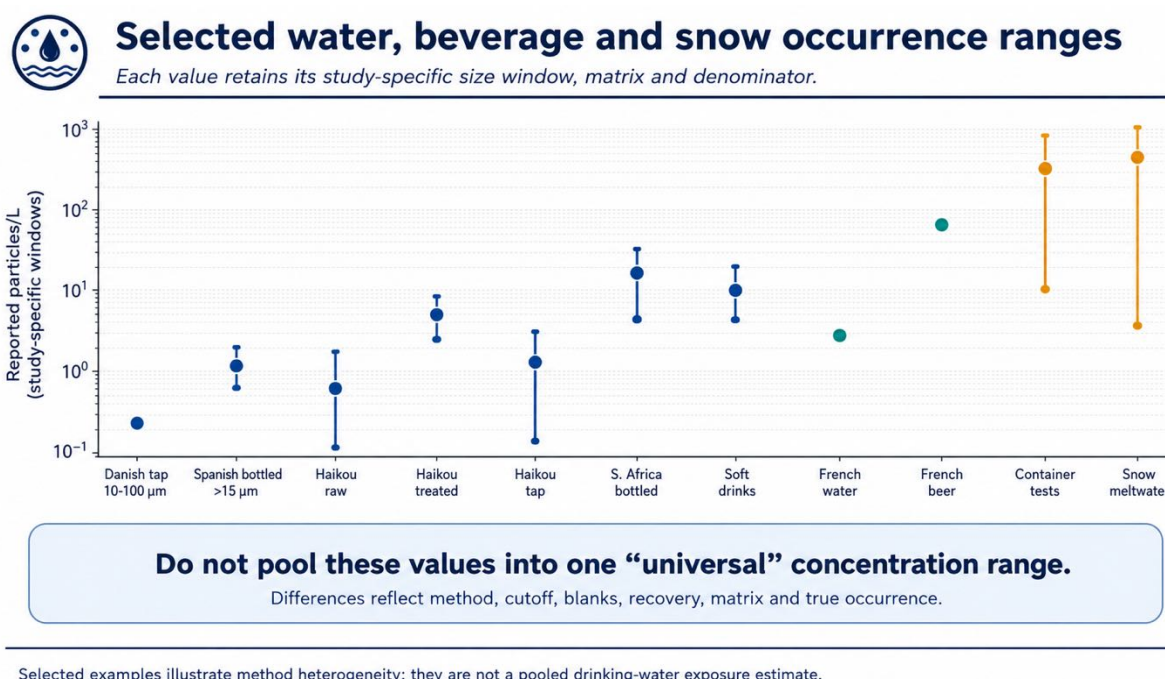


Figure 6. Selected water and beverage concentrations retained with study-specific denominators and size windows.

Integrated evidence synthesis

Drinking-water evidence should be organized along the source-to-tap pathway. Raw-water concentration, treatment performance, finished-water storage, distribution-pipe condition, premise plumbing and sampling hardware can each alter the observed result. A particle found at the tap may have entered the source water, survived treatment, been introduced during distribution, or arisen from the sample container or laboratory. Paired sampling and process-stage mass balance are therefore more informative than isolated grab samples.

Analytically rigorous tap-water studies can find very few confirmed particles, sometimes at levels comparable with blanks or quantification limits (Feld et al. 2021). Other studies report higher values, particularly when smaller size windows, staining or thermal methods are used. Those results may all be internally valid within their method boundaries, but they are not directly interchangeable. Lowering the cutoff can increase counts by orders of magnitude because environmental particle distributions are steeply weighted toward smaller sizes.

Two 2026 studies extend the measured lower-size range. Jamison Hart and Lenhart used SEM plus optical photothermal infrared spectroscopy across 300 nm to 42.3 micrometres in samples from four treatment plants and six bottled-water brands; bottled water had significantly higher micro- and nanoplastic concentrations, particularly in smaller size classes. Jüngling et al. demonstrated automated Raman analysis down to 500 nm in a proof-of-principle potable-water sample, where only $0.36 \pm 0.13\%$ of particles in the 0.5-10 micrometre range were identified as plastics. Both results are method- and sample-specific occurrence evidence; neither establishes absorbed dose or health risk.

Treatment commonly reduces particles through coagulation, sedimentation, filtration and membrane processes, but removal is size- and process-dependent and transfers material to residuals or sludge (Xu et al. 2024). Finished-water values should not be described as treatment failure without the influent concentration and recovery. Conversely, a high percentage removal can coexist with a measurable residual when raw-water concentration is high.

Plastic pipes and fittings can release particles under abrasion, ageing or installation conditions, but attribution requires polymer confirmation and comparison with scale, corrosion products, mineral debris and other distribution-system particles. The relevant risk question is the confirmed plastic fraction, its size and mass, not the total number of particles in water. Evidence from controlled source-to-tap studies does not support assuming that every drinking-water particle is plastic or that plastic pipe is necessarily the dominant source.

Evidence-weighted interpretation

Source-to-tap data do not support a single monotonic narrative in which treatment always removes particles and pipes always add them. In the Haikou study, mean counts were reported as 0.6 particles/L in raw water, 5.2/L after treatment and 1.2/L at household taps (Li et al. 2024). Another supply-chain study reported 2.3-17.3/L in raw water, 12.0-25.3/L in treated water and 8.0-19.7/L in household water (Sun et al. 2024). These patterns show that treatment, storage, distribution, sampling and analytical variability can produce increases or decreases at different stages; they do not identify one pipe material as the unique source.

The highest-quality tap-water occurrence record in the database found only seven confirmed particles across all Danish samples and an average of approximately 0.2 particles/L in the 10-100-micrometre subset (Feld et al. 2021). Low counts create wide statistical uncertainty, and the method does not establish the absence of smaller particles. The proper conclusion is very low confirmed occurrence within the validated window, not zero exposure.

Treatment performance must be reported with the endpoint measured. Conventional plants in East Java reported approximately 62-66% count removal, with coagulation-flocculation contributing the largest step and smaller particles removed less efficiently (Radityaningrum et al. 2023). A separate plant study reported changes in particle mass across stages (Xu et al. 2024). Count removal and mass removal need not agree, and neither demonstrates destruction unless retained solids, residual streams and mineralization are measured.

Distribution-network studies show deposition, retention and remobilization as well as possible local inputs (Adediran et al. 2024). A difference between treatment-plant effluent and tap water is therefore a system observation, not proof that plastic piping generated the difference. Source attribution requires material-specific signatures, controls for treatment chemicals and equipment, repeated sampling and a complete hydraulic and particulate mass balance.

Table 6. Drinking-water evidence from source to tap

Study/system	Reported result	Interpretation supported	Interpretive boundary
Danish tap water (Feld et al. 2021)	7 confirmed particles total; about 0.2/L in 10-100 micrometres	Very low confirmed counts in validated window	Low-count uncertainty; smaller particles not covered
Haikou supply chain (Li et al. 2024)	0.6 raw; 5.2 treated; 1.2 tap particles/L	Counts can rise and fall across system	No unique pipe-source attribution
Typical supply chain (Sun et al. 2024)	2.3-17.3 raw; 12.0-25.3 treated; 8.0-19.7 household particles/L	Removal, introduction and remobilization all plausible	One system; method-specific
East Java treatment (Radityaningrum et al. 2023)	About 62-66% count removal	Conventional treatment can reduce measured counts	Count removal is not mass removal or destruction

Study/system	Reported result	Interpretation supported	Interpretive boundary
Distribution network (Adediran et al. 2024)	Concentration changed through reservoirs and taps	Deposition/retention/remobilization occur	Material source not uniquely identified
Mass-based plant study (Xu et al. 2024)	Particle mass changed across stages	Mass endpoint complements counts	Single plant and size/recovery assumptions
Submicrometre treated/bottled water comparison (Jamison Hart & Lenhart 2026)	300 nm-42.3 micrometres; bottled water significantly higher, especially for smaller particles	Extends the observed size window and supports source-specific comparison	Four plants and six brands; method-specific occurrence, not absorbed dose or risk

Selected evidence records

- **(High)**. Feld, L., da Silva, V. H., Murphy, F., Hartmann, N. B., & Strand, J. (2021). A study of microplastic particles in Danish tap water. *Water*, 13(15), 2097. <https://doi.org/10.3390/w13152097>
- **(Moderate)**. Gbotemi A. Adediran, et al. 2024. Fate and behaviour of microplastics (>25 µm) within the water distribution network, from water treatment works to service reservoirs and customer taps. *Water Research* 255:121508.
- **(Not scored - secondary synthesis)**. Xiangying Sun, Yunjie Zhu, Lihui An, Yan Liu, Yin Zhuang, Yubang Wang, Mingdong Sun & Qiujin Xu. 2024. Microplastic Transportation in a Typical Drinking Water Supply: From Raw Water to Household Water. *Water* 16(11):1567.
- **(High)**. Arata Mukotaka. 2021. Rapid analytical method for characterization and quantification of microplastics in tap water using a Fourier-transform infrared microscope. *Science of the Total Environment*, 790 (2021) 148231.
- **(Moderate)**. Radityaningrum, A.D., Trihadiningrum, Y., & Soedjono, E.S. (2023). Performance of conventional drinking water treatment plants in removing microplastics in East Java, Indonesia. *Journal of Ecological Engineering*, 24(6), 129–143. <https://doi.org/10.12911/22998993/162785>
- **(Supplementary source outside the frozen baseline)**. Jamison Hart, M. N., & Lenhart, J. J. (2026). What's in your water? A comparative analysis of micro- and nanoplastics in treated drinking water and bottled water. *Science of the Total Environment*, 1011, 181148. <https://doi.org/10.1016/j.scitotenv.2025.181148>

10. Human Ingestion

Human ingestion is plausible through food, beverages, drinking water and hand-to-mouth transfer. Direct stool detection confirms gastrointestinal passage but does not quantify absorption. Diet-based exposure estimates vary substantially because they combine heterogeneous occurrence datasets and often extrapolate from limited food categories.

Particle number is particularly unstable as the lower size cutoff decreases. Count-to-mass conversions require assumptions about size, density, shape and polymer. Corrected exposure papers and provenance records show that high-profile intake estimates have changed after numerical correction. Intake should therefore be reported separately as measured count, measured mass, modeled count and modeled mass, with retained dose treated as a distinct quantity. (Milne 2024); (Schwabl et al. 2019); (Nor et al. 2021); (Cox et al. 2020); (University of Newcastle and WWF 2019)

The selected adult model (Nor et al. 2021) estimated 883 particles/person/day and 583 ng/person/day. These are model outputs assembled from heterogeneous occurrence studies, not direct total-intake measurements. The transparent 70-year constant-intake scenario yields 14.91 mg external mass and should not be interpreted as absorbed or retained dose.

Integrated evidence synthesis

Ingestion estimates combine concentration with consumption, but the resulting number remains external intake. The selected adult model estimated 883 particles and 583 ng per person per day across eight food categories and inhalation (Nor et al. 2021). This estimate is useful for scale, yet it depends on the included categories, heterogeneous methods and the modeled 1-5,000 micrometre input domain. It should not be represented as a directly measured daily dose for every person.

Particle count and mass can imply different exposure rankings. A sample dominated by very small particles may have a high count but little mass, whereas a few larger particles may dominate mass. Without a measured size distribution and density, converting count to mass can produce unstable results. Surface area and particle number may be relevant to some mechanisms, but they likewise require characterized geometry rather than an assumed spherical particle.

Stool studies support ingestion and excretion but do not measure the amount absorbed. Recovery from faeces depends on transit time, digestion, sample mass and analytical cutoff. The fact that particles are excreted is consistent with limited uptake of larger material, while an apparent difference between intake and excretion cannot be converted to absorption unless all input pathways and analytical recoveries are measured.

The most defensible ingestion conclusion is therefore qualitative and range-based: ordinary human intake occurs, likely varies widely among individuals and settings, and is dominated in uncertainty by the smallest measured size classes and by unmeasured foods. Quantitative internal dose remains substantially less certain than external occurrence.

Evidence-weighted interpretation

Two quantitative exposure approaches in the evidence base should not be conflated. The protein-food survey begins with measured occurrence in selected foods above a relatively large size cutoff and then applies consumption assumptions (Milne 2024). The multimedia model combines heterogeneous food and inhalation datasets across a broader 1-5,000-micrometre input domain and estimates 883 particles and 583 ng per adult per day (Nor et al. 2021). The estimates answer different questions and cannot be averaged.

The stool case series reported a median of about 20 particles per 10 grams, equivalent to approximately 2 particles per gram within its analytical window (Schwabl et al. 2019). Stool occurrence confirms gastrointestinal passage and excretion. It does not provide an absorption fraction because daily intake, whole-stool output, particle recovery, transit time and all exposure routes were not measured concurrently.

Heo et al. 2024 systematically reviewed 76 studies estimating dietary microplastic intake. Reported count-based intakes varied across many orders of magnitude because studies sampled different foods, lower-size cutoffs and analytical methods. The review supports method dependence and the need for quality assessment; it does not supply a single compatible mass estimate that can be pooled with Nor et al. 2021.

Xu et al. 2025 compared 258 colorectal-cancer cases with 493 controls and reported higher fecal particle counts in cases (median 62 versus 43 items/g dry weight) and an adjusted odds ratio of 11.3 for the highest versus lowest quartile. This is a large association, but fecal concentration is not a validated historical internal-dose measure. Colorectal disease, diet, bowel transit, treatment and microbiome differences can alter fecal particles, so temporality and reverse causation remain unresolved.

Selected evidence records

- **(Low).** Madeleine H. Milne. 2024. Exposure of U.S. adults to microplastics from commonly-consumed proteins. *Environmental Pollution*, 343 (2024) 123233.
- **(Low).** Schwabl, P., Köppel, S., Königshofer, P., Bucsics, T., Trauner, M., Reiberger, T., & Liebmann, B. (2019). Detection of various microplastics in human stool: a prospective case series. *Annals of Internal Medicine*, 171(7), 453–457. <https://doi.org/10.7326/M19-0618>
- **(Not scored - secondary synthesis).** Nur Hazimah Mohamed Nor, Merel Kooi, Noël J. Diepens, and Albert A. Koelmans. 2021. Lifetime Accumulation of Microplastic in Children and Adults.
- **(Context only).** Cox, K.D., Covernton, G.A., Davies, H.L., Dower, J.F., Juanes, F., and Dudas, S.E. (2020). Correction to Human Consumption of Microplastics. *Environmental Science & Technology*, 54, 10974.
- **(Not scored - technical/context).** University of Newcastle and World Wide Fund for Nature. 2019. Assessing Plastic Ingestion from Nature to People. Commissioned quantitative analysis.

11. Human Inhalation

Humans continuously inhale a complex mixture of airborne particles; plastic is one component of that ordinary dust background. Human lung-tissue studies and airway-deposition models support respiratory deposition, but the quantitative inhaled and retained plastic dose remains uncertain. Particle deposition is size-, shape- and airflow-dependent, and clearance by mucociliary transport or macrophage-mediated pathways is not adequately incorporated in many exposure claims. Detection in lung tissue is therefore expected for many particle classes and does not by itself establish material-specific disease.

Textile studies show that synthetic fibres can become airborne, but natural and semi-synthetic fibres also contribute substantially. Occupational settings may involve higher concentrations than general environments and require separate evaluation. Current evidence does not justify translating environmental deposition counts directly into lung disease risk. (Wright et al. 2020); (Islam et al. 2023); (Jenner et al. 2022); (Song et al. 2024); (Amato-Lourenço et al. 2021)

The Paris airborne-fibre study reported 1-60 fibres/m³ indoors and 0.3-1.5 fibres/m³ outdoors (Dris et al. 2017), with approximately two-thirds classified as natural or cellulosic and one-third petrochemical. OSHA particulate limits are expressed as workplace mass concentrations, not environmental fibre counts. The two datasets cannot be converted to a common axis without particle-resolved size, density and aerodynamic information; occupational limits are contextual and not microplastic-specific thresholds.

A separate global outdoor synthesis provides a broader but methodologically distinct benchmark. Evangelou et al. 2026 reported a median atmospheric microplastic concentration of 0.03 particles/m³ and an interquartile range of 0.002-0.1 particles/m³. This supports very low typical outdoor counts within the included analytical windows, but the values cannot be converted directly into inhaled mass or deposited lung dose without particle-resolved size, density, shape, breathing-zone and respiratory-deposition information.

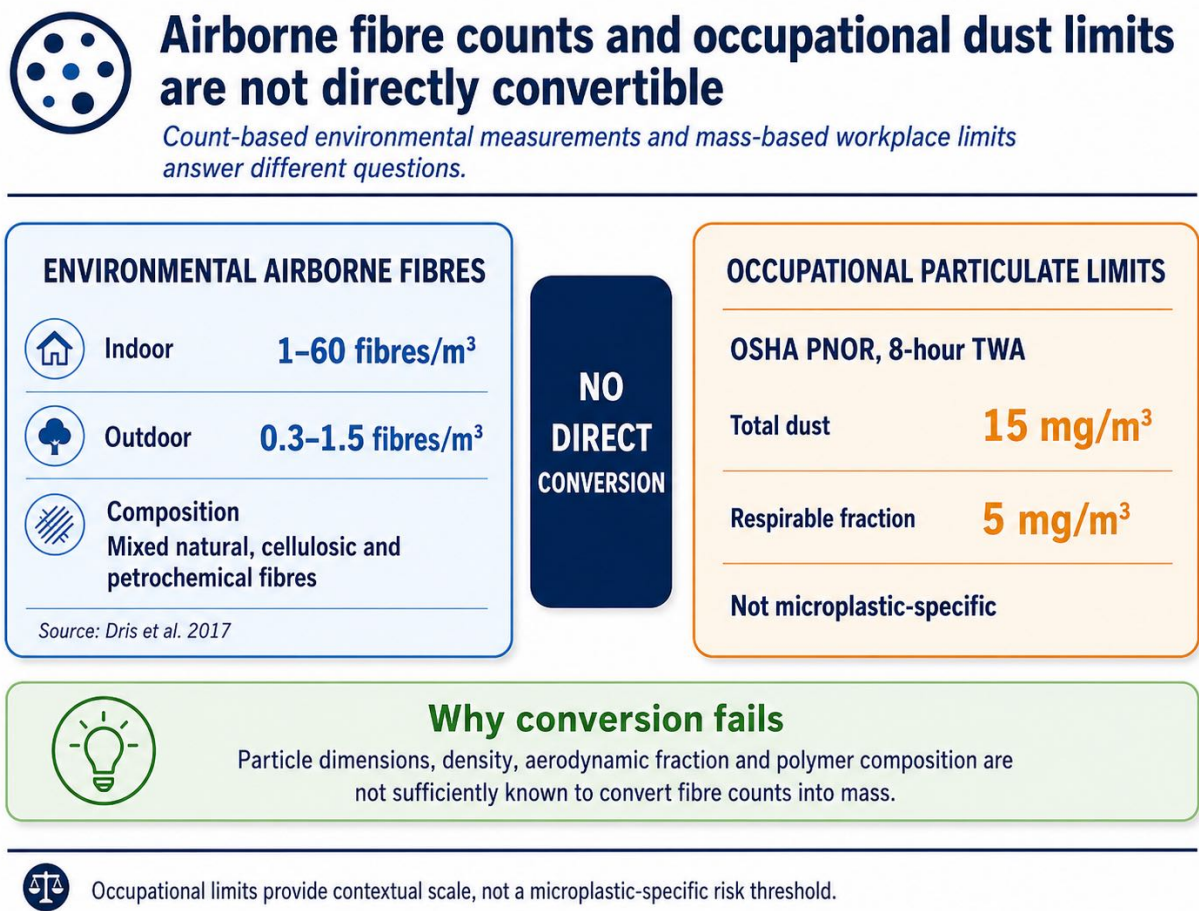


Figure 7. Airborne fibre counts and occupational particulate mass context are not directly convertible.

Integrated evidence synthesis

Inhalation risk depends on the concentration in the breathing zone, aerodynamic properties, breathing rate, duration, deposition region and clearance. Physical length is particularly important for fibres, while compact fragments are more often described by aerodynamic diameter. A fibre that is visible by microscopy may not behave like a spherical particle of the same largest dimension, and an optical count cannot be translated directly to respirable mass.

Environmental studies report fibres and fragments in indoor and outdoor air, but polymer-confirmation rates vary. Natural and regenerated-cellulose fibres can dominate total fibre counts in many environments, so plastic-specific inhalation estimates require chemical identification rather than visual assignment. Procedural blanks are essential because clothing, wipes, filters and laboratory air can contribute fibres (Wright et al. 2020; Islam et al. 2023).

The quantitative comparison displays environmental airborne fibre counts separately from OSHA particulate mass limits. This is contextual only: particles per cubic metre and milligrams per cubic metre are different quantities, the regulated dust category is not microplastic-specific, and occupational limits are not universal safe thresholds. The comparison illustrates the absence of a validated conversion, not a regulatory conclusion.

Human lung detections support the plausibility of respiratory deposition for some particles, but they do not quantify inhaled dose or retention time. A robust inhalation assessment requires longitudinal or controlled deposition data, validated lung-matrix methods, size-resolved exposure and distinction between ambient, household and occupational conditions.

Evidence-weighted interpretation

Measured airborne counts remain sparse and method-bounded. The Paris survey reported approximately 1-60 fibres/m³ indoors and 0.3-1.5 fibres/m³ outdoors, but total fibres were not synonymous with plastic and cannot be converted to mg/m³ without size, density and shape information (Dris et al. 2017). Occupational particulate limits expressed in mass units are therefore contextual only and cannot be used as direct thresholds for these environmental fibre counts.

The global synthesis additionally found medians of 0.08 particles/m³ over land and 0.003 particles/m³ over the ocean. Those medians are more informative for typical outdoor occurrence than the highly skewed arithmetic mean, but they remain study- and size-window-dependent and do not replace personal exposure measurements (Evangelou et al. 2026).

The lung micro-FTIR study reported a blank-adjusted mean of approximately 0.69 particles per gram of tissue, but only one sample met the study's quantification criterion, exposure histories were unavailable and the surgical cohort was not population-representative (Jenner et al. 2022). This supports plausible deposition of some particles; it does not establish inhaled dose, retention time, accumulation rate or disease causation.

A complementary inorganic-particle study provides essential context for interpreting lung detections. Hayden et al. 2023 used automated in-situ FE-SEM/EDS on 38 selected surgical lung biopsies from deployed and nondeployed U.S. military service members and characterized 124,018 inorganic particles across 45 mineral or particle classes. Common material included clays, feldspars, quartz, titanium dioxide and numerous metal or metal-oxide particles; rare lead-, uranium- and thorium-containing particles were also documented. More than 90% of analyzed particles had a greatest dimension below 2.5 micrometres. The study therefore shows directly that plastic, when detected in lung tissue, is encountered within a large and chemically diverse background of nonplastic particulate matter. Hayden does not provide a numerical plastic-to-nonplastic ratio because FE-SEM/EDS was qualitative, examined a fixed tissue area and was optimized for inorganic particles with backscattered-electron contrast above carbon. Its value is contextual rather than compositional: discussing plastic lung detections while excluding the many other particles present removes the particulate background needed for meaningful exposure and risk interpretation.

Controlled human tracer evidence further constrains what inhalation can imply. Miller et al. 2017 exposed healthy male volunteers to approximately 4-5 nm primary gold nanoparticles for 2 hours under controlled conditions and detected gold in blood in 12 of 14 subjects at 24 hours. A second human experiment found much greater systemic appearance after approximately 4 nm than approximately 34 nm primary particles; mouse experiments spanning 2-200 nm likewise showed a steep size dependence, with translocation falling markedly above the approximately 10-30 nm range. The published correction reduced the authors' illustrative 24-hour translocation estimate to approximately 0.02% of inhaled mass. This supports limited systemic passage of ultrasmall nanoparticles but does not justify extrapolation to micrometre-scale microplastics, whose dimensions are orders of magnitude larger.

Airway-deposition modeling predicts substantial dependence on particle shape, size, flow and anatomy (Islam et al. 2023). A complete inhalation assessment therefore requires particle-resolved exposure distributions rather than a single count concentration. It must also include mucociliary clearance, macrophage-mediated clearance, dissolution or leaching, and the possibility that long fibres behave differently from compact fragments.

Selected evidence records

- **(Moderate)**. Stephanie L. Wright, Julia Ulke, Anais Font, Kelly L. A. Chan & Frank J. Kelly. 2020. Atmospheric microplastic deposition in an urban environment and an evaluation of transport. *Environment International* 136:105411.
- **(Moderate)**. Islam, M. S., et al. (2023). How microplastics are transported and deposited in realistic upper airways? *Physics of Fluids*, 35, 063319. <https://doi.org/10.1063/5.0150703>
- **(Low)**. Lauren C. Jenner, Jeanette M. Rotchell, Robert T. Bennett, Michael Cowen, Vasileios Tentzeris & Laura R. Sadofsky. 2022. Detection of microplastics in human lung tissue using micro-FTIR spectroscopy. *Science of the Total Environment* 831:154907.
- **(Moderate)**. Song, S., van Dijk, F., Vasse, G. F., Liu, Q., Gosselink, I. F., Weltjens, E., et al. (2024). Inhalable textile microplastic fibers impair airway epithelial differentiation. *American Journal of Respiratory and Critical Care Medicine*, 209(4), 427–443. <https://doi.org/10.1164/rccm.202211-2099OC>
- **(Not scored - secondary synthesis)**. Luís Fernando Amato-Lourenço, Regiani Carvalho-Oliveira, Gabriel Ribeiro Júnior, et al. 2021. Presence of airborne microplastics in human lung tissue. *Journal of Hazardous Materials* 416:126124.
- **(Supplementary primary context outside frozen baseline)**. Hayden, L., Strausborger, S., & Lewin-Smith, M. (2023). 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), 235-243. <https://doi.org/10.1093/micmic/ozac015>.
- **(Supplementary primary kinetic/context publication lineage outside frozen baseline)**. Miller, M. R., Raftis, J. B., Langrish, J. P., et al. (2017). Inhaled Nanoparticles Accumulate at Sites of Vascular Disease. *ACS Nano*, 11(5), 4542-4552. <https://doi.org/10.1021/acsnano.6b08551>. Correction: *ACS Nano*, 11, 10623-10624. <https://doi.org/10.1021/acsnano.7b06327>.

12. Absorption, Clearance and Retention

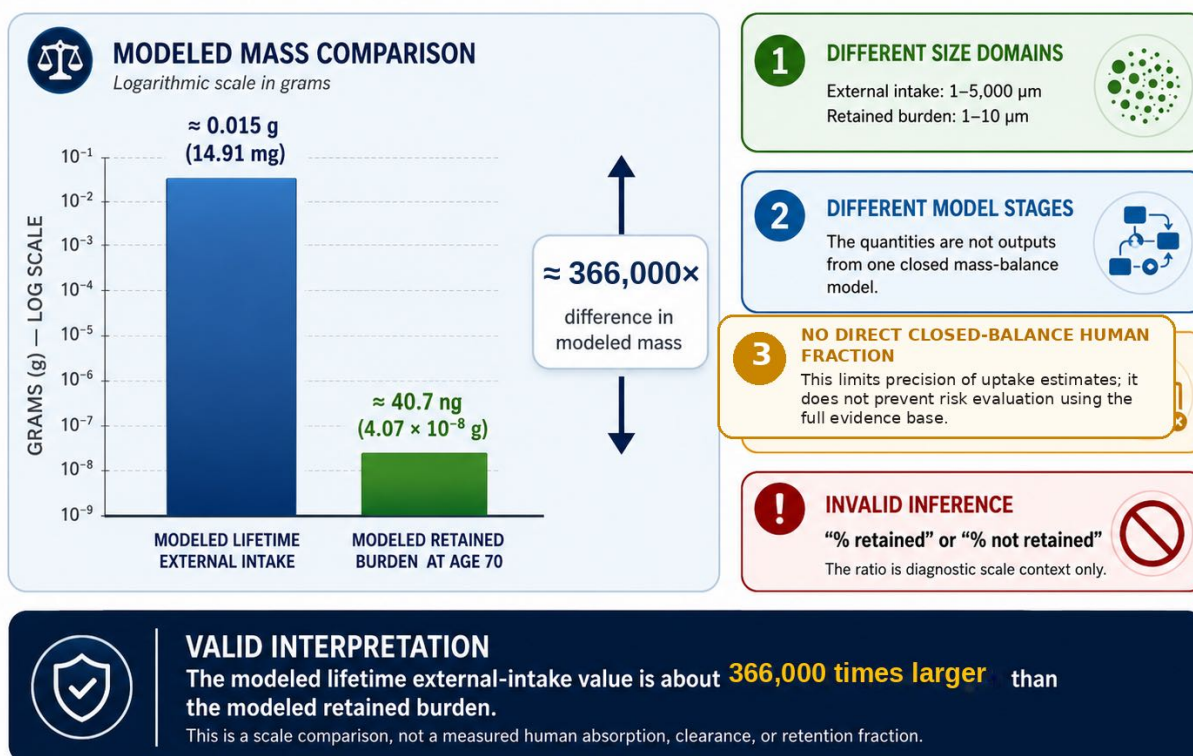
Absorption is not equivalent to ingestion, and tissue detection is not equivalent to long-term retention. Specific animal studies report that most administered fragments remain in the gastrointestinal tract and are eliminated rapidly, while some tracer studies report distant-tissue signals. These results differ by particle size, labeling, dose, polymer, exposure duration and detection method.

Earthworm and invertebrate studies often find gut-associated particles with low tissue translocation, illustrating the need to distinguish gut contents from internal tissue. Lifetime accumulation models generally predict retained mass far below total intake, but their outputs depend on assumed uptake and clearance parameters. A directly measured closed-balance human kinetic fraction is not yet available; this limits precision of uptake estimates but does not prevent risk evaluation using the broader evidence base. (Lee et al. 2023); (Letwin et al. 2026); (Straub et al. 2017); (Garcia et al. 2024); (Deng et al. 2017); (Nor et al. 2021)

The lifetime model (Nor et al. 2021) estimated 40.7 ng retained at age 70 for a 1–10 micrometre model, compared with 14.91 mg calculated external mass across a broader 1–5,000 micrometre intake model. Using the displayed values, the modeled external-intake mass is approximately 366,000 times greater than the modeled retained burden. This is a diagnostic scale comparison only: the models represent different stages and use incompatible size domains, so their ratio is not a measured absorption or retention fraction.

TWO MODELED QUANTITIES – NOT A RETENTION FRACTION

Lifetime external intake and modeled retained burden use different size domains and model stages.



Source context: Nor et al. 2021; Phase 5 synthesis. Values are model outputs with incompatible size domains.

Figure 8. Modeled lifetime external intake versus modeled retained burden by age 70. Using the displayed values, modeled external intake is approximately 366,000 times greater in mass. The two quantities use different size domains and model stages; the comparison is diagnostic scale context, not a measured human absorption or retention fraction. The absence of a directly measured closed-balance human fraction limits precision of uptake estimates; it does not prevent risk evaluation using the full evidence base.

Integrated evidence synthesis

Translocation across biological barriers is strongly size-, shape-, surface- and formulation-dependent. Larger particles are generally expected to remain within the gastrointestinal lumen, while smaller particles may interact with

mucus, epithelial cells or lymphoid structures. Nanometre-scale particles present additional challenges because aggregation, protein coronas and label stability can change the effective exposure during an experiment.

Fluorescent or radiolabelled studies can be informative only when the label remains attached to the particle and free label is quantified. Dye leaching, lipid partitioning or dissolution can create distant-tissue signals without intact-particle translocation. Tissue digestion can also alter particles or increase contamination. Orthogonal confirmation of particle identity and location is therefore more persuasive than signal detection alone (Lee et al. 2023; Letwin et al. 2026; Straub et al. 2017).

Available controlled studies often show that most administered oral material remains in the gastrointestinal tract or is eliminated. Some report low-level distant signals, particularly for smaller particles or specialised preparations. These findings support the possibility of limited uptake under selected conditions but do not define a general human absorption fraction across polymers and sizes.

Controlled inhalation provides a route-specific kinetic anchor for the smallest particles. In Miller et al. 2017, gold served as an effectively inert and analytically distinctive tracer. Systemic gold appeared after inhalation, but transfer was strongly size-dependent and quantitatively small. The authors' correction changed the illustrative 24-hour estimate from approximately 0.2% to approximately 0.02% of inhaled mass; they emphasized that this was not a closed mass balance because alveolar deposition, continued translocation, hepatic clearance and non-urinary routes were not fully captured. The study therefore demonstrates that barrier crossing can occur for ultrasmall nanoparticles without supplying a general absorption fraction for microplastics or nanoplastics of other compositions and sizes.

The selected toxicokinetic model estimated a median retained mass of 40.7 ng at age 70 for 1-10 micrometre particles (Nor et al. 2021). The value is not a measured tissue burden and has a wide uncertainty interval. Comparing it with the modeled 14.91 mg lifetime external intake produces a diagnostic difference of roughly five orders of magnitude, but the input and retained models use different size boundaries. The comparison is therefore a scale check, not a measured retention percentage.

Evidence-weighted interpretation

A valid kinetic study requires a recoverable input dose, time-resolved gastrointestinal and tissue measurements, excretion, method recovery, blank correction and a closing mass balance. Most existing studies measure only one or two stages. An apparent tissue signal without quantified input and excretion cannot yield an absorption fraction, while a faecal signal without whole-output measurement cannot yield clearance.

Several controlled studies nevertheless constrain the plausible kinetic range for larger particles. Fragmented polypropylene in mice remained predominantly gastrointestinal and most fluorescent signal was eliminated within about 24 hours (Lee et al. 2023). Earthworm particles were primarily gut-associated with low accumulation factors and no clear tissue translocation (Letwin et al. 2026). Amphipods ingested and egested both plastic and silica reference particles (Straub et al. 2017). These findings are consistent with limited uptake of larger material, although they do not resolve nanoscale or chronic exposure.

Other mouse experiments report particle- or label-associated signals in distant tissues (Garcia et al. 2024; Deng et al. 2017). Their interpretation depends on whether fluorescence remained particle-bound, whether tissue processing redistributed material, whether the signal was above contamination background and whether the administered bolus resembles environmental exposure. The existence of a distant signal establishes a possible transport pathway under the test conditions; it does not establish a population retention rate.

The Miller tracer study is methodologically stronger for the narrow question of pulmonary translocation because the exposure was controlled, endogenous gold background was negligible, particle size was characterized, and systemic gold was measured with HR-ICPMS while particulate gold in selected tissues was supported by Raman microscopy. Its inference remains size- and material-specific: approximately 4-5 nm primary particles are not kinetically interchangeable with 1-100 micrometre microplastics, and the corrected approximately 0.02% value is an illustrative 24-hour estimate rather than a measured universal uptake fraction.

The lifetime retained-mass estimate of 40.7 ng at age 70 is a model output for a narrower 1-10-micrometre domain, not a measured human burden (Nor et al. 2021). It should be used to identify influential assumptions and research needs, not to back-calculate a measured uptake fraction from the broader external-intake model.

Table 7. Evidence stages for uptake, clearance and retention

Evidence stage	Representative record	Finding	Inference limit
Gastrointestinal clearance	Lee et al. 2023	Most PP-associated signal remained in GI tract and cleared by about 24 h	Fluorescent label and short duration
Gut versus tissue	Letwin et al. 2026	Earthworm particles mainly gut-associated; low accumulation	Model soil/particles and detection limits
Ingestion and egestion comparator	Straub et al. 2017	Plastic and silica ingested and excreted	Species- and particle-specific
Distant-tissue signal	Deng et al. 2017	Signals reported after oral bolus exposure	Label stability, high dose and processing artefacts
Human lifetime model	Nor et al. 2021	40.7 ng retained at age 70 in 1-10-micrometre model	Modeled, not measured; uptake/clearance assumptions dominate
Controlled inhaled nanoparticle translocation	Miller et al. 2017 + correction	Ultrasmall gold nanoparticles entered systemic samples; corrected illustrative 24-h translocation estimate ~0.02%; strong inverse size dependence	Engineered gold tracer and controlled exposure; not a closed mass balance and not transferable to micrometre-scale plastics

Selected evidence records

- **(Low).** Sijoon Lee, Dongseon Kim, Kyung-Ku Kang, et al. 2023. Toxicity and biodistribution of fragmented polypropylene microplastics in ICR mice. *International Journal of Molecular Sciences* 24:8463.
- **(Moderate).** Letwin, A., et al. (2026). Assessing the accumulation of microplastics in earthworms (*Eisenia fetida*) using traditional bioaccumulation modeling and synchrotron-based microcomputed tomography. *Environmental Toxicology and Chemistry*, 45, 1553–1566. <https://doi.org/10.1093/etojnl/vgag072>
- **(Moderate).** Sandrine Straub, Philipp E. Hirsch and Patricia Burkhardt-Holm. 2017. Biodegradable and Petroleum-Based Microplastics Do Not Differ in Their Ingestion and Excretion but in Their Biological Effects in a Freshwater Invertebrate *Gammarus fossarum*.
- **(Moderate).** Garcia, M. A., et al. (2024). In Vivo Tissue Distribution of Polystyrene or Mixed Polymer Microspheres and Metabolomic Analysis after Oral Exposure in Mice. *Environmental Health Perspectives*, 132. <https://doi.org/10.1289/EHP13435>
- **(Moderate).** Yongfeng Deng, Yili Zhang, Bernardo Lemos & Hongqi Ren. 2017. Tissue accumulation of microplastics in mice and biomarker responses suggest widespread health risks of exposure. *Scientific Reports* 7:46687.
- **(Not scored - secondary synthesis).** Nur Hazimah Mohamed Nor, Merel Kooi, Noël J. Diepens, and Albert A. Koelmans. 2021. Lifetime Accumulation of Microplastic in Children and Adults.
- **(Supplementary primary kinetic/context publication lineage outside frozen baseline).** Miller, M. R., Raftis, J. B., Langrish, J. P., et al. (2017). Inhaled Nanoparticles Accumulate at Sites of Vascular Disease. *ACS Nano*, 11(5), 4542-4552. <https://doi.org/10.1021/acsnano.6b08551>; corrected quantitative interpretation in *ACS Nano*, 11, 10623-10624. <https://doi.org/10.1021/acsnano.7b06327>.

13. Human Tissue and Biomonitoring Evidence

The human tissue literature is rapidly expanding, but it remains weak for quantitative body-burden estimation and causal inference; this is one evidence stream within the broader risk evaluation, not the whole evidence base. The version-controlled corpus contains 63 human observational or clinical primary records: two Moderate, 50 Low and 11 Very low. No human observational record is High quality. Matrices include lung, blood, placenta, semen, testis, vascular plaque, brain, stool and other tissues. A supplementary 2026 QA/QC audit independently evaluated 133 datasets from 101 studies and found that none met all 14 essential criteria for direct quantitative use in human-health risk assessment.

Particle-resolved spectroscopy can support occurrence for objects above the method size limit when contamination controls and spectra are adequate. Destructive thermal analysis measures polymer-associated marker mass and does not establish intact particle number, shape, size or tissue location. Lipid-rich matrices can generate false polyethylene signals, and improved multidimensional methods have rejected matrix-related apparent positives.

Clinical and tissue studies may generate important hypotheses, but occurrence cannot establish barrier crossing, accumulation, pathology or causation. The single-donor post-mortem study Dzierżyński et al. 2025 is illustrative: it provides exploratory occurrence and method-development evidence but cannot establish population prevalence or organ-specific bioaccumulation (Amato-Lourenço et al. 2024; Jenner et al. 2022; Zhao et al. 2023; Montano et al. 2023; Marfella et al. 2024; Bissonnette et al. 2025; Dzierżyński et al. 2025; Rauert et al. 2025).

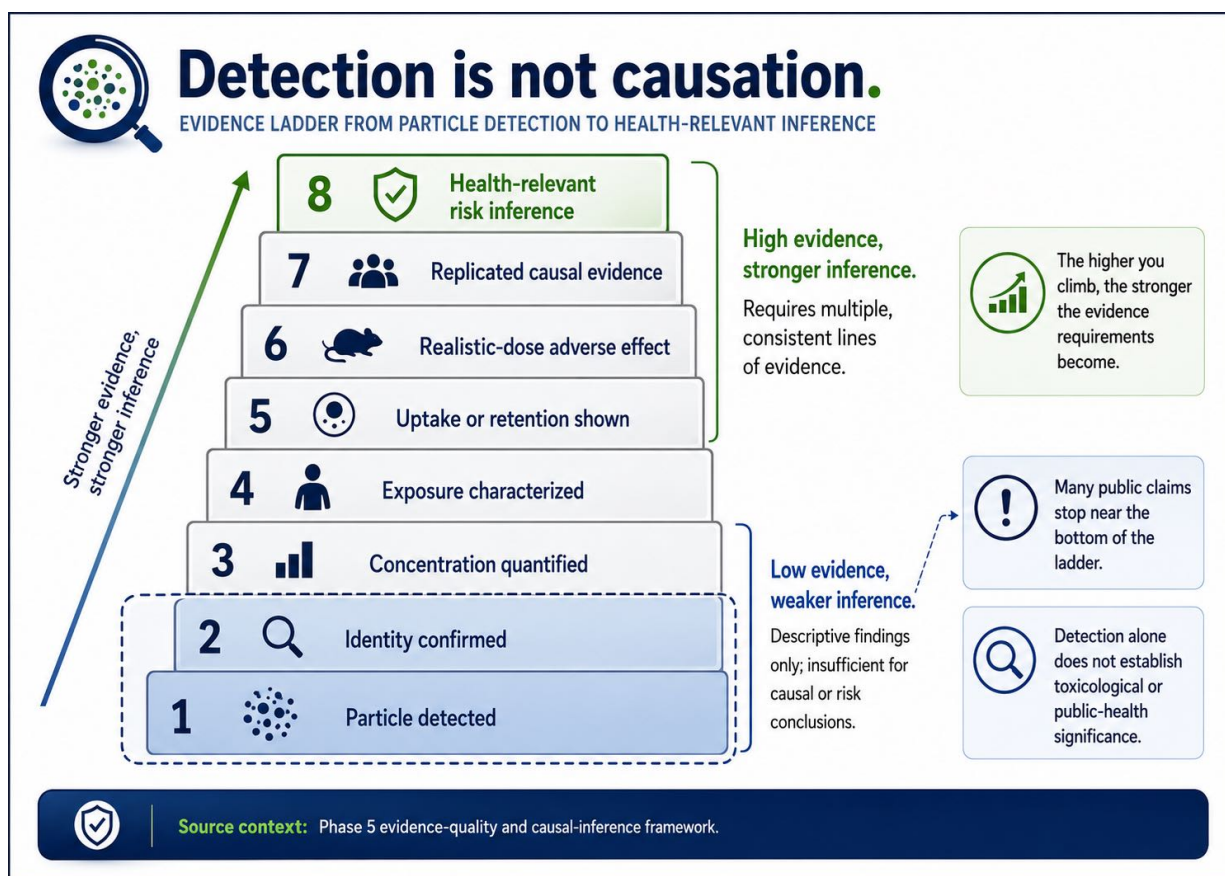


Figure 9. Evidence ladder from particle detection to health-relevant risk inference. Detection and identity are descriptive findings; causal and public-health conclusions require realistic exposure, uptake or retention, adverse effects, causation and independent replication.

Integrated evidence synthesis

Human tissue evidence should be ranked by what the method can establish. Particle-resolved Raman or FTIR can support polymer identity, morphology and approximate size for objects within the validated window. Histological co-location can strengthen anatomical plausibility. Destructive thermal analysis can estimate polymer-associated marker mass but cannot establish whether intact particles were present, their size or their position in tissue.

Fluorescent staining, SEM-EDS carbon morphology and DLS are screening or characterization tools rather than stand-alone proof of plastic identity.

Hayden et al. 2023 provides both a useful method contrast and an important particulate-context lesson. In lung tissue, FE-SEM/EDS resolved the morphology and elemental composition of very large numbers of inorganic particles and distinguished mineral and metal classes including quartz, feldspars, clays, titanium and iron oxides, zircon/zirconia, cerium and other metal-bearing particles. The same result does not make SEM-EDS a polymer-identification method: carbon-rich morphology remains non-specific for plastic. Many particles occurred in macrophage-associated clusters, overlapping particles generated mixed EDS spectra, and thresholding/border rules could undercount particles, so the authors described the method as qualitative rather than a quantitative tissue-burden measurement. The broader implication is nevertheless important: any valid plastic detection in lung tissue should be interpreted within the much wider mixture of nonplastic particles already present, rather than as though plastic were the only foreign particulate material.

Contamination control must extend beyond an empty procedural blank. Surgical materials, collection containers, laboratory air, clothing, filters, digestion reagents and embedding media can introduce polymers. Tissue-specific blanks, field blanks, airborne controls and recovery experiments are needed. Low counts near the blank distribution should be interpreted cautiously, and a result below a study's quantification criterion should not be presented as a precise burden (Jenner et al. 2022; Rauert et al. 2025).

Anatomical plausibility matters. Particles in the gastrointestinal lumen, airway mucus or surface-contaminated tissue do not demonstrate systemic translocation. A claimed particle within a vessel wall, placenta, brain or other protected compartment requires convincing localization and exclusion of processing contamination. Even a valid detection establishes occurrence at the time of sampling, not the exposure route, residence time or cumulative burden.

A controlled tracer experiment shows why particle localization within diseased vascular tissue must not be confused with disease causation. Miller et al. 2017 exposed patients with severe pre-existing carotid atherosclerosis to traceable approximately 5 nm gold nanoparticles only 24 hours before endarterectomy. Raman microscopy subsequently detected particulate gold in excised carotid plaques from the three exposed patients, but not in non-exposed controls. Because the plaques necessarily pre-dated the gold exposure, the study demonstrates directly that circulating nanoparticles can preferentially accumulate at an already diseased vascular site. In the companion atherosclerosis-prone mouse model, gold likewise accumulated preferentially in plaque-rich vascular regions, yet repeated gold administration did not increase plaque size. The study therefore provides a clear causal-inference lesson: finding a particle within an atherosclerotic plaque establishes localization, not that the particle initiated or caused the plaque. By extension, detection of plastic or other particles within another diseased vascular structure, including a thrombus, cannot by itself establish that those particles created the lesion; that would require independent evidence of temporality, dose-response and biological effect. The human plaque arm was small, and the study did not directly test thrombus formation, so the defensible inference is about localization versus causation rather than a universal claim about all clots.

Clinical inference is a separate step. Tissue occurrence cannot by itself establish inflammation, thrombosis, neurotoxicity, reproductive harm or another disease process. The Miller tracer experiment is a particularly strong demonstration of this principle because an inert, identifiable particle accumulated in vascular disease that already existed before exposure. Demonstrating causation for plastic or any other particle would require validated quantitative exposure, temporality, dose-response, specificity or mechanistic mediation, appropriate controls and replication. The current human tissue literature is valuable for hypothesis generation, but localization alone is not a quantitative risk assessment and is not evidence that the detected material created the lesion.

Independent QA/QC audit of human biomonitoring studies

Lane et al. 2026 screened 133 datasets from 101 peer-reviewed human studies. None met all 14 essential criteria for direct quantitative use in health-risk assessment. This means current human biomonitoring is not yet reliable for precise estimates of representative body burden or particle-specific dose-response. It does not mean that the health risk of ordinary microplastic exposure is unknowable, because risk characterization also draws on measured external exposure, toxicology, kinetics, comparative particle evidence and epidemiology.

The dominant weaknesses were not merely incomplete reporting. Approximately 77% of datasets used no positive recovery controls, about 74% reported no LOD/LOQ values, around 71% omitted particle shape, about 44% did not report a size range and nearly half lacked representative sample-description detail. These shortcomings mainly

weaken claims about exact internal burden, organ accumulation and quantitative dose-response; they do not overturn the broader evidence that ordinary external exposure is low and that common-polymer particles do not show exceptional toxicity at relevant doses.

Representativeness is both population- and organ-specific. A small biopsy from one anatomical region should not be multiplied by whole-organ mass unless spatial distribution is known. Region-specific detections may be real yet unsuitable for whole-organ or population burden estimates. The framework therefore supports multi-donor, anatomically defined and, where feasible, multi-region sampling.

Biological plausibility is a separate check. The review identified reports of particles up to 3 mm in blood, greater than 1 mm in vein tissue, 2.5 mm in lung and greater than 4 mm in nasal lavage. Large particles in directly exposed airway fluids are not equivalent to systemic translocation, while compact millimetre-scale objects in blood require exceptional validation of identity, dimensionality, collection and contamination. Unexpected findings should be investigated, not accepted or rejected by assertion.

Human biomonitoring is still developing – but human-health risk can be evaluated from the full evidence base

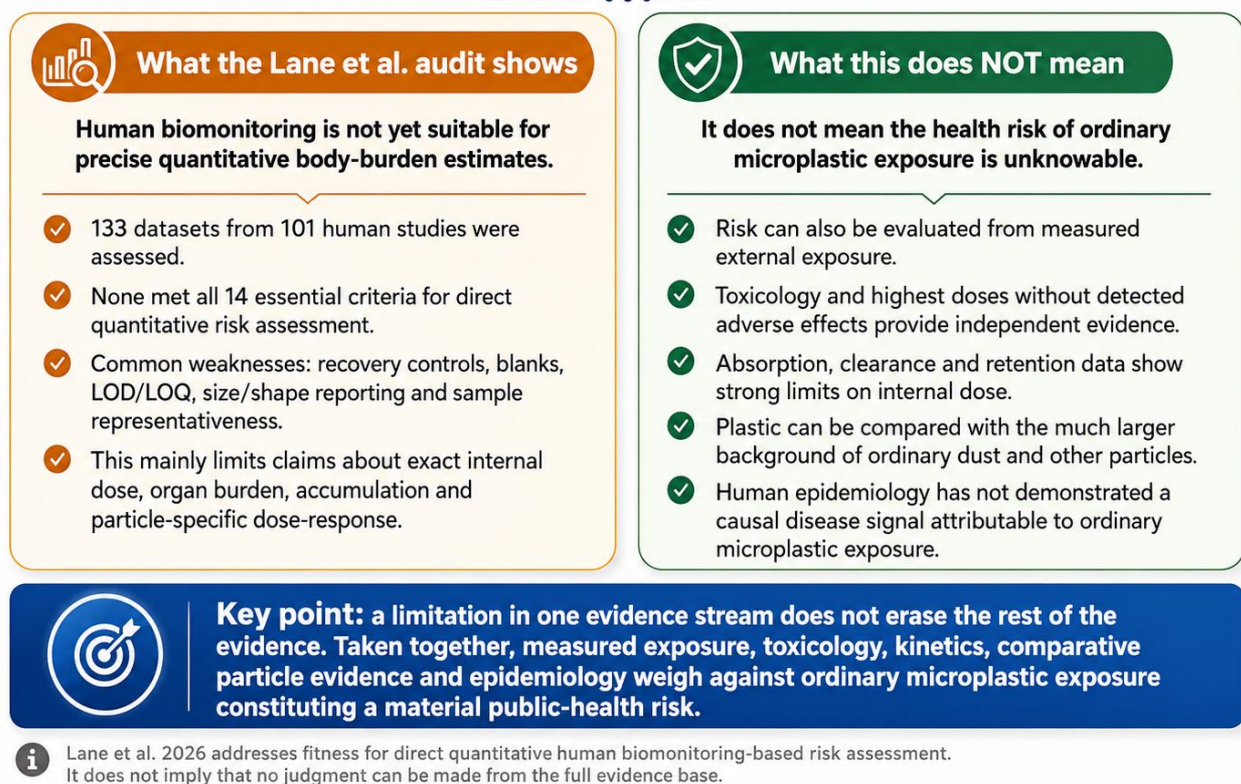


Figure 9A. Human biomonitoring is still developing, but health risk can be evaluated from the full evidence base. Lane et al. 2026 found that none of 133 datasets from 101 human studies met all 14 essential criteria for direct quantitative risk assessment, which limits exact body-burden estimation. This does not mean that risk is unknowable, because measured exposure, toxicology, kinetics, comparative particle evidence and epidemiology provide independent evidence streams.

Selected study-level QA/QC cross-check

Dataset evaluated by Lane et al.	TAS / 28	Zero criteria	Permitted use in this report
Amato-Lourenço 2024 olfactory bulb	23	1	Bounded occurrence; missing positive recovery control prevents quantitative burden use.
Montano 2023 semen	22	2	Bounded occurrence only; representativeness and recovery limitations prevent prevalence or fertility-risk inference.
Jenner 2022 lung	21	1	Plausible deposition in selected tissue; not population burden, retention time or disease.
Zhao 2023 semen / testis	16 / 16	4 / 5	Exploratory reproductive-tissue occurrence; not a quantitative fertility-risk or population-burden estimate.
Nihart 2025 brain / liver / kidney	12 each	6 each	Method-contested polymer-associated mass; not a validated intact-particle organ burden.
Marfella 2024 carotid plaque	11	6	Important prospective clinical association; exposure measure is not fit for quantitative intact-particle dose assessment.

Lane scores assess fitness for direct quantitative risk assessment and are not equivalent to this report's High/Moderate/Low evidence grades. A dataset may support a narrow occurrence statement while remaining unsuitable for representative body-burden estimation. That limitation does not erase the rest of the evidence base or alter the report's overall risk conclusion.

Evidence-weighted interpretation

Human tissue evidence should be ranked by the physical inference each method supports. Particle-resolved FTIR or Raman can support polymer identity, morphology and size above a validated limit. Thermal methods can support targeted polymer-marker mass after destructive analysis but cannot reconstruct intact particle number, size or location. DLS reports hydrodynamic scattering distributions and SEM-EDS reports morphology and elemental composition; neither independently proves polymer identity (Rauert et al. 2025; Bissonnette et al. 2025; Dzierżyński et al. 2025).

The olfactory-bulb and lung studies provide plausible narrow occurrence evidence because they reported spectroscopically assigned objects (Amato-Lourenço et al. 2024; Jenner et al. 2022). Their case-series designs, small samples, limited exposure histories and absence of disease-comparison groups prevent inference of accumulation or neurological and pulmonary harm. A particle found at one time point may reflect recent deposition, persistent retention or contamination; the design cannot distinguish them.

Table 8. Human tissue evidence: permitted and prohibited inferences

Evidence	Narrow finding	Permitted inference	Not established
Olfactory bulb micro-FTIR (Amato-Lourenço et al. 2024)	16 assigned objects in 8 of 15 cases	Some samples contained assigned particles	Neurological injury, accumulation or route
Lung micro-FTIR (Jenner et al. 2022)	39 objects; blank-adjusted mean 0.69/g	Plausible respiratory deposition	Population burden, residence time or disease
Semen Raman (Montano et al. 2023)	Assigned particles in a subset	Bounded occurrence	Fertility effect or exposure source
Atheroma thermal/analytical study (Marfella et al. 2024)	Polymer-related material associated with later events	Clinical association requiring replication	Particle count, mechanism or causation
Single-donor multi-method study (Dzierżyński et al. 2025)	Selected fibres assigned; other non-specific signals	Single-donor descriptive occurrence	Bioaccumulation or nanoplastic barrier crossing
Matrix validation (Rauert et al. 2025; Bissonnette et al. 2025)	False-positive pathways and improved discrimination	Confidence depends on matrix controls	Retrospective validation of unvalidated studies

Reproductive-tissue studies similarly establish bounded occurrence rather than clinical effect. Raman-assigned particles were reported in some semen samples, and polymer-associated material was reported in testis and semen (Montano et al. 2023; Zhao et al. 2023). Neither study reconstructed external exposure, quantified longitudinal internal dose or established a dose-response relationship with fertility.

The single-donor post-mortem study illustrates the danger of combining methods into a stronger claim than any method supports. O-PTIR assignments can support selected fibres, but DLS peaks do not prove nanoplastic identity and MALDI-TOF thermal fragments do not establish intact particle morphology or tissue localization (Dzierżyński et al. 2025). Terms such as bioaccumulation and barrier crossing require repeated or kinetic evidence and should not be inferred from a single cross-sectional donor.

Nihart et al. 2025 reported higher polymer-associated mass in decedent brain than liver or kidney and higher values in dementia cases. The authors described the findings as associative. An author correction addressed errors in supplementary images and methods reporting. The German BfR subsequently judged the study noteworthy but concluded that the reported amounts appear implausibly high and that false or misassigned signals cannot be excluded. The study therefore remains important contrary occurrence evidence, not a validated measure of intact-particle brain burden or proof of neurological harm.

He et al. 2025 analyzed two small cerebrospinal-fluid cohorts and reported four polymer types, with higher PE and PVC measurements in amyloid-positive participants and associations between PE, Alzheimer biomarkers and cognitive change. The study is hypothesis-generating. Small cohorts, disease-state sampling, uncertain exposure history, multiple analyses and biological-matrix method limitations prevent causal interpretation.

Nihart et al. 2025 is now accompanied by a formal Nature Medicine Matters Arising exchange. Monikh et al. 2025 challenged digestion, contamination control, matrix interference and the reliability of polymer-mass assignment in lipid-rich brain tissue. Campen et al. 2025 acknowledged uncertainties in current nanoplastic detection and responded with methodological justifications. The exchange does not nullify the occurrence finding, but it confirms that the reported quantitative brain burden is method-contested and should not be treated as settled intact-particle mass.

Li et al. 2026a analyzed 156 diseased brain samples from 113 living brain-tumour patients and 35 nominally healthy samples from only five postmortem donors. MNP-associated signals were nearly universal, peritumoural concentrations exceeded healthy-brain concentrations, and particle surface area correlated with tumour proliferation. The design cannot establish whether particles preceded disease: tumour-associated blood-brain-barrier disruption, surgical and postmortem differences, unequal donor structure and disease-state retention provide plausible alternative explanations. The authors state that health risks and causal links remain unknown. A subsequent correction addressed small rounding errors without changing the reported text values.

Li et al. 2026b reported Raman-assigned microplastics in matched blood, bone and intervertebral-disc samples from 21 spinal-fusion patients and Py-GC/MS polymer-associated nanoplastic mass in a five-donor subset. The matched design is useful for within-person tissue comparison, but it is a small selected surgical cohort. Py-GC/MS cannot establish nanoplastic size or morphology, and the reported tissue pattern is preliminary rather than kinetic proof of long-term accumulation. The authors explicitly characterize the study as hypothesis-generating and state that long-term toxicity is not established.

Selected evidence records

- **(Low).** Amato-Lourenço, L. F., Dantas, K. C., Ribeiro Júnior, G., Paes, V. R., Ando, R. A., de Oliveira Freitas, R., da Costa, O. M. M., Rabelo, R. S., Soares Bispo, K. C., Carvalho-Oliveira, R., & Mauad, T. (2024). Microplastics in the Olfactory Bulb of the Human Brain. *JAMA Network Open*, 7(9), e2440018. <https://doi.org/10.1001/jamanetworkopen.2024.40018>
- **(Low).** Lauren C. Jenner, Jeanette M. Rotchell, Robert T. Bennett, Michael Cowen, Vasileios Tentzeris & Laura R. Sadofsky. 2022. Detection of microplastics in human lung tissue using micro-FTIR spectroscopy. *Science of the Total Environment* 831:154907.
- **(Moderate).** Zhao, Q., et al. (2023). Detection and characterization of microplastics in the human testis and semen. *Science of the Total Environment*, 877, 162713. <https://doi.org/10.1016/j.scitotenv.2023.162713>
- **(Moderate).** Montano, L., et al. (2023). Raman microspectroscopy evidence of microplastics in human semen. *Science of the Total Environment*, 901, 165922. <https://doi.org/10.1016/j.scitotenv.2023.165922>

- **(Low).** Marfella, R., Prattichizzo, F., Sardu, C., et al. (2024). Microplastics and nanoplastics in atheromas and cardiovascular events. *New England Journal of Medicine*, 390, 900–910.
<https://doi.org/10.1056/NEJMoa2309822>
- **(Moderate).** Bissonnette, J. R., Harvey, N. E., Rowsell, M. L., Kieffe, S., Houthuijs, K. J., Been, F. M., Lamoree, M. H., Cahill, L. S., & Jobst, K. J. (2025). Identification of micro-/nanoplastics in human placental blood using comprehensive multidimensional pyrolysis-gas chromatography × ion mobility mass spectrometry. *Analytica Chimica Acta* 1376, 344606. <https://doi.org/10.1016/j.aca.2025.344606>
- **(Very low).** Dzierżyński, E., Blicharz-Grabias, E., Komanińska, I., Panek, R., Forma, A., Gawlik, P. J., Puźniak, D., Flieger, W., Choma, A., Suśniak, K., Teresiński, G., Baj, J., Kupisz, K., & Flieger, J. (2025). Post-mortem evidence of microplastic bioaccumulation in human organs: insights from advanced imaging and spectroscopic analysis. *Archives of Toxicology*, 99, 4051–4066. <https://doi.org/10.1007/s00204-025-04092-2>
- **(High).** Cassandra Rauert, et al. 2025. Assessing the Efficacy of Pyrolysis-Gas Chromatography-Mass Spectrometry for Nanoplastic and Microplastic Analysis in Human Blood. *Environmental Science & Technology* 59:1984–1994.
- **(Supplementary source outside the frozen baseline).** Lane, T., de Ruijter, V. N., Wardani, I., Barchiesi, M., & Koelmans, A. A. (2026). A QA/QC screening framework to assess studies reporting microplastic presence in human organs, tissues, and bodily fluids. *Environment International*, 214, 110429.
<https://doi.org/10.1016/j.envint.2026.110429>

14. Analytical Reliability and False Positives

Analytical reliability is the central determinant of confidence. Interlaboratory comparison shows substantial platform-, laboratory- and workflow-dependent variability. Soil, wastewater, water and biological matrices each require different extraction and cleanup procedures, and no single method simultaneously measures particle number, morphology and total polymer mass across the full size range.

False-positive pathways include airborne and reagent contamination; polymer-containing laboratory materials; lipid and tissue pyrolysis products; incomplete digestion; spectral-library mismatch; stain-positive nonplastics; carbon-rich SEM-EDS morphology; and interpreting DLS hydrodynamic peaks as polymer-identified nanoplastics. Thermal marker mass must not be presented as intact particle count or location without orthogonal evidence.

The database also contains high-quality evidence that improved methods can reduce error. Matrix-matched cleanup, tandem mass spectrometry, isotope standards, ion mobility, recovery experiments, reagent-specific blanks and orthogonal microscopy or spectroscopy materially increase confidence. (Ciornii et al. 2025); (Rauert et al. 2025); (Rauert et al. 2022); (Liu et al. 2024); (Bissonnette et al. 2025); (Jüngling 2026); (Peneva 2025); (Lares 2019)

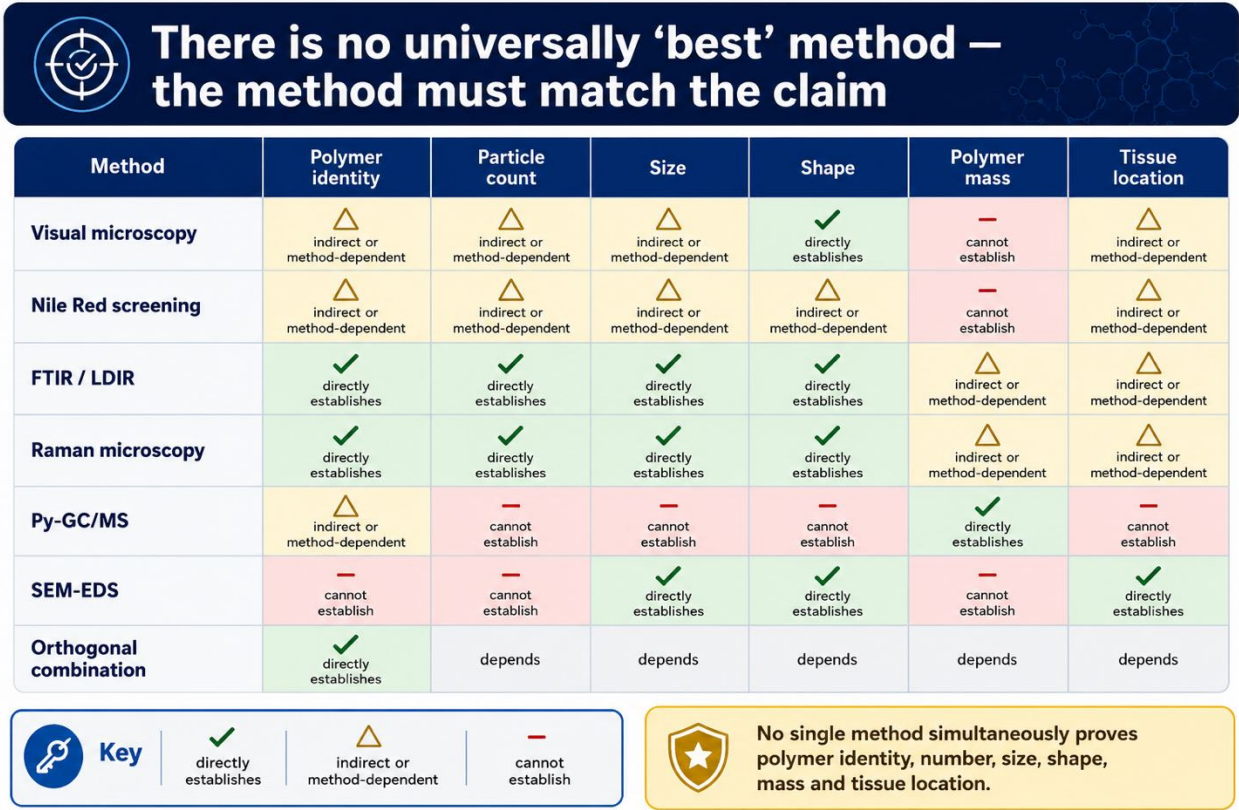


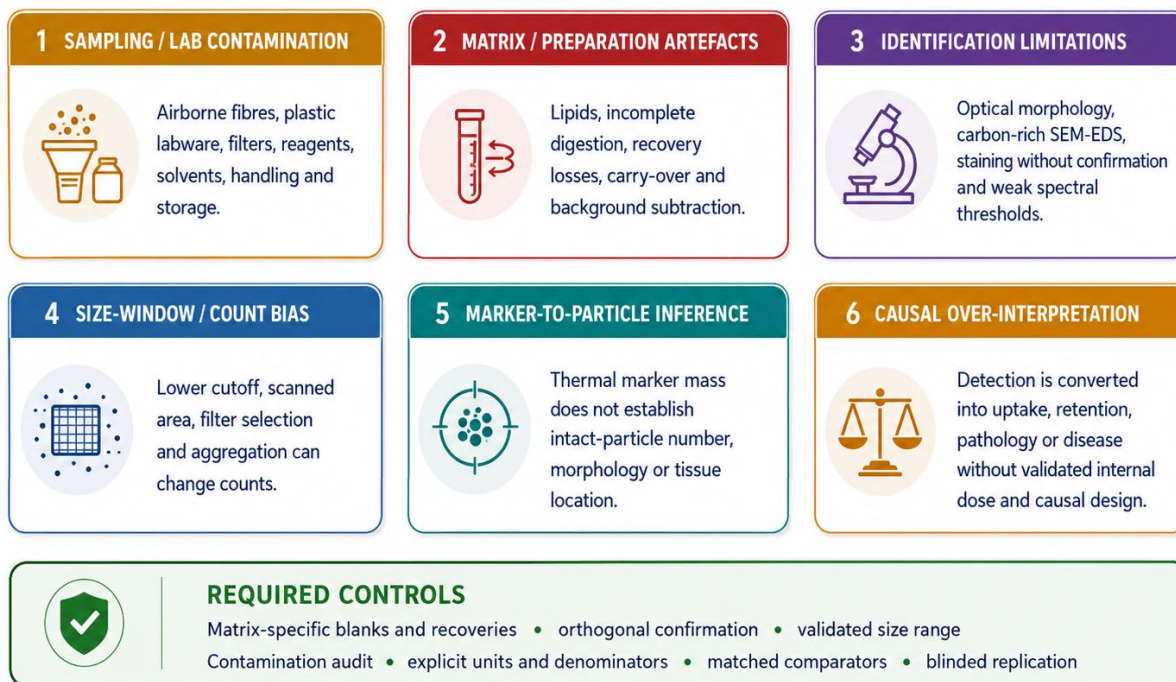
Figure 10. Analytical-method capability matrix. Different methods answer different questions. Raman and FTIR can directly support polymer identity, approximate count, size and morphology within validated size windows; Py-GC/MS supports polymer-associated mass but not intact-particle number, size, shape or location. No single method establishes every required property at once.

Selected analytical windows span 1-5000 micrometres in an intake model, 10-100 micrometres in a Danish tap-water subset, greater than 15 micrometres in Spanish bottled water, at least 45 micrometres in protein foods and 50-500 micrometres in human stool. Pooling particle counts across these windows would be invalid. Count-to-mass conversion is defensible only when dimensions, shape, density, polymer assignment and the geometric model are reported and propagated with uncertainty.



Analytical false-positive and over-interpretation pathways

The strongest evidence chain anticipates measurement artefacts and causal overreach.



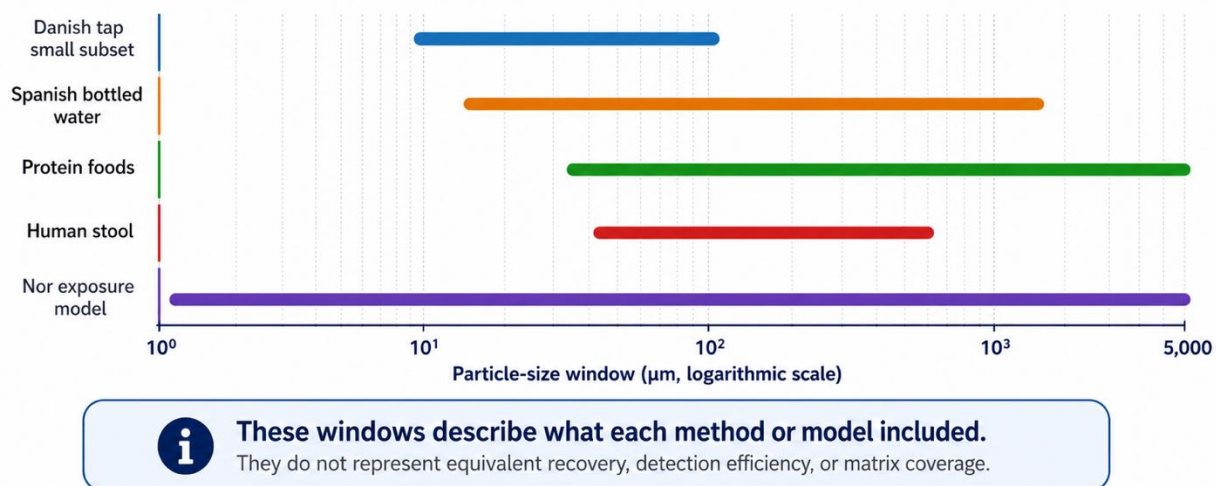
Grounded in certified method and QA records including STUDY-00466, STUDY-01619, STUDY-01622, STUDY-01624 and STUDY-01654.

Figure 11. Analytical false-positive and over-interpretation pathways.



Lower-size cutoffs preclude simple count comparisons

A count is inseparable from the analytical size window and sampling sensitivity.



Selected windows: 1–5,000 μm model; 10–100 μm Danish tap; >15 μm Spanish bottled water; ≥ 45 μm protein foods; 50–500 μm human stool.

Figure 12. Selected analytical particle-size windows demonstrate why raw particle counts cannot be pooled across studies with different lower-size limits and denominators.

Analytical method interpretation

Method	What it can establish	Principal limitation	Required controls
Raman / FTIR microscopy	Polymer identity and morphology for particles within validated spectral and size range	Limited throughput and lower size limit; spectral quality and library rules matter	Blanks, recovery, spectral acceptance criteria, representative scanning
Py-GC/MS and related thermal methods	Polymer-associated marker mass after destructive analysis	Does not establish intact particle count, shape, size or location; matrix interference	Matrix cleanup, isotope standards, blanks, recovery, orthogonal confirmation
Nile Red / fluorescence	Rapid screening of hydrophobic particles	Stains nonplastics and requires confirmation	Procedural blanks and spectroscopic confirmation
SEM-EDS	Morphology and elemental composition; strong classification of inorganic mineral and metal PM	Carbon-rich morphology is not polymer-specific; clustered particles can yield mixed spectra and source origin may remain ambiguous	Orthogonal polymer identification for plastic claims; standards/QA and spectrum review for inorganic classification
DLS	Hydrodynamic size distribution of suspended scatterers	Does not identify polymer and is sensitive to aggregation	Chemically specific confirmation and validated matrix
Optical microscopy	Count and morphology above visual limit	Cannot establish plastic identity alone	Chemical confirmation of representative or all particles

Human-biomonitoring QA/QC requirements

Lane et al. 2026 converts the broad analytical principles used in this report into 14 human-sample criteria. For quantitative risk assessment, a credible dataset must document what and how much was sampled, donor and anatomical context, collection and digestion; laboratory contamination controls; multiple blanks with LOD/LOQ and transparent correction; recovery controls; and particle-resolved size, count, polymer identity and shape. Technique labels alone do not confer reliability.

Positive recovery controls were the weakest criterion: 110 of 133 datasets scored zero. The paper also notes that Py-GC/MS can be useful for polymer-associated mass but cannot by itself provide particle size, count or shape, and that lipid pyrolysis can generate polyethylene marker compounds. This independently corroborates the report's marker-to-particle and matrix-interference cautions.

Merely stating that blanks were included is insufficient. The number and type of blanks, individual results or summary distribution, LOD/LOQ and the correction rule should be reported. Clean-air or laminar-flow handling, filtered reagents, non-synthetic clothing and plastic-free protocols reduce contamination but do not replace workflow-complete blanks or recovery controls.

Integrated evidence synthesis

Analytical reliability begins with a precise measurand. A study may seek particle number above a defined size, polymer mass, morphology, surface chemistry or a screening signal. Results become misleading when the method measures one quantity and the interpretation claims another. In particular, polymer-associated pyrolysis markers cannot be converted into intact particle counts without an independently measured size and shape distribution.

A credible workflow reports sampling materials, field and procedural blanks, contamination prevention, matrix digestion, filter recovery, spike recovery, spectral acceptance criteria, library construction, detection and quantification limits, and the proportion of objects chemically confirmed. Blank subtraction should reflect the distribution and identity of contamination, not merely subtract a mean count that may produce negative or unstable values.

Raman and FTIR are particle-resolved but have throughput, fluorescence and lower-size limitations. Thermal methods can access total polymer-associated mass and smaller material but destroy morphology and can suffer matrix-specific interferences. Nile Red is useful for screening but stains other hydrophobic material. SEM-EDS provides morphology and elemental information, but carbon is not polymer-specific. DLS reports scattering-derived hydrodynamic size, not chemical identity (Ciornii et al. 2025; Rauert et al. 2022; Liu et al. 2024).

Interlaboratory exercises and matrix-validation studies show that analyst choices, spectral thresholds and extraction methods can materially change results. Reliability therefore depends on validated performance in the actual matrix and size range. A method that works for reference particles in clean water should not be assumed valid for blood, adipose tissue, placenta or complex food without recovery and interference testing.

The practical conclusion is selective confidence. Well-controlled, particle-resolved results above the validated cutoff can be credible. Screening-only or marker-only results may still be useful, but their claims must be bounded to the quantity actually measured. This approach avoids both blanket acceptance and blanket rejection of the field.

Evidence-weighted interpretation

Interlaboratory evidence shows that analytical uncertainty can be of the same order as reported environmental differences. A multi-laboratory comparison found method-, preparation-, classification- and laboratory-dependent variation (Ciornii et al. 2025). Wastewater/sludge and soil comparisons reached the same general conclusion in complex matrices (Lares 2019; Peneva 2025). Method validation is therefore part of the result, not a separate technical detail.

Aves et al. (2026) applied five analytical methods to the same remote-air samples. Every method produced a different concentration and polymer profile; 98% of particle-resolved detections were below 10 micrometres, and Nile Red counts exceeded micro-Raman counts by more than 13-fold. This is direct evidence that method, lower-size limit, measurand, calibration and calculation determine the reported result and that unlike methods should not be pooled.

Jüngling et al. (2026) achieved automated Raman analysis down to 500 nm, with $67 \pm 10\%$ recovery for 500 nm polystyrene beads. Illumination settings overestimated particle size by approximately 0.5 ± 0.26 micrometres, and analysis of each 500 mL potable-water sample required about three days. The authors explicitly treated the water application as proof of principle rather than a general estimate of drinking-water composition.

Clough et al. (2026) demonstrated that dry contact with common nitrile and latex gloves can deposit stearate-rich residues that spectral-library matching misclassifies as microplastics. Traditional library matching produced a mean of approximately 2,000 apparent false positives per square millimetre across seven glove types, compared with approximately 100 per square millimetre for a cleanroom nitrile glove. Complete procedural blanks, direct-contact controls and glove-free handling are therefore essential where small-particle identification is conclusion-controlling.

False-positive pathways are empirical. Lipid-rich matrices can generate pyrolysis products used as polyethylene markers, producing large overestimation without adequate cleanup (Rauert et al. 2022). Blood-method validation documented continuing difficulty for polyethylene- and PVC-related signals (Rauert et al. 2025). Multidimensional pyrolysis with ion mobility rejected matrix-related apparent polyethylene signals in placental blood (Bissonnette et al. 2025). These studies do not show that every thermal result is false; they show that matrix validation, isotope standards, recoveries and orthogonal confirmation determine confidence.

Reagents and solvents can contribute micro- and nanoplastic contamination, so procedural blanks must represent the complete workflow rather than only laboratory air or glassware (Liu et al. 2024). A reported blank value is also insufficient unless the subtraction rule, detection limit and uncertainty are stated.

Selected evidence records

- **(High).** Dmitri Ciornii, Vladimir-Dan Hodoroaba, Nizar Benismail, et al. 2025. Interlaboratory Comparison Reveals State of the Art in Microplastic Detection and Quantification Methods. *Analytical Chemistry* 97:8719-8728.

- **(High)**. Cassandra Rauert, et al. 2025. Assessing the Efficacy of Pyrolysis-Gas Chromatography-Mass Spectrometry for Nanoplastic and Microplastic Analysis in Human Blood. *Environmental Science & Technology* 59:1984–1994.
- **(Moderate)**. Cassandra Rauert, Yufei Pan & Kevin V. Thomas. 2022. Extraction and Pyrolysis-GC-MS analysis of polyethylene in samples with medium to high lipid content. *Journal of Environmental Exposure Assessment* 1:13.
- **(High)**. Yingnan Liu, Jie Han, Yanhua Wang, Aoze Li, Jian Zhao, Yu Su, Lezu Shen & Baoshan Xing. 2024. Suspected sources of microplastics and nanoplastics: Contamination from experimental reagents and solvents. *Water Research* 249:120925.
- **(Moderate)**. Bissonnette, J. R., Harvey, N. E., Rowsell, M. L., Kieffe, S., Houthuijs, K. J., Been, F. M., Lamoree, M. H., Cahill, L. S., & Jobst, K. J. (2025). Identification of micro-/nanoplastics in human placental blood using comprehensive multidimensional pyrolysis-gas chromatography × ion mobility mass spectrometry. *Analytica Chimica Acta* 1376, 344606. <https://doi.org/10.1016/j.aca.2025.344606>
- **(High)**. Jüngling, I. S., Schmitt, L., De Franceschi, F., et al. (2026). Challenges and solutions in the analysis of micro- and nanoplastics down to 500 nm with automated Raman microspectroscopy: suitable filters, accuracy in detection, identification and quantification. *Analytical and Bioanalytical Chemistry*. <https://doi.org/10.1007/s00216-026-06567-2>
- **(Supplementary source outside the frozen baseline)**. Aves, A., MacDonald, A., Naeher, S., et al. (2026). Comparative assessment of five analytical methods for airborne microplastics highlights importance of identifying sub-10 micrometre methods. *Scientific Reports*, 16, 22321. <https://doi.org/10.1038/s41598-026-49405-2>
- **(Supplementary source outside the frozen baseline)**. Clough, M. E., et al. (2026). Avoiding and reducing microplastic false positives from dry glove contact. *Analytical Methods*, 18, 2914-2926. <https://doi.org/10.1039/D5AY01801C>
- **(High)**. Stoyana Peneva. 2025. Microplastic analysis in soils: A comparative assessment. *Ecotoxicology and Environmental Safety*, 289 (2025) 117428.
- **(High)**. Mirka Lares. 2019. Intercomparison study on commonly used methods to determine microplastics in wastewater and sludge samples. *Environmental Science & Pollution Research*, 26, 12109–12122. <https://doi.org/10.1007/s11356-019-04584-6>

15. Animal Toxicology

Animal and cell studies demonstrate that microplastic preparations can produce biochemical, inflammatory, histological, behavioral and functional effects under selected conditions. However, external validity varies widely. Common limitations include pristine spherical particles, bolus oral dosing, concentrations far above measured exposure, short duration, small groups, multiple endpoints and no matched nonplastic-particle comparator.

Positive hazard studies should be retained, but their inference must be bounded. A response at 300 to 1,000 mg/kg or approximately 10 mg/kg/day is not direct evidence of risk from trace environmental intake. Conversely, null findings at high dose are informative for bounding hazard even though they cannot prove the absence of every chronic, nanoscale or formulation-specific effect. (Deng et al. 2017); (Stock 2021); (Fatima et al. 2026); (Lin et al. 2025); (Ilechukwu et al. 2022); (Bacchetta et al. 2024); (Horn et al. 2020)

Integrated evidence synthesis

Animal toxicology spans acute, subchronic, reproductive, developmental, inflammatory and organ-specific endpoints. Study interpretation requires attention to polymer, particle size distribution, shape, surface condition, additives, dose metric, administration route, dispersion medium and duration. A nominal concentration is insufficient when particles agglomerate, settle or adhere to dosing equipment.

Many positive studies use gavage or bolus administration at concentrations remote from ordinary environmental exposure. Such studies can identify a hazard mechanism, but they do not establish that the mechanism operates at realistic continuous exposure. High dose can also cause generic particle overload, altered nutrition, physical abrasion or vehicle effects. Matched mineral, cellulose or other low-toxicity particle controls are needed to determine whether an effect is plastic-specific (Deng et al. 2017; Stock 2021; Fatima et al. 2026).

Pristine monodisperse spheres are useful experimental tools but do not represent the diversity of weathered fragments and fibres. This distinction is particularly important for polystyrene: many widely cited mammalian PS studies use commercial or synthesized spheres, while other high-dose studies do not yield a clean no-adverse-effect level for fragmented or weathered material. The 2020 Kim conference-abstract title identifies acute oral PP and PS testing, but the accessible source does not verify dose, material preparation, endpoints or results. The 2021 full paper is a different PP-only study and must not be used as PS evidence. Accordingly, no verified repeated-dose oral NOAEL is assigned to real fragmented/weathered PS in this report. Conversely, environmental particles may carry additives, biofilms or sorbed chemicals that confound attribution to the polymer. Studies using commercial fluorescent particles must test dye leaching and free-dye controls, particularly when distant-tissue fluorescence is a central endpoint.

Null findings are scientifically informative. They define conditions under which adverse effects were not detected and constrain claims of extreme potency. They should be interpreted with the same attention to statistical power, endpoint sensitivity and duration as positive findings. The combined animal evidence establishes hazard potential under selected conditions but does not support a single toxicity value for all microplastics.

Evidence-weighted interpretation

Animal-toxicology evidence should be separated into standard apical toxicity, mechanistic biomarkers and exploratory pathology. Changes in oxidative-stress markers, gene expression or metabolomics can identify a biological response, but they are not automatically adverse outcomes. Standard clinical observations, growth, organ weights, haematology, chemistry and histopathology provide a different evidentiary tier and should not be displaced by a large number of statistically significant molecular endpoints.

Several prominent adverse studies use dose scales far above ordinary exposure. An eight-week rat study reported effects concentrated at 1,000 mg/kg, with several outcomes not significant at 300 mg/kg (Fatima et al. 2026). An atherosclerosis study used a disease-prone mouse model and a regimen around 10 mg/kg/day without an equal-dose nonplastic comparator (Lin et al. 2025). A mouse accumulation study and an in-vitro multi-polymer study also used simplified preparations and high concentrations (Deng et al. 2017; Stock 2021). These studies support hazard mechanisms under the administered conditions, not direct estimates of ordinary human risk.

Supplementary contrary evidence outside the frozen baseline: Sánchez et al. (2026) exposed female BALB/c mice by oral gavage to aged or non-aged PVC packaging microplastics at 0.02 mg/day, reported as 1 mg/kg body

weight, for 70 days. The authors reported oxidative-stress and inflammatory changes, altered intestinal histology, microbiota composition and short-chain fatty acids. This is important positive animal evidence because it uses irregular packaging-derived and aged particles rather than only pristine microspheres. It does not establish ordinary human risk: the administered dose is approximately 120,000 times the selected modeled adult external mass exposure on a body-weight basis, gavage differs from diffuse dietary contact, one sex and species were tested, and no matched nonplastic-particle comparator isolated PVC-specific effects. DOI: 10.1016/j.tox.2026.154427.

Two oral studies reporting pulmonary granulomatous findings require separate treatment from the stronger no-adverse-effect anchors. Lee et al. (2022) reported lung findings after 28-day gavage of fragmented PE at 500–2,000 mg/kg/day; conventionally, 500 mg/kg/day is the male highest tested dose without the reported lung finding, while a female NOAEL was not established because findings occurred at the lowest 500 mg/kg/day dose. Kim et al. (2025) reported PET NOAELs of 1.75 mg/kg/day in males and 7 mg/kg/day in females. Those PET values are not used as principal quantitative anchors because the presumed particles in lesions were not chemically co-localized, pooled-lung Raman did not identify the particles inside the lesions, pathology was not quantitatively scored, and only one slide per organ was examined. The PET toxicity arm appears to have used unlabelled particles; Cy5.5-COOH/DMSO-labelled PET was used in a separate biodistribution arm. Residual dye or DMSO therefore cannot be asserted as the cause of the toxicity finding, but the biodistribution interpretation remains limited because free dye, residual solvent and dye leaching were not adequately excluded.

New PET evidence broadens but does not simplify the dose-response picture. Lu et al. (2025) gavaged male mice with irregular 6–12 micrometre PET at 5, 50 or 500 micrograms/day for 17 weeks; the authors reported no significant liver pathology or liver-function changes at 5 micrograms/day, but concentration-related hepatic injury at 50 and 500 micrograms/day. Lewis et al. (2026) reported mild, sex-dependent and partly non-monotonic metabolic, platelet, LDL and duodenal gene-expression changes during approximately 18 hours after a single 5 or 50 mg/kg gavage. Mierzejewski et al. (2025) exposed approximately 20 kg piglets to 0.1 or 1 g/day PET for four weeks; the low dose changed one pancreatic gene, while the high dose changed 86 genes and oxidative-stress indicators. These studies are useful hazard evidence, but none supplies a robust general PET NOAEL: endpoint adversity, duration, dose metric, sex, sample size, publication lineage and the absence of matched nonplastic particles remain limiting.

Selected evidence records

- **(Moderate)**. Yongfeng Deng, Yili Zhang, Bernardo Lemos & Hongqi Ren. 2017. Tissue accumulation of microplastics in mice and biomarker responses suggest widespread health risks of exposure. *Scientific Reports* 7:46687.
- **(Moderate)**. Valerie Stock. 2021. Uptake and cellular effects of PE, PP, PET and PVC microplastic particles. *Toxicology in Vitro*, 70 (2020) 105021.
- **(Very low)**. Fatima, R., Parveen, K., Anjum, S., Abduh, M. S., Zaman, A., Naseer, F., & Ahmad, T. (2026). Dose-Dependent Toxicological Effects of Polyvinyl Chloride and Polystyrene Microplastics on Wistar Albino Rats. *Journal of Applied Toxicology*. <https://doi.org/10.1002/jat.70080>
- **(Low)**. Ting-An Lin, et al. 2025. Microplastic exposure elicits sex-specific atherosclerosis development in lean low-density lipoprotein receptor-deficient mice. *Environment International*:109938.
- **(Low)**. Ilechukwu, I., Ehigior, B.E., Ben, I.O., Okonkwo, C.J., Olorunfemi, O.S., Modo, U.E., Ilechukwu, C.E., & Ohagwa, N.J. (2022). Chronic toxic effects of polystyrene microplastics on reproductive parameters of male rats. *Environmental Analysis Health and Toxicology*, 37(2), e2022015. <https://doi.org/10.5620/eaht.2022015>
- **(Moderate)**. Bacchetta, R., Pica, A., Santo, N., Tremolada, P., & Sugni, M. (2024). Comparative effects of polyvinyl chloride microplastics on the brittle star *Ophiactis virens* and the amphibian *Xenopus laevis*. *Aquatic Toxicology*, 272, 106975. <https://doi.org/10.1016/j.aquatox.2024.106975>
- **(Moderate)**. Horn, D. A., Granek, E. F., & Steele, C. L. (2020). Effects of environmentally relevant concentrations of microplastic fibers on Pacific mole crab (*Emerita analoga*) mortality and reproduction. *Limnology and Oceanography Letters*, 5(1), 74–83. <https://doi.org/10.1002/lol2.10137>
- **Supplementary source** outside the frozen baseline. Sánchez, A., Ríos, D., Martínez, M., Monedero, V., Zúñiga, M., Vélez, D., & Devesa, V. (2026). Microplastic-induced alterations in the intestinal environment: Aging as a modulating factor. *Toxicology*, 522, 154427. <https://doi.org/10.1016/j.tox.2026.154427>

16. Standardized Oral Toxicology and Highest Doses Without Detected Adverse Effects

Standardized toxicology is sparse but disproportionately informative. The strongest available oral no-adverse-effect anchors include a 94-96-day cellulose-acetate dietary study, a 13-week OECD/GLP dietary study of cryogenically milled PE/PET fabric, a GLP OECD Test Guideline 407 PA-6 limit study, repeated-dose fragmented polypropylene and PTFE studies, and a GLP weathered-fragmented-PP study. Newer and archival evidence adds a 90-day standalone-PE lead, a lifespan 10% VCM-free PVC control diet, source-recovered historical PTFE studies and acute and chronic PET studies. Source-level correction confirms that Kim et al. (2021) tested PP only; the separate 2020 conference abstract naming PP and PS is insufficiently reported for a numerical PS value. Otaka et al. (1978) tested PET chloroform extracts rather than PET particles and is therefore treated as extractables toxicology. These datasets differ in verification, material, preparation, dose metric and design and cannot be collapsed into one universal threshold.

The PE/PET fabric study found no apparent target-organ toxicity at the highest target concentration of 5% of the diet (4.7% measured). For a 50:50 PE/PET composition, this equals 2.5% target PE plus 2.5% target PET, or approximately 2.35% measured of each component. Using the reported food consumption and body-weight trajectory, each polymer was ingested at approximately 1.6-2.0 g/kg-bw/day. This is a component exposure within a tested mixture, not an independently derived pure-PE or pure-PET NOAEL (Merski et al. 2008).

Other selected values include a formal study NOAEL of 5,000 mg/kg/day for cellulose acetate, the highest dose tested over 94-96 days; a formal study NOAEL of 25 mg/kg/day for weathered fragmented polypropylene, the highest feasible dose actually tested; 1,000 mg/kg/day without treatment-related local or systemic toxicity for PA-6; and author-reported NOAELs of at least 2,000 mg/kg/day for fragmented polypropylene and PTFE. The Kim et al. (2021) abstract states that the NOAEL is higher than 25 mg/kg/day, whereas the paper's conclusion states 25 mg/kg/day. Conventional NOAEL usage supports 25 mg/kg/day because no higher dose was tested. Lower and conflicting PE/PET findings are retained. Lu et al. 2025 adds a longer PET dose-response in which 5 micrograms/day produced no significant liver pathology or liver-function change, while 50 and 500 micrograms/day produced increasing hepatic effects. None of the lower PET findings is treated as an equivalent principal quantitative anchor because of differences in endpoint adversity, study design, pathology verification, delivery and dose metric (Thomas et al. 1991; Kim et al. 2021; Buesen et al. 2025; Lee et al. 2022; Lee et al. 2023; Kim et al. 2025; Lu et al. 2025).

Terminology rule: a formal NOAEL is the highest tested dose at which no adverse effect was observed under the stated study conditions. A study may suggest that the true threshold is higher, but an untested dose must not be presented as an experimentally demonstrated NOAEL.

Selected standardized and study-specific oral toxicity evidence

Study / citation	Material, preparation and design	Dose and dosing	Highest dose without detected adverse effects / formal term	Limitations and exact permitted inference
Kim et al. 2021 (PP)	Bulk PP resin; cryomilled, sieved and heat/UV-B weathered; irregular fragments, mean 85.2 µm; GLP OECD TG 407; rats; 28 days	0, 6.25, 12.5 and 25 mg/kg/day; 25 maximum feasible	Formal study NOAEL = 25 mg/kg/day, the highest tested dose; no treatment-related apical or micronucleus effects	Abstract says >25, but the conclusion and conventional interpretation support 25; a higher threshold was not tested
Buesen et al. 2025	Irregular PA-6; GLP OECD TG 407; rats; 28 days	Limit dose 1,000 mg/kg/day	No treatment-related local or systemic toxicity at 1,000 mg/kg/day	Single limit dose and narrow preparation; descriptive highest no-adverse-effect dose unless source terminology is used
Merski et al. 2008	Cryogenically milled 50:50 PE/PET spunbond fabric; OECD/GLP rat diet; 13 weeks	0.5, 2.5 and 5% target diet; high diet measured 4.7%	Mixture: no apparent target-organ toxicity at 5%; each component 2.5% target (~2.35% measured), approximately 1.6-2.0 g/kg/day	Particle dimensions not measured; PE and PET were not tested separately; component values apply only within the mixture

Study / citation	Material, preparation and design	Dose and dosing	Highest dose without detected adverse effects / formal term	Limitations and exact permitted inference
Lee et al. 2022 (PTFE)	Fragmented PTFE, ~5 and 10-50 µm; mice; oral gavage; 28 days	500, 1,000 and 2,000 mg/kg/day	Author-reported NOAEL at least 2,000 mg/kg/day for both particle sizes	Four-week, formulation-specific study; blood Raman non-detection does not quantify uptake below the method limit
Lee et al. 2022 (PE)	Fragmented PE, 10-50 µm; mice; oral gavage; 28 days	500, 1,000 and 2,000 mg/kg/day	Male: 500 mg/kg/day without reported lung finding; female NOAEL not established because finding occurred at 500 mg/kg/day	Lung particles were presumed rather than lesion-co-localized; author wording "NOAEL <1,000/<500" is nonstandard
Kim et al. 2025 (PET)	Fragmented PET, mean ~5.6 µm; mice; oral gavage; 28 days	1.75, 7 and 28 mg/kg/day	Authors reported 1.75 mg/kg/day in males and 7 mg/kg/day in females; not used as a principal anchor	Very low quantitative confidence: lesion identity/co-localization and pathology quantitation inadequate; labelled biodistribution arm was separate
Lee et al. 2023	Fragmented PP, ~5 and 10-50 µm; mice; 28-day toxicity arm	500, 1,000 and 2,000 mg/kg/day	Author-reported NOAEL at least 2,000 mg/kg/day; no standard toxicological changes	Short duration; separate fluorescent arm informs clearance but does not define the toxicity threshold
Lefaux 1968 / Phillips & Marks 1961 (PE; secondary chain)	Standalone commercial Marlex 50 PE dietary study in rats; 90 days; summarized by Lefaux 1968, citing Phillips & Marks 1961	1.25, 2.5 and 5% diet (secondary report)	No adverse effects reported at 5% diet; archival highest tested concentration, not a verified formal NOAEL	Secondary-source evidence; original Phillips & Marks article, formulation, particle size, methods, endpoints and dose conversion remain unverified
Feron et al. 1981 (PVC control)	Carina S 65-02 PVC powder; VCM-free <0.3 ppm; Wistar rats; lifespan VCM study	10% PVC powder in the control diet; males to 135 wk, females to 144 wk	No PVC-polymer NOAEL derived; supportive chronic tolerance context at 10% diet	Designed to test VCM, not PVC; ad-lib extra control lacked histology; resin/formulation-specific
Lu et al. 2025 (PET)	Irregular PET, ~6-12 µm; male ICR mice; oral gavage; 17 weeks	0, 5, 50 and 500 µg/day	No significant liver pathology/function change at 5 µg/day; adverse liver findings at 50-500 µg/day; no formal NOAEL assigned	Low dose increased liver coefficient; one sex, non-guideline gavage, limited endpoints and no matched particle comparator
Lewis et al. 2026 (PET)	Cryomilled PET nurdles; male/female Sprague-Dawley rats; single gavage; ~18 h	0, 5 or 50 mg/kg once	No repeated-dose NOAEL; mild sex-dependent and partly non-monotonic metabolic, platelet, LDL and gene-expression changes	Acute short-duration mechanistic study; biomarker adversity uncertain; no matched nonplastic particles
Mierzejewski et al. 2025 (PET)	PET 7.6-416.9 µm; ~20 kg piglets; oral capsules; 4 weeks; n=5/group	0.1 or 1 g/day (~5 or 50 mg/kg/day)	No formal NOAEL; one pancreatic gene changed at low dose and 86 at high dose; oxidative-stress changes reported	Transcriptomic/molecular endpoints, small groups, no matched particles and shared pig-study lineage; not apical toxicity
Thomas et al. 1991 (cellulose acetate)	Cellulose acetate; CD Sprague-Dawley rats; dietary admixture; 94-96 days	500, 2,500 and 5,000 mg/kg/day	Formal subchronic NOAEL = 5,000 mg/kg/day, the highest dose tested	Semi-synthetic thermoplastic; high dose was approximately 7.2-10% of diet and reduced nutritional efficiency; not a weathered microplastic preparation
Haskell Reports 56-61 and 224-68 (PTFE)	Multiple PTFE powders; rats; 25% diet for 3 weeks and 10% diet for 21 days; residual dispersants varied	10% diet, approximately 6,000 mg/kg/day by later EPA conversion; 25% formulation screen	Low-residual-dispersant PTFE caused no detected body-weight, liver-weight or liver-histology effect in the limited 21-day study; no formal NOAEL	Surfactant-rich preparations caused reduced weight gain or hepatic changes; short duration and limited endpoints; original 90-day Report 49-60 remains unrecovered
Kim et al. 2020 (PS)	Conference-abstract title identifies acute oral PP and PS testing in Sprague-Dawley rats; complete source unavailable	Dose and material preparation not verified	No numerical acute no-effect level or repeated-dose PS NOAEL can be supported	Do not infer PS results from Kim et al. 2021, which tested weathered PP only; source recovery is required

Broader common-polymer no-adverse-effect context

Current polymer-specific status is uneven. PP has two direct fragmented-particle repeated-dose studies: the GLP 28-day weathered-PP study with a formal NOAEL of 25 mg/kg/day, and a separate 28-day fragmented-PP study reporting a NOAEL of at least 2,000 mg/kg/day. PA-6 and PTFE have 28-day study-specific no-adverse-effect

anchors at 1,000 and at least 2,000 mg/kg/day, respectively. Cellulose acetate, a semi-synthetic thermoplastic, has a formal 94-96-day oral NOAEL of 5,000 mg/kg/day. PE has a 13-week mixture-qualified high-dose result, an archival standalone 5% dietary lead and discordant fragmented-PE pulmonary findings. PET has the same mixture-qualified high-dose result plus several lower-dose, methodologically heterogeneous studies; no single general PET point of departure is justified.

Polyethylene structure-activity context. PE is the high-molecular-mass limit of a saturated hydrocarbon chain. Increasing chain length and molecular mass generally reduce volatility, water solubility and gastrointestinal bioavailability, providing a mechanistic basis for very low intrinsic oral chemical toxicity. This is supporting evidence rather than a numerical PE NOAEL and is not a claim that every lower alkane is harmless. The expectation is consistent with the Merski mixture study and the secondary-cited standalone 5% dietary PE record (Merski et al. 2008; Welle & Franz 2018).

PVC and PS remain the clearest common-thermoplastic gaps. The 10% VCM-free PVC control diet provides lifespan resin-tolerance context, but a dedicated multi-dose polymer-only oral study has not established a formal PVC or uPVC NOAEL; Sánchez et al. (2026) provides positive hazard evidence at 1 mg/kg/day for one aged packaging-derived PVC preparation. For PS, no verified repeated-dose oral NOAEL was identified for fragmented or weathered material derived from bulk resin or product. Lefaux (1968), citing Phillips and Marks (1961), reports no abnormalities in growth, blood examinations or anatomopathology in rats fed 4% commercial Styron 475 and Styron 666 polystyrene grades. Because the original 1961 article has not been independently reviewed and the duration, formulation, particle form, group design and pathology scope remain unverified, this is archival commercial-resin tolerance context rather than a formal NOAEL or direct evidence for fragmented/weathered PS. The 2020 Kim conference-abstract title indicates acute PP and PS testing, but dose, material characterization, endpoints and results are not available at source level; the 2021 full paper must not be used as PS evidence because it tests PP only.

Supplementary archival and newer PET context

A standalone-PE study is reported secondarily in the Cosmetic Ingredient Review assessment and in Welle and Franz (2018). Lefaux (1968) summarizes the underlying commercial-polymer work and cites Phillips and Marks (1961): rats received Marlex 50 polyethylene at 1.25%, 2.5% or 5% of the diet for 90 days without reported adverse effects. The original 1961 article was not independently reviewed for this edition. Accordingly, 5% diet is retained only as secondary-source archival commercial-resin tolerance evidence, not a formal mg/kg/day NOAEL or principal quantitative anchor.

Historical commercial-polystyrene context. Lefaux (1968), citing Phillips and Marks (1961), reports that rats received a diet containing 4% Styron 475 and Styron 666 polystyrene grades without reported abnormalities in growth, blood examinations or anatomopathology. The original article remains unrecovered, so study duration, grade formulation, particle form, group design and pathology scope cannot be independently verified. This evidence supports only a secondary-source archival tolerance statement for commercial PS resins; it is not a formal NOAEL and does not resolve the repeated-dose evidence gap for fragmented or weathered PS.

Feron et al. (1981) provides direct chronic PVC context. All dietary groups, including the main control, received 10% Carina S 65-02 PVC powder; the control powder contained less than 0.3 ppm VCM, and animals were followed to 135 weeks in males and 144 weeks in females. This is unusually long-duration high-diet exposure, but the study was designed to test VCM carcinogenicity rather than PVC-polymer toxicity, and the additional ad-libitum PVC control was not histologically examined. It therefore supports formulation-specific chronic tolerance context but does not establish a formal PVC or uPVC NOAEL.

Recovered Haskell PTFE reports provide direct historical formulation evidence. In Report 224-68, low-residual-dispersant PTFE powders incorporated at 10% of the diet for 21 days - approximately 6,000 mg/kg/day in the later EPA conversion - produced no detected adverse effect on body-weight gain, liver weight or liver histopathology. PTFE recovered directly from aqueous dispersions retained more surfactant and produced reduced weight gain, enlarged livers or hepatocellular changes. Report 56-61 likewise found formulation-dependent liver-weight effects at 25% of the diet over three weeks. These reports support very low intrinsic PTFE oral toxicity and the importance of residual processing aids, but their short duration and limited endpoints do not establish a universal 6,000 mg/kg/day NOAEL. The earlier 90-day Report 49-60 remains unrecovered.

Lu et al. (2025) provides the most relevant new repeated-dose PET evidence: male mice received 0, 5, 50 or 500 micrograms/day of irregular 6-12 micrometre PET for 17 weeks. The authors stated that 5 micrograms/day produced no significant pathological or liver-function changes, whereas 50 and 500 micrograms/day produced increasing liver injury. Because the low dose increased the liver coefficient, only one sex was tested, gavage was used and no matched nonplastic comparator was included, 5 micrograms/day is described here as a study-specific highest dose without significant liver pathology or liver-function change, not a general PET NOAEL.

Lewis et al. (2026) and the piglet study by Mierzejewski et al. (2025) add acute and large-animal mechanistic information but no repeated-dose apical NOAEL. Lewis used one 5 or 50 mg/kg dose with approximately 18 hours of follow-up and found mild, sex-dependent, partly non-monotonic responses. Mierzejewski used 0.1 or 1 g/day for four weeks in five piglets per group and found one differentially expressed pancreatic gene at the low dose and 86 at the high dose, together with oxidative-stress changes. These studies remain outside the principal dose-comparison figure.

Otaka et al. (1978) is retained as PET extractables evidence rather than direct polymer-particle toxicology. Rats received chloroform extracts of titanium- or germanium-catalyzed PET resin by gavage for 90 days at doses up to 400 and 180 mg/kg/day, respectively, without treatment-related toxicity. Because the administered material was the solvent-extractable fraction - not PET resin particles or the high-molecular-mass macromolecule - these values are excluded from the polymer NOAEL table and Figure 13. The paper identifies Haskell Laboratory Report 17-73, Medical Research Project 1609, as a separate direct PET-resin dietary study that remains a priority for source recovery.

Integrated evidence synthesis

Guideline and GLP designs carry disproportionate weight because they predefine husbandry, dose administration, clinical observations, clinical pathology and histopathology. The Thomas cellulose-acetate study used a 94-96-day dietary design with 20 rats per sex per group and comprehensive apical endpoints. The Merski PE/PET study used an OECD 90-day dietary design with 10 rats per sex per group, the PA-6 study used an OECD TG 407 limit design, and Kim et al. (2021) used GLP OECD TG 407 with bulk PP resin converted by cryogenic milling, sieving, heat and UV-B weathering into irregular fragments averaging 85.2 micrometres. Neither result is universal, but these designs provide stronger apical-toxicity information than isolated biomarker screens. The archival PE, PVC and Haskell PTFE contexts do not carry equivalent weight because the original PE experiment remains unverified, PVC was the carrier/control matrix in a VCM study, and the PTFE studies were short and formulation-focused. Lu et al. 2025 is longer-duration and dose-responsive but non-guideline, one-sex and focused on hepatic and mechanistic endpoints.

The fragmented-PTFE and fragmented-PP studies also reported no adverse standard toxicological findings at 2,000 mg/kg/day for four weeks. Their short duration limits chronic inference, but the toxicity arms should not be described as fluorescent-label studies merely because separate biodistribution experiments used labelled material. The label-related limitations apply principally to tracking and translocation claims. The recovered Haskell reports independently show that PTFE outcome depends on residual dispersants: relatively clean powder was tolerated at very high dietary concentration, while surfactant-rich preparations produced hepatic effects.

A formal NOAEL is specific to the tested material, endpoints, duration, species and sex. A highest tested dose without detected adverse effects is a narrower finding. For the PE/PET fabric, the defensible statement is that approximately 1.6-2.0 g/kg/day of each component was tolerated when administered together; it is not an independently derived pure-polymer NOAEL. Percentage-diet, micrograms/day or grams/day findings should remain in their original units unless source-level food consumption, body weight and dosing data support conversion. Conversely, the low PET values are study-specific and too heterogeneous to serve as a single principal PET point of departure.

The principal gaps are chronic route-relevant exposure, complete particle characterization, validated toxicokinetics, sensitive life stages and matched nonplastic comparators. Immediate source-recovery priorities are Haskell Laboratory Report 49-60, the direct PET-resin feeding study Haskell Report 17-73 / Medical Research Project 1609, the complete Phillips and Marks (1961) commercial PE/PS article cited by Lefaux (1968), and the complete Kim et al. 2020 conference abstract or study report. Experimental priorities are dedicated guideline-quality oral studies of uPVC and fragmented/weathered PS and independent replication of the discordant PET findings with blinded quantitative pathology and particle co-localization. Future studies should separate particles from dyes, solvents, additives and leachates and chemically co-localize any particles implicated in lesions.

Evidence-weighted interpretation

The combined evidence demonstrates why polymer name alone cannot determine a toxicity value. Selected preparations of PP, PA-6, PTFE, cellulose acetate and a PE/PET mixture produced no adverse apical effects at high administered doses. Archival standalone-PE, lifespan PVC control-diet and Haskell PTFE evidence broaden the duration and formulation context but do not establish universal standalone NOAELs. No numerical repeated-dose value is assigned to real fragmented/weathered PS because the relevant source is not sufficiently reported. Different PE and PET preparations generated disputed pulmonary, hepatic, metabolic or molecular findings. Study quality, material preparation, delivery and endpoint verification govern the permitted inference.

These data constrain claims of extreme generic toxicity at trace mass exposure but do not prove harmlessness for every particle size, formulation or duration. The cellulose-acetate study adds a formal 5,000 mg/kg/day subchronic anchor, the 13-week PE/PET dietary result remains especially useful for duration and dose, and the GLP PP study provides a directly characterized weathered-fragment anchor at 25 mg/kg/day. The Haskell evidence strengthens PTFE formulation context, while Otaka is correctly moved to extractables toxicology. The upper end of the selected Figure 13 dose scale therefore increases to 5,000 mg/kg/day, but the ordinary-exposure risk conclusion does not change. The absence of a verified PS NOAEL is reported as a data gap, not filled by extrapolation from PP or laboratory spheres.

Research should prioritize independent replication of the discordant PE and PET lung and liver findings using chemically co-localized lesion particles, quantitative blinded pathology, multiple tissue sections, route-relevant dosing, matched nonplastic particles and complete controls for dye, solvent and vehicle. Haskell Reports 49-60 and 17-73 and the original standalone-PE source should be recovered before their historical findings are elevated beyond secondary or archival context.

Selected evidence records

- **(Moderate)**. Merski, J. A., Johnson, W. D., Muzzio, M., Lyang, N.-L., & Gaworski, C. L. (2008). Oral toxicity and bacterial mutagenicity studies with a spunbond polyethylene and polyethylene terephthalate polymer fabric. *International Journal of Toxicology*, 27, 387-395. <https://doi.org/10.1080/10915810802408729>
- **(Moderate)**. Buesen, R., Vogel, S., Thoma, T., et al. (2025). Oral toxicity of small microplastic of polyamide assessed by a standardized guideline study. *Microplastics and Nanoplastics*, 5, 31. <https://doi.org/10.1186/s43591-025-00137-6>
- **(Low)**. Lee, S., Kang, K.-K., Sung, S.-E., et al. (2022). Toxicity Study and Quantitative Evaluation of Polyethylene Microplastics in ICR Mice. *Polymers*, 14, 402. <https://doi.org/10.3390/polym14030402>
- **(Low)**. Lee, S., Kang, K.-K., Sung, S.-E., et al. (2022). In Vivo Toxicity and Pharmacokinetics of Polytetrafluoroethylene Microplastics in ICR Mice. *Polymers*, 14, 2220. <https://doi.org/10.3390/polym14112220>
- **(Low)**. Sijoon Lee, Dongseon Kim, Kyung-Ku Kang, et al. (2023). Toxicity and biodistribution of fragmented polypropylene microplastics in ICR mice. *International Journal of Molecular Sciences*, 24, 8463.
- **(Moderate)**. Jinsik Kim. (2021). Acute and subacute repeated oral toxicity study of fragmented microplastics in Sprague-Dawley rats. *Ecotoxicology and Environmental Safety*, 228, 112964.
- **(Low confidence / supplementary source outside the frozen baseline)**. Kim, D., Kim, D., Kim, H.-K., et al. (2025). Organ-specific accumulation and toxicity analysis of orally administered polyethylene terephthalate microplastics. *Scientific Reports*, 15, 6616. <https://doi.org/10.1038/s41598-025-91170-1>
- **(Moderate / supplementary primary source outside the frozen baseline)**. Thomas et al. (1991). Subchronic oral toxicity of cellulose acetate in rats. *Food and Chemical Toxicology*, 29(7), 453-458.
- **(Historical supplementary primary source outside the frozen baseline)**. Haskell Laboratory. (1968). Three-Week Feeding Study in Rats with Polytetrafluoroethylene Resins (Teflon). Medical Research Project No. 1080, Report No. 224-68.
- **(Historical supplementary primary source outside the frozen baseline)**. Haskell Laboratory. (1961). Medical Research Project No. 1080, Report No. 56-61. Unpublished archival PTFE feeding report.

17. Dose and Exposure Realism

Dose realism is the principal bridge between hazard and risk. Many poorly soluble particles can cause inflammation, impaired clearance or other effects when delivered or retained at sufficiently high doses; lung particle overload is a well-established example (Borm & Driscoll 2019). The primary corpus contains a large measured-occurrence component, but microplastic mechanistic toxicology is often high-dose or not quantitatively linked to environmental exposure. Administered mass, particle number, surface area and internal dose are rarely reported in mutually convertible forms.

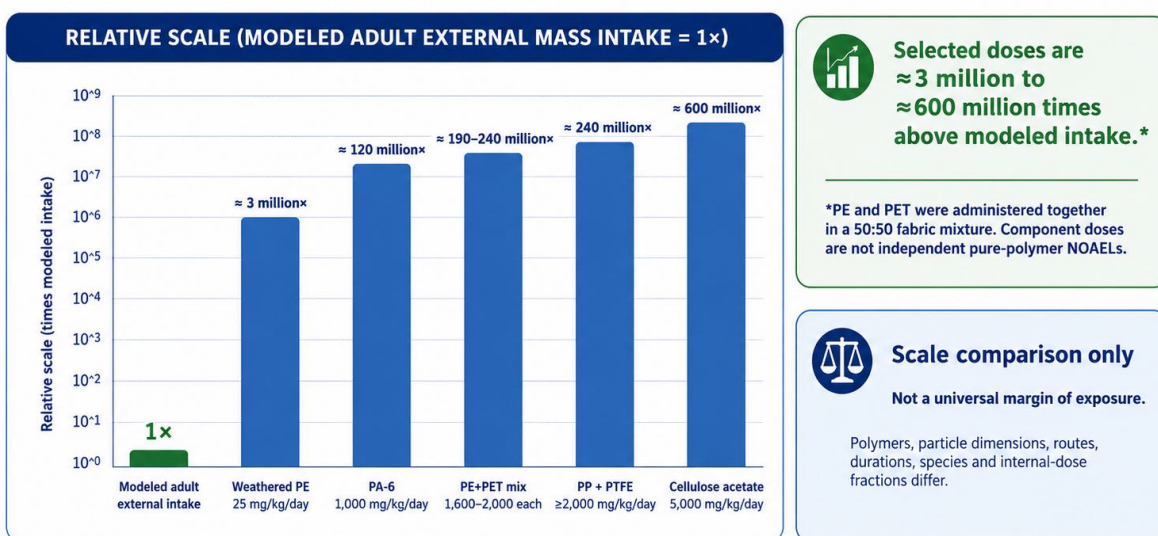
A rigorous comparison requires polymer identity, particle-size distribution, shape, density, exposure route, frequency, duration, body weight, recovery and clearance. Studies using high-dose proof-of-concept designs remain useful for mechanism discovery but should not be presented as evidence of likely population harm. Studies reporting environmentally relevant concentrations require independent verification that the concentration and particle mixture actually match the relevant matrix. (Fatima et al. 2026); (Lin et al. 2025); (Deng et al. 2017); (Stock 2021); (Horn et al. 2020); (Buesen et al. 2025)

On a common external-mass scale, the modeled adult intake of 8.33×10^{-6} mg/kg/day is approximately 3.0 million times below 25 mg/kg/day, 120 million times below 1,000 mg/kg/day, approximately 190-240 million times below the estimated 1,600-2,000 mg/kg/day PE and PET component doses in the 50:50 mixture, approximately 240 million times below 2,000 mg/kg/day for fragmented PP and PTFE, and approximately 600 million times below the 5,000 mg/kg/day cellulose-acetate NOAEL. These are diagnostic scale comparisons, not formal margins of exposure.



Real-world exposure is far below selected oral doses

Cross-study scale comparison using primary mammalian evidence.



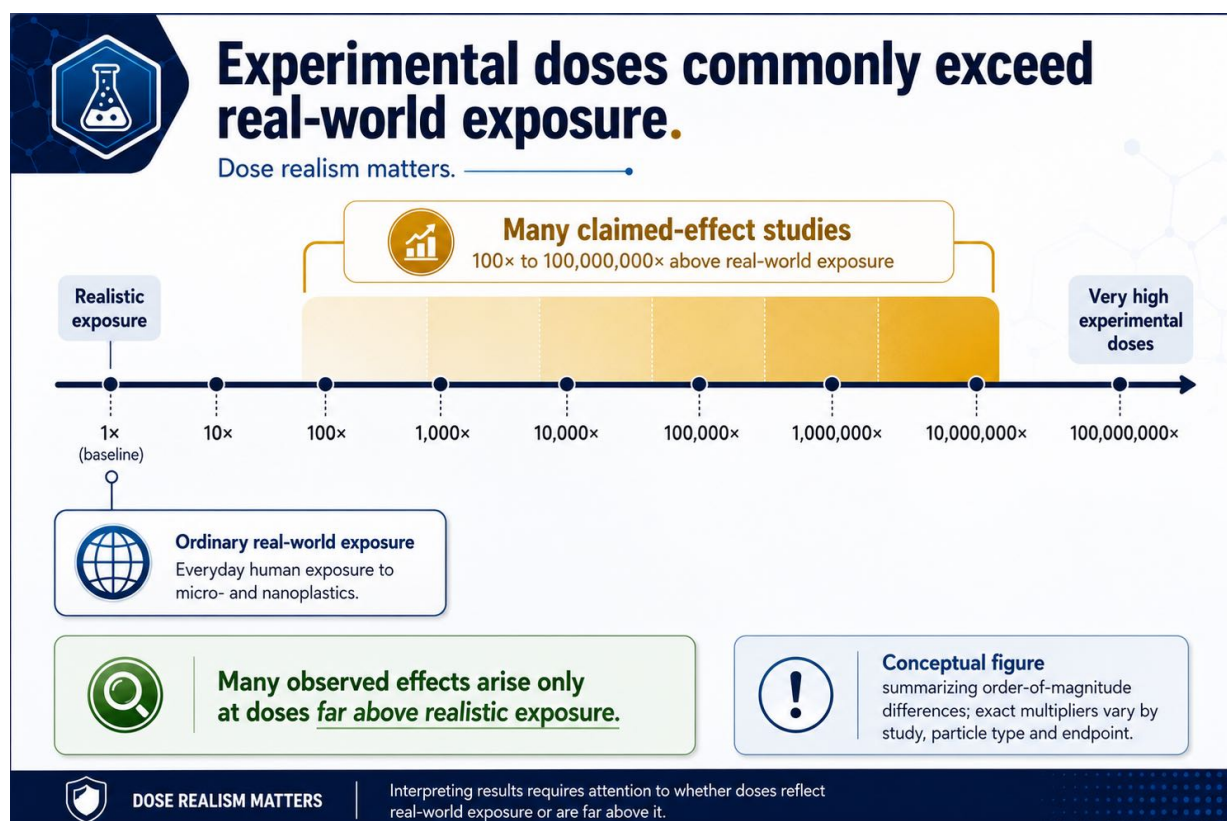
REAL-WORLD EXPOSURE IS MANY ORDERS OF MAGNITUDE BELOW SELECTED STUDY-SPECIFIC DOSES WITHOUT DETECTED ADVERSE EFFECTS.

Sources: Nor et al. 2021; Kim 2021; Buesen et al. 2025; Merski et al. 2008; Lee et al. 2022 (PTFE); Loe et al. 2023; Thomas et al. 1991.

Figure 13. Modeled ordinary adult external mass intake compared with selected study-specific oral doses without detected adverse effects for weathered fragmented polypropylene, PA-6, the PE and PET components of a 50:50 dietary mixture, fragmented polypropylene and PTFE, and cellulose acetate. The 25 mg/kg/day PP and 5,000 mg/kg/day cellulose-acetate values are formal study NOAELs and the highest tested doses under their respective designs. The PE/PET component doses were administered together and are not independent pure-polymer NOAELs. All ratios are cross-study scale comparisons, not universal margins of exposure.

The cellulose-acetate NOAEL is added to Figure 13 because it is a direct repeated-dose mg/kg/day study with comprehensive apical endpoints. The archival 5% PE dietary report, the 10% PVC control diet, the source-recovered Haskell percentage-diet PTFE records, Otaka PET-extract doses, Lu et al. PET doses expressed in micrograms/day, the single-dose Lewis study, the piglet grams/day regimen and the metadata-only PS conference

abstract are not added. They are not directly compatible with the selected repeated-dose polymer mg/kg/day anchors or lack a source-verified direct-polymer dose and result.



Source context: Phase 5 dose-realism appraisal; multipliers vary by study.

Figure 14. Conceptual dose-realism scale showing that many claimed-effect experiments use doses orders of magnitude above ordinary real-world exposure. Exact multipliers vary by study, particle type, route and endpoint.

Sensitivity analysis for exposure uncertainty

The scale separation is not dependent on assuming that the exposure model is exact. Multiplying modeled ordinary exposure by 1,000 would reduce the selected range from approximately 3.0-600 million-fold to approximately 3,000-600,000-fold. Thus, very large uncertainty in external exposure does not by itself bring ordinary intake close to the selected study-specific no-adverse-effect dose scale.

This sensitivity analysis cannot replace compatible toxicokinetics or a polymer-specific point of departure. Its value is plausibility testing: a hypothesis of severe ordinary-exposure toxicity must explain trace external mass in the context of much larger administered doses, including a 13-week dietary PE/PET study and four-week PP, PA-6 and PTFE studies, while preserving every material- and study-specific limitation.

Exposure-estimate reconciliation

Exposure estimates are stratified by measurement construct rather than forced into a nominal low-to-high interval. Nor et al. 2021 is retained as Compatibility Class A because it integrates multiple pathways with explicit harmonization and sensitivity assumptions. Yadav et al. 2022 is Class C because mass is generated from assumed three-dimensional geometry applied to counts; Song et al. 2024 is Class D because destructive polymer-marker mass in complex matrices does not verify intact particles and has conclusion-controlling interference risk. The apparent disagreement is therefore methodological, not a coherent exposure distribution.

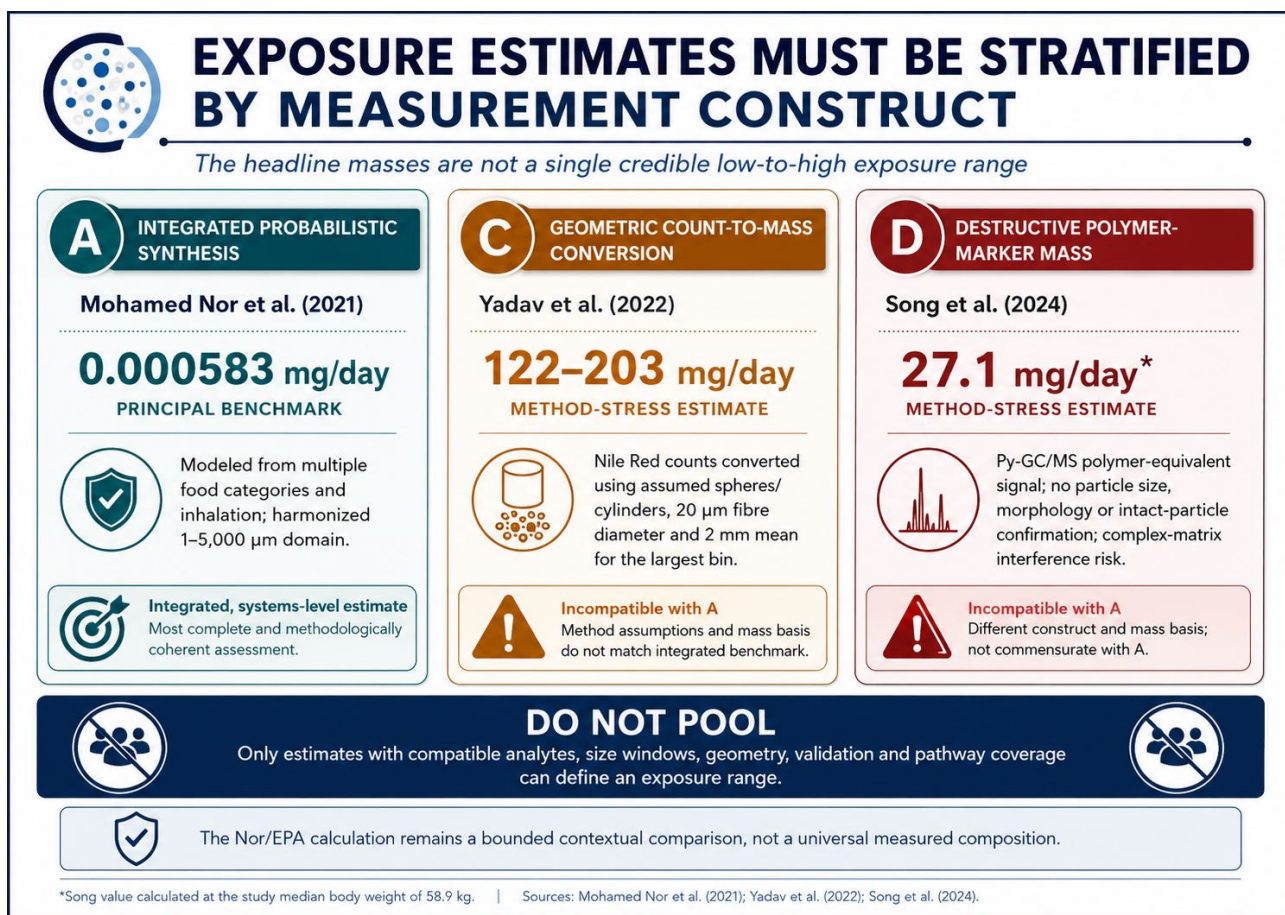


Figure 15. Exposure-estimate reconciliation by construct. The Nor et al. 2021 integrated model is retained as the principal benchmark; Yadav and Song are method-stress estimates and must not be pooled as an exposure interval. Values are on a logarithmic scale and do not imply equal evidentiary quality.

Table 9. Exposure-estimate reconciliation and permitted inference

Source	Population/pathways	Analytical basis	Central result	Major uncertainty	Class	Permitted inference
Nor et al. 2021	Adults; multiple foods plus inhalation; 1–5,000 μm	Integrated probabilistic model; heterogeneous particle-resolved inputs harmonized	583 ng/day; 883 particles/day	Modeled central estimate; incomplete pathways and smaller particles	A - integrated modeled estimate	Use for bounded contextual calculations; not a measured total personal exposure or upper bound.
Yadav et al. 2022 - supplementary method-stress source	Five volunteers for water/air; two for cooked food	Nile Red/visual counts; selected FTIR; spheres/cylinders and assumed size-bin means for mass	122–203 mg/day calculated mass	Cubic geometry makes a few large objects dominate; mass not measured	C - geometric apparent mass	Use to demonstrate count-to-mass instability; do not pool or treat as credible upper bound
Song et al. 2024 - supplementary method-stress source	26 students; 17 foods, 7 beverages, 2 air samples	Py-GC/MS polymer-associated marker mass; no intact-particle size or morphology	460.2 $\mu\text{g/kg/day}$; 27.1 mg/day at median body weight	Complex-matrix interference, sparse air sampling and internal inconsistencies	D - polymer-equivalent apparent mass	Retain as strongest contrary high-mass result; not intact-particle exposure
Compatibility rule	All populations and routes	Measured particle mass, modeled mass, geometric mass and marker mass are distinct constructs	No pooled interval	Compatibility must be demonstrated before comparison	Method rule	Present construct-specific estimates separately
Heo et al. 2024	76 food and drinking-water studies	Systematic review and quality assessment of count-based estimated daily intake	Reported estimates span many orders of magnitude	Food coverage, cutoff, methods and units are highly heterogeneous	B - systematic heterogeneity evidence	Use to demonstrate count-estimate dispersion; do not pool count ranges with mass estimates.

Sensitivity decision: the 0.0012–0.0019% value remains defensible only as the Nor et al. 2021/EPA comparison. The 30 mg/day EPA factor is the general-adult central-tendency denominator; 50 mg/day is retained as a separate traditional rural/wilderness sensitivity scenario. Because Yadav and Song are incompatible constructs, substituting them into the denominator calculation would not be a valid sensitivity analysis.

Integrated evidence synthesis

Dose realism is not a binary label. A study can be relevant in route but unrealistic in concentration, relevant in mass but unrealistic in particle size, or realistic in external concentration while using an artificial pristine material. The appraisal therefore considers route, dose magnitude, duration, frequency, preparation and the relationship between administered and internal dose.

The modeled adult external mass intake of 583 ng/day corresponds to approximately 8.33×10^{-6} mg/kg/day for an assumed 70 kg adult (Nor et al. 2021). Selected study-specific oral doses without detected adverse effects span 25 mg/kg/day for weathered PP, 1,000 mg/kg/day for PA-6, approximately 1,600-2,000 mg/kg/day for each PE and PET component administered together, at least 2,000 mg/kg/day for fragmented PP and PTFE, and 5,000 mg/kg/day for cellulose acetate. These values are about 3.0 million to 600 million times higher on the external-mass scale and are not formal margins of exposure.

Number-based comparisons can be equally misleading. A high count of nanometre particles may represent little mass but a large theoretical surface area. A small number of large fragments may dominate mass. Surface-area calculations based on assumed spheres can be wrong for fibres or irregular fragments. The report therefore retains the reported dose metric and avoids converting to another metric unless the necessary physical measurements are available.

Experimental relevance is strengthened by environmentally realistic concentration, continuous or dietary administration, weathered particles, verified dispersion, relevant duration and matched particles. It is weakened by extreme bolus dosing, uncharacterized stock suspensions, surfactant or solvent effects, particle-free controls only, and endpoints measured without correction for multiple comparisons.

Dose realism should be used to interpret scope, not to discard mechanistic evidence. High-dose studies can identify possible pathways and guide endpoint selection. The error occurs when a proof-of-concept hazard is presented as evidence of population risk without demonstrating exposure and internal dose at the effective level.

Evidence-weighted interpretation

The selected external-exposure model estimates 583 ng/person/day, or 0.000583 mg/day (Nor et al. 2021). Dividing by an illustrative 70 kg adult gives 8.33×10^{-6} mg/kg/day. Against that external-mass scale, 25 mg/kg/day is approximately 3.0 million-fold higher, 1,000 mg/kg/day approximately 120 million-fold higher, the 1,600-2,000 mg/kg/day PE/PET component range approximately 190-240 million-fold higher, 2,000 mg/kg/day approximately 240 million-fold higher, and 5,000 mg/kg/day approximately 600 million-fold higher.

These ratios are diagnostic scale comparisons, not formal margins of exposure. The modeled intake combines routes and heterogeneous occurrence studies; the toxicity doses are administered oral doses in animals; polymer, size, shape, preparation, duration and internal dose differ. A formal margin would require a compatible point of departure, validated human exposure distribution, toxicokinetics and uncertainty factors.

Dose realism is multidimensional. A study can use an environmentally reported mass concentration but an unrealistic pristine sphere, or a realistic weathered fragment at an implausible bolus dose. Evaluation should report mass, particle number, size distribution, surface area where relevant, route, duration, dispersion stability, background particles and the fraction of the administered material that reaches the target tissue.

Table 10. Experimental-dose scale compared with modeled external intake

Record	Study-specific highest dose without detected adverse effects	Approximate ratio to 8.33×10^{-6} mg/kg/day	Interpretation
Kim et al. 2021	25 mg/kg/day	3.0 million-fold	Weathered-PP rat study; study-specific scale comparison
Buesen et al. 2025	1,000 mg/kg/day	120 million-fold	GLP OECD 28-day PA-6 limit study
Merski et al. 2008	Approximately 1,600-2,000 mg/kg/day of each PE/PET component	Approximately 190-240 million-fold	13-week 50:50 mixture; components co-administered, not independent pure-polymer NOAELs
Lee et al. 2023	At least 2,000 mg/kg/day	Approximately 240 million-fold	28-day fragmented-PP toxicity arm; separate labelled clearance arm
Lee et al. 2022 (PTFE)	At least 2,000 mg/kg/day	Approximately 240 million-fold	28-day fragmented-PTFE study; author-reported NOAEL

Record	Study-specific highest dose without detected adverse effects	Approximate ratio to 8.33×10^{-6} mg/kg/day	Interpretation
Thomas et al. 1991	5,000 mg/kg/day	Approximately 600 million-fold	94-96-day cellulose-acetate dietary study; formal NOAEL, semi-synthetic thermoplastic
Compatibility rule	Modeled external human intake versus administered animal dose	Not a formal margin of exposure	Polymer, preparation, route, species, duration, internal dose and endpoints differ

Selected positive studies: reported findings interpreted with exposure realism and causal reach

Study	Design / exposure	Reported finding with essential context	Permitted inference
Sánchez et al. 2026	Female mice; aged/non-aged packaging-derived PVC; oral gavage; 1 mg/kg/day for 70 days; external administered dose	Intestinal oxidative, inflammatory, histological and microbiome changes. Essential context: About 120,000x selected modeled adult external mass exposure; no matched nonplastic particles.	Positive mammalian hazard evidence for tested PVC preparation; not ordinary human-risk evidence.
Lu et al. 2025	Male mice; irregular 6-12 µm PET; 17-week oral gavage; 5, 50 and 500 µg/day	No significant liver pathology/function change at 5 µg/day; hepatic injury at 50-500 µg/day. Essential context: Lowest dose still far above selected modeled adult mass exposure on a body-weight basis; one sex and no matched nonplastic particles.	Important chronic PET hazard and dose-response evidence; 5 µg/day is endpoint-specific, not a general PET NOAEL.
Marfella et al. 2024	Human carotid plaque cohort with prospective follow-up; No administered dose; plaque polymer-associated marker	Higher subsequent composite cardiovascular-event rate. Essential context: Human disease cohort, but plaque marker is not validated historical exposure or intact-particle dose.	Important association with prospective outcomes; causation not established.
Xu et al. 2025	Human case-control; fecal particle counts; No administered dose; fecal concentration	Strong CRC association. Essential context: Fecal count may reflect disease or diet and is not internal dose.	Strong contrary association; temporality and exposure validity unresolved.
He et al. 2025	Human CSF cohorts; No administered dose; CSF polymer measurements	Associations with amyloid and cognitive measures. Essential context: Small cohorts; analytical and multiple-testing uncertainty.	Hypothesis-generating neurological association.
Paolisso et al. 2026	Human coronary/peripheral blood; cross-sectional; No administered dose; polymer signals/counts	Higher detection in STEMI. Essential context: Smoking/co-exposure and acute disease; smoking only independent predictor of MNP presence.	Association and co-exposure evidence only.
Li et al. 2026a	Living brain-tumour patients plus five postmortem healthy donors; No administered dose; tissue particle and polymer-associated measurements	Peritumoural > healthy-brain signal; surface area correlated with proliferation. Essential context: Disease-state BBB disruption, unmatched living/postmortem comparison and selected clinical population.	Important living-brain occurrence/association evidence; causation and accumulation not established.
Chen et al. 2026	Human intestinal tumour cohort with follow-up; No administered dose; in-situ tumour particle detection	Inflammation, symptoms and recurrence trend in MP-positive tumours. Essential context: Detection within diseased tissue is not historical exposure; reverse causation and confounding remain.	Important disease-state association; not evidence that particles initiated tumour or recurrence.
Fang et al. 2026	Human CSF case-control study of intracranial aneurysm; No administered dose; one-time CSF concentration	Higher CSF concentrations associated with aneurysm status. Essential context: Selected clinical cases, uncertain temporality and exposure history.	Hypothesis-generating cerebrovascular association.
Lu et al. 2026	Case-control analysis in 239 bronchoscopy patients; No administered dose; BALF LDIR counts	Small positive associations with hyperlipidemia/dyslipidemia. Essential context: Selected respiratory-clinic population; BALF is not validated systemic historical dose.	Association with lipid measures; causation and attributable risk not established.

Table 11. Selected positive findings with dose/exposure context and permitted inference kept together.

Selected evidence records

- **(Very low).** Fatima, R., Parveen, K., Anjum, S., Abduh, M. S., Zaman, A., Naseer, F., & Ahmad, T. (2026). Dose-Dependent Toxicological Effects of Polyvinyl Chloride and Polystyrene Microplastics on Wistar Albino Rats. *Journal of Applied Toxicology*. <https://doi.org/10.1002/jat.70080>
- **(Low).** Ting-An Lin, et al. 2025. Microplastic exposure elicits sex-specific atherosclerosis development in lean low-density lipoprotein receptor-deficient mice. *Environment International*:109938.
- **(Moderate).** Yongfeng Deng, Yili Zhang, Bernardo Lemos & Hongqi Ren. 2017. Tissue accumulation of microplastics in mice and biomarker responses suggest widespread health risks of exposure. *Scientific Reports* 7:46687.

- **(Moderate)**. Valerie Stock. 2021. Uptake and cellular effects of PE, PP, PET and PVC microplastic particles. *Toxicology in Vitro*, 70 (2020) 105021.
- **(Moderate)**. Horn, D. A., Granek, E. F., & Steele, C. L. (2020). Effects of environmentally relevant concentrations of microplastic fibers on Pacific mole crab (*Emerita analoga*) mortality and reproduction. *Limnology and Oceanography Letters*, 5(1), 74–83. <https://doi.org/10.1002/lol2.10137>
- **(Moderate)**. Buesen, R., Vogel, S., Thoma, T., Strauss, V., Gröters, S., Becker, K., Müller, F., Gründling, T., Pfohl, P., Vogel, A., Haase, A., & Wohleben, W. (2025). Oral toxicity of small microplastic of polyamide assessed by a standardized guideline study. *Microplastics and Nanoplastics*, 5, 31. <https://doi.org/10.1186/s43591-025-00137-6>

18. Human Epidemiology and Clinical Associations

Human association studies report relationships with cardiovascular events, coagulation markers, fertility measures, birth outcomes, endocrine and lipid biomarkers, cerebrovascular disease and disease-state tissues. The most prominent prospective cardiovascular study reported an association between polymer-associated plaque signals and subsequent events. It remains observational and is limited by exposure classification, possible matrix interference, residual confounding and inability to establish whether disease processes altered particle retention. Newer case-control and cross-sectional studies broaden the association landscape but do not repair those causal limitations.

Across the human literature, cross-sectional design, selected clinical populations, small samples, multiple comparisons and uncertain exposure measurement are common. Reverse causation is plausible because diseased tissues may differ in perfusion, lipid content, inflammation, surgery or handling. The current evidence can support hypothesis generation and targeted replication, but it does not establish that microplastics cause human disease (Marfella et al. 2024; Lee et al. 2024; Kong et al. 2025; Shen et al. 2026; Zhang et al. 2025; Xu et al. 2025; Nihart et al. 2025; He et al. 2025; Li et al. 2026a; Chen et al. 2026; Fang et al. 2026; Lu et al. 2026; Paolisso et al. 2026).

Integrated evidence synthesis

The principal limitation of current human epidemiology is exposure measurement. Many studies classify exposure from a single tissue, blood, stool or environmental sample whose analytical validity and temporal representativeness are uncertain. A disease process may alter tissue permeability, inflammation, diet, medication or clinical handling, creating reverse causation or differential contamination.

The Lane et al. audit adds a prerequisite often missed in epidemiological appraisal: the internal-exposure variable must first be analytically and physiologically fit for quantitative use. A study can have prospective follow-up and comparatively low risk of bias under ROBINS-E yet still lack a validated particle dose because blanks, recovery, size, count, shape or sample representativeness are inadequate. Statistical adjustment cannot repair a measurement that does not quantify the claimed internal exposure.

Cross-sectional associations establish co-occurrence, not temporality. Case-control studies require controls drawn from the same source population and comparable sample collection. Prospective cohorts require a validated exposure measure that precedes the outcome. Statistical adjustment cannot correct an exposure variable that does not measure the claimed internal dose (Marfella et al. 2024; Kong et al. 2025).

Clinical populations often differ in age, smoking, occupation, socioeconomic status, diet, comorbidities, medication and healthcare contact. These factors can influence both disease and particle exposure or detection. Multiple polymers, tissues and biomarkers also create a high multiplicity burden; nominally significant associations require correction and replication.

Mechanistic plausibility from animal or cell studies is insufficient to establish human causation when doses and preparations differ substantially. The causal interpretation of the individual human association studies remains unresolved: current studies generate hypotheses but do not quantify attributable risk or establish a disease caused by realistic microplastic exposure. That does not make the integrated ordinary-exposure risk conclusion unresolved; the broader exposure, toxicology, kinetic and comparative-particle evidence weighs against a material public-health effect.

A decisive epidemiological program would combine repeated validated external measurements, size- and polymer-resolved internal biomarkers, prospective follow-up, preregistered outcomes, negative controls, independent replication and toxicokinetic evidence connecting external exposure to the measured internal dose.

Association is not the same as causation

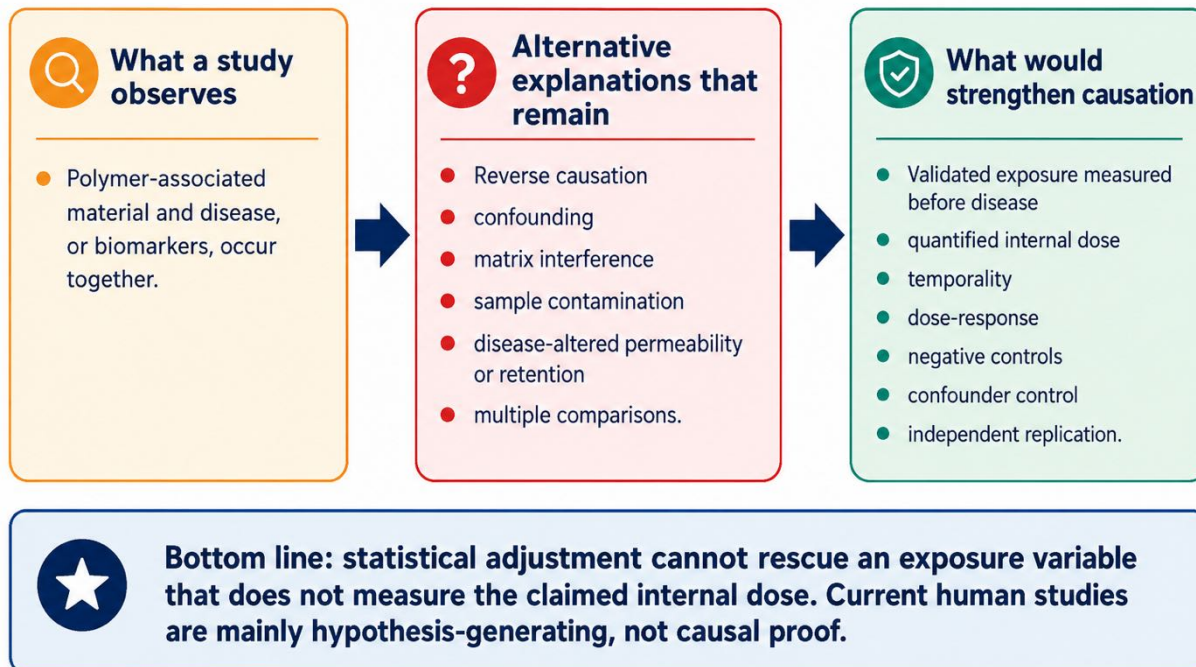


Figure 15A. Association is not the same as causation. Human observational studies may show that polymer-associated material and disease occur together, but alternative explanations remain unless exposure is measured validly before disease, internal dose is quantified, temporality is demonstrated, confounding is controlled and the finding is independently replicated.

Evidence-weighted interpretation

The human observational literature currently provides hypothesis-generating associations rather than causal estimates. The atheroma study measured polymer-related material in plaques from a selected surgical cohort and observed higher subsequent event rates in the detection group (Marfella et al. 2024). Detection preceded the follow-up events but did not establish the timing or source of plaque material, the initiation of atherosclerosis, or freedom from residual confounding. The study is clinically important and requires replication, but it does not by itself demonstrate that microplastics caused the events.

Cross-sectional biomarker studies are even more limited for causality. The blood study related particle counts to coagulation and inflammatory markers in 36 adults (Lee et al. 2024). Placental studies related delivery-time particle measures to neonatal anthropometry or thyroid markers (Shen et al. 2026; Zhang et al. 2025). These designs cannot establish temporality, are vulnerable to multiple testing and residual confounding, and do not show that modest biomarker differences produce clinical disease.

The infertility-cohort study used targeted thermal-marker mass in follicular fluid and seminal plasma (Kong et al. 2025). Thermal polymer mass does not establish nanoplastic size, morphology or intact-particle location, and a selected clinical population may differ systematically in age, treatment, lifestyle and underlying disease. Association with fertilization or sperm measures is not equivalent to impaired fertility caused by particles.

To materially change the present conclusion, human evidence would need validated exposure or internal-dose measurement, temporal separation of exposure and disease, sufficient sample size, prespecified outcomes, control of major confounders, replication and a coherent dose-response. No current study satisfies that full chain.

Table 12. Selected human observational evidence: each reported association is kept together with its causal boundary.

Study	Design / exposure measure	Reported association and permitted inference
Atheroma/events (Marfella et al. 2024)	Carotid endarterectomy cohort; plaque polymer-associated material; prospective outcome follow-up	Association/occurrence only: Higher subsequent event rate in the detection group. Causal boundary: Prospective outcome follow-up is important, but diseased plaque is not a pre-disease exposure measure; selection, residual confounding, signal identity and causal direction remain unresolved.
Blood/coagulation (Lee et al. 2024)	Cross-sectional; micro-FTIR counts	Association/occurrence only: Associations with selected markers. Causal boundary: Small sample, multiple tests and no temporality.
Fertility (Kong et al. 2025)	Infertility cohort; targeted thermal-marker mass	Association/occurrence only: Selected fertilization/sperm differences. Causal boundary: Marker mass is not nanoplastic morphology; clinical confounding.
Birth anthropometry (Shen et al. 2026)	Placenta at delivery; LD-IR	Association/occurrence only: Selected anthropometric associations. Causal boundary: Cross-sectional surrogate, model selection and residual confounding.
Neonatal thyroid (Zhang et al. 2025)	Placenta at delivery; LD-IR	Association/occurrence only: Selected hormone associations. Causal boundary: Multiple outcomes and uncertain clinical significance.
Semen/testis occurrence (Zhao et al. 2023; Montano et al. 2023)	Cross-sectional analytical detection	Association/occurrence only: Material reported in samples. Causal boundary: No causal outcome analysis or dose-response.
Colorectal cancer (Xu et al. 2025)	Case-control; fecal LDIR counts	Association/occurrence only: Median 62 vs 43 items/g; adjusted highest-quartile OR 11.3. Causal boundary: Reverse causation and fecal-exposure validity; disease, diet, transit and treatment can change stool counts.
CSF/Alzheimer changes (He et al. 2025)	Small CSF cohorts; polymer measurements and biomarkers	Association/occurrence only: PE/PVC associated with amyloid status and selected cognitive measures. Causal boundary: Small cohorts, disease-state sampling, multiple analyses and method uncertainty.
Coronary circulation (Paolisso et al. 2026)	Cross-sectional; coronary and peripheral blood in 61 patients	Association/occurrence only: More frequent detection in STEMI; smoking only independent predictor of MNP presence. Causal boundary: Selected clinical sample, confounding/co-exposure and acute disease; no causal temporality.
Decedent brain (Nihart et al. 2025)	Postmortem tissue; Py-GC/MS plus complementary methods	Association/occurrence only: Higher apparent brain mass and dementia subgroup values. Causal boundary: Associative; corrected supplementary errors; analytical quantities and signal assignment disputed.

Recent human evidence and systematic review

Tran et al. 2026 identified 25 living-human studies and concluded that micro- and nanoplastic measurements were associated with inflammatory, functional or clinical findings across several systems, while heterogeneity and bias constrained causal inference. Its ROBINS-E assessment rated 11 studies low risk, eight with some concerns and six high risk. This does not conflict with the report's lack of High causal-evidence records: ROBINS-E assesses risk of bias in non-randomized exposure studies, whereas the present grade additionally requires validated particle/exposure measurement, temporal and dose relevance, outcome adversity and causal sufficiency.

The 2026 update adds several important association studies. Chen et al. detected in-situ Raman-assigned particles in 106 of 188 intestinal tumour specimens and reported localized inflammation, more severe gastrointestinal symptoms and a trend toward recurrence in the detection group. Fang et al. reported higher cerebrospinal-fluid microplastic concentrations in intracranial-aneurysm cases. Lu et al. reported small positive associations between bronchoalveolar-lavage microplastic counts and lipid disorders in 239 bronchoscopy patients. Paolisso et al. found higher coronary-blood detection in STEMI, but smoking was the only independent predictor of MNP presence. These studies increase the need for replication; none provides a validated historical ordinary-exposure dose or excludes reverse causation and residual confounding.

Study	Design / exposure measure	Reported finding and causal boundary
Marfella et al. 2024	Prospective follow-up after carotid plaque sampling	Association/occurrence only: Polymer-associated plaque material predicted subsequent cardiovascular events. Causal boundary: Exposure proxy was diseased plaque; residual confounding, selection, signal identity and causal direction remain unresolved. Prospective outcomes strengthen temporality for events, not causation of plaque formation.
Xu et al. 2025	Case-control; fecal LDIR counts; 258 cases/493 controls	Association/occurrence only: CRC cases had higher counts; highest quartile OR 11.3 (95% CI 6.77-19.5). Causal boundary: Fecal particles may be consequence

Study	Design / exposure measure	Reported finding and causal boundary
		or correlate of disease, diet, bowel transit or treatment; no validated historical exposure dose.
Nihart et al. 2025	Postmortem organ comparison and dementia subgroup	Association/occurrence only: Higher polymer-associated mass in brain and dementia cases. Causal boundary: Associative postmortem design; correction issued; analytical signal assignment and implausibly high quantities disputed by BfR.
He et al. 2025	Small CSF cohorts with Alzheimer biomarkers and follow-up	Association/occurrence only: PE/PVC measures associated with amyloid status and cognitive measures. Causal boundary: Small selected samples, multiple comparisons, disease-state sampling and uncertain biological-matrix validation.
Paolisso et al. 2026	Cross-sectional coronary/peripheral blood study; 61 angiography patients	Association/occurrence only: Detection: 84.2% STEMI, 40% chronic coronary syndrome, 31.8% controls. Causal boundary: Smoking was the only independent predictor of MNP presence; small selected clinical sample and acute disease can alter sampling.
Li et al. 2026a	Brain-tumour tissue series; 113 living patients and five postmortem healthy donors	Association/occurrence only: Nearly universal detection; peritumoural > healthy signal; surface area correlated with proliferation. Causal boundary: Disease-state BBB disruption, unequal donor structure and living/postmortem comparison prevent temporal or causal inference.
Chen et al. 2026	188 intestinal tumour specimens with mean 34.1-month follow-up	Association/occurrence only: 106/188 MP-positive; localized inflammation, symptom elevation and recurrence trend. Causal boundary: Tumour detection is not antecedent exposure; reverse causation, residual confounding and detection-group selection remain.
Fang et al. 2026	Case-control; CSF microplastic concentration and intracranial aneurysm	Association/occurrence only: Higher CSF concentrations associated with aneurysm risk. Causal boundary: One-time disease-state CSF sample; selected cases, uncertain temporality and exposure validity.
Lu et al. 2026	Case-control analysis; BALF LDIR in 239 bronchoscopy patients	Association/occurrence only: Each 100-unit increase in selected MP metrics was associated with modestly higher odds of lipid disorders. Causal boundary: Selected clinical population; BALF count is not a validated historical systemic dose; residual confounding and reverse causation remain.

Table 13. Recent positive human studies: reported findings and causal boundaries shown together.

Selected evidence records

- **(Low).** Marfella, R., Prattichizzo, F., Sardu, C., et al. (2024). Microplastics and nanoplastics in atheromas and cardiovascular events. *New England Journal of Medicine*, 390, 900–910. <https://doi.org/10.1056/NEJMoa2309822>
- **(Low).** Lee, D.-W., Jung, J., Park, S.-A., et al. (2024). Microplastic Particles in Human Blood and Their Association with Coagulation Markers. *Scientific Reports*. <https://doi.org/10.1038/s41598-024-81931-9>
- **(Low).** Kong, F., Fang, L., Gui, Y., Lan, H., Zhao, P., et al. (2025). Polyethylene and Polyvinyl Chloride Nanoplastics in Human Follicular Fluid and Seminal Plasma: Impact on Fertilization and Sperm Quality. *ACS Nano*. <https://doi.org/10.1021/acsnano.5c00918>
- **(Low).** Shen, Y., Ning, Y., Chen, F., Wang, X., Zheng, D., Sun, Y., Zhang, X., & Zhang, X. (2026). Impact of placental microplastics on birth anthropometrics: A cross-sectional study. *Ecotoxicology and Environmental Safety* 309, 119572. <https://doi.org/10.1016/j.ecoenv.2025.119572>
- **(Low).** Zhang, X., Li, L., Zhang, Y., Liu, B., Wang, X., & Sun, L. (2025). Placental microplastics contamination and its impact on thyroid function in newborns. *Ecotoxicology and Environmental Safety* 304, 119056. <https://doi.org/10.1016/j.ecoenv.2025.119056>
- **(Moderate).** Zhao, Q., et al. (2023). Detection and characterization of microplastics in the human testis and semen. *Science of the Total Environment*, 877, 162713. <https://doi.org/10.1016/j.scitotenv.2023.162713>
- **(Moderate).** Montano, L., et al. (2023). Raman microspectroscopy evidence of microplastics in human semen. *Science of the Total Environment*, 901, 165922. <https://doi.org/10.1016/j.scitotenv.2023.165922>

19. Ecotoxicology

Controlled ecotoxicology studies report mortality, reproductive, developmental, behavioral and physiological effects in some organisms. Field relevance is strongest when concentrations are measured in the relevant habitat, particle properties are environmentally representative and apical outcomes are used. Some studies report effects at concentrations intended to be environmentally relevant; others report no gross pathology or no effect under comparable or lower conditions.

Plastic-specific attribution remains a recurrent weakness. Matched comparator studies show that responses can reflect generic suspended-particle stress, food dilution, surface area, shape or density rather than polymer chemistry. Larger debris ingestion and entanglement are separate hazards and should not be conflated with microplastic toxicology. (Horn et al. 2020); (Bacchetta et al. 2024); (Straub et al. 2017); (Gerdes 2019); (Ogonowski et al. 2018); (Clukey et al. 2017)

Integrated evidence synthesis

Ecotoxicology includes ingestion, feeding, growth, reproduction, behaviour, biochemical markers, histopathology and mortality across diverse species. Effects observed in one organism cannot be generalized to all taxa because feeding mode, gut anatomy, habitat, life stage and particle encounter differ. Sessile filter feeders, deposit feeders, pelagic fish and terrestrial invertebrates experience different exposure pathways.

Laboratory concentrations often exceed measured environmental levels or use monodisperse spheres that remain suspended more readily than weathered particles. Food dilution, gut filling or physical interference can produce adverse effects at high particle loads without implying chemical toxicity. Particle-free controls identify an effect of adding material, but matched natural or nonplastic particles are needed to establish plastic specificity (Straub et al. 2017; Gerdes 2019; Ogonowski et al. 2018).

Field occurrence in gut contents demonstrates encounter and ingestion, not necessarily tissue uptake or adverse outcome. Population-level risk requires evidence that individual effects occur at environmental concentration and scale to survival or reproduction under natural variability. Local hotspots, aquaculture systems, industrial releases or sediment sinks may create higher risk than broad regional averages.

Chemical formulation and shape can matter. Fibres may entangle or persist differently from spheres, and leachates or additives may produce effects not attributable to intact polymer. Ecological assessment should therefore report both particle and chemical exposure, include relevant comparators and distinguish temporary physiological responses from adverse effects on growth, reproduction or population performance.

Evidence-weighted interpretation

Ecotoxicity is strongest when four elements align: measured environmental concentration, a comparable experimental particle, a matched nonplastic-particle control, and an ecologically adverse endpoint such as survival, growth or reproduction. Many studies satisfy only one or two elements. A particle-free control establishes an effect of adding material, not that plastic is uniquely responsible.

The Pacific mole-crab study reported increased mortality and reproductive effects at concentrations described as environmentally relevant, but it used one polypropylene fibre preparation without a matched natural-fibre or mineral control (Horn et al. 2020). A PVC study found no adverse effect at 0.1 micrograms/mL and species-specific effects at 1-10 micrograms/mL, again without a matched natural-particle comparator (Bacchetta et al. 2024). These are credible hazard observations with incomplete plastic-specific attribution.

Comparator studies provide a stronger interpretive framework. Amphipods ingested and excreted both plastics and silica, with modest material-dependent biological differences (Straub et al. 2017). The mixing-test/reference-material study was specifically designed to separate particle-suspension artefacts from microplastic effects (Gerdes 2019). Field turtle studies show that ingestion can be frequent while gross lesions or poorer body condition are not demonstrable in a selected sample (Duncan et al. 2019; Clukey et al. 2017).

Table 14. Ecological findings with the plastic-specificity boundary kept with each result.

Evidence type	Reported result with plastic-specificity boundary	Evidentiary meaning
Matched-particle method	Reference-particle mixing test (Gerdes 2019). Boundary: Comparator must match size, shape, density and surface.	High-quality approach to generic particle effects
Plastic versus silica	Ingestion/egestion and modest effects (Straub et al. 2017). Boundary: Field concentration and exact matching remain limited.	Direct comparator
Polypropylene fibres	Mole-crab mortality/reproduction effects (Horn et al. 2020). Boundary: No matched natural-fibre/mineral control.	Functional adverse endpoints
PVC cross-species test	No effect at 0.1; effects at 1-10 micrograms/mL (Bacchetta et al. 2024). Boundary: No matched nonplastic comparator; field benchmarking incomplete.	Dose- and species-dependent response
Field ingestion	Turtles frequently ingested debris (Duncan et al. 2019). Boundary: Not tissue uptake or population effect.	Robust exposure evidence
Field null pathology	91% ingestion; no attributed lesions/body-condition effect (Clukey et al. 2017). Boundary: Cross-sectional bycatch sample.	Important null evidence

Selected evidence records

- **(Moderate)**. Horn, D. A., Granek, E. F., & Steele, C. L. (2020). Effects of environmentally relevant concentrations of microplastic fibers on Pacific mole crab (*Emerita analoga*) mortality and reproduction. *Limnology and Oceanography Letters*, 5(1), 74–83. <https://doi.org/10.1002/lol2.10137>
- **(Moderate)**. Bacchetta, R., Pica, A., Santo, N., Tremolada, P., & Sugni, M. (2024). Comparative effects of polyvinyl chloride microplastics on the brittle star *Ophiactis virens* and the amphibian *Xenopus laevis*. *Aquatic Toxicology*, 272, 106975. <https://doi.org/10.1016/j.aquatox.2024.106975>
- **(Moderate)**. Sandrine Straub, Philipp E. Hirsch and Patricia Burkhardt-Holm. 2017. Biodegradable and Petroleum-Based Microplastics Do Not Differ in Their Ingestion and Excretion but in Their Biological Effects in a Freshwater Invertebrate *Gammarus fossarum*.
- **(High)**. Zandra Gerdes. 2019. A novel method for assessing microplastic effect in suspension through mixing test and reference materials. *Scientific Reports*.
- **(Moderate)**. Ogonowski, M., Wenman, V., Danielsson, S., & Gorokhova, E. (2018). Hydrophobic organic contaminants are not linked to microplastic uptake in Baltic Sea herring. *bioRxiv*. <https://doi.org/10.1101/363127>
- **(Moderate)**. Clukey, K. E., Lepczyk, C. A., Balazs, G. H., Work, T. M., & Lynch, J. M. (2017). Investigation of plastic debris ingestion by four species of sea turtles collected as bycatch in pelagic Pacific longline fisheries. *Marine Pollution Bulletin*, 120(1–2), 117–125. <https://doi.org/10.1016/j.marpolbul.2017.04.064>

20. Soil, Crops and Terrestrial Systems

Soil and terrestrial studies show that plastic residues can persist, alter physical properties and interact with organisms and chemicals. Effects vary with polymer, shape, loading, soil type, aging and endpoint. Long-term field evidence is more relevant than short-term high-loading microcosms, but it remains limited.

Several studies find no effect at lower or field-relevant loadings while effects appear at high percentages by soil mass. Crop-yield and global productivity claims require direct crop evidence and must account for plastic-mulch benefits, background mineral and biological particles, and the distinction between measured outcomes and modeled extrapolation. Zhu et al. 2025 is retained as a secondary synthesis with major causal and modeling limitations rather than direct crop-yield evidence. (Schöpfer et al. 2022); (Lin et al. 2020); (Letwin et al. 2026); (Li et al. 2021); (Weber 2022); (Ramage et al. 2025); (Zhu et al. 2025)

Integrated evidence synthesis

Terrestrial exposure arises from agricultural films, compost, biosolids, litter, tyre and road-derived particles, atmospheric deposition and irrigation. Soil is a complex particle-rich matrix containing minerals, organic matter, biological fragments and anthropogenic debris. Recovering and identifying plastic against this background is more difficult than in clean water, and small-particle estimates are especially method-dependent.

Experimental soil studies report changes in aggregation, bulk density, water movement, microbial communities and plant responses. The direction and magnitude depend on polymer, shape, concentration, soil type and moisture. Fibres can alter physical structure at high loadings, but a particle-free soil control does not reveal whether the effect is unique to plastic rather than to adding another low-density fibre or fragment (Schöpfer et al. 2022; Lin et al. 2020).

Claims of crop uptake require rigorous exclusion of surface contamination and proof of polymer identity inside plant tissue. Fluorescent nanosphere studies can demonstrate transport of a tracer-like preparation under controlled conditions, but free dye, aggregation and unrealistic hydroponic exposure limit extrapolation to field crops. Larger particles are less biologically plausible for intact translocation into edible tissues.

Crop-yield claims require direct exposure-effect evidence. Global yield-loss estimates should not be inferred by combining unrelated climate or productivity relationships with assumed plastic effects. At the same time, a complete agronomic assessment must include both potential particle releases and the documented functions of plastic mulch, irrigation and protective materials. Risk-benefit and substitution consequences are separate from toxicology but essential to policy.

The strongest terrestrial priorities are validated soil methods, field-realistic long-term experiments, matched particle comparators, polymer mass balance, and direct measurements of crop yield and edible-tissue transfer under actual agricultural practice.

Evidence-weighted interpretation

Long-term field records demonstrate persistence and redistribution more clearly than harm. Plastic residues remained detectable decades after sewage-sludge application and repeated sludge use was associated with higher soil burdens (Weber 2022; Ramage et al. 2025). Those findings establish a durable source pathway; they do not show that plastic is a large fraction of total soil particles or that the detected concentration caused adverse effects.

Field and field-based experiments report both null and mixed responses. An arable-soil study found persistence without detectable effects on microbial biomass, enzyme activity or crop yield under the tested conditions (Schöpfer et al. 2022). A field addition study reported reduced selected soil-fauna measures but stimulated microbial activity (Lin et al. 2020). These findings are compatible because polymers, concentrations, shapes, soils, duration and endpoints differ.

Global crop- and photosynthesis-loss claims require particular caution. The retained synthesis record does not directly measure crop yield under representative field exposure; it transfers heterogeneous laboratory chlorophyll responses through additional models (Zhu et al. 2025). A credible agricultural assessment must directly measure yield and quality, include field-realistic concentrations and nonplastic comparators, and separately account for the agronomic benefits and substitution consequences of plastic products.

Selected evidence records

- **(Moderate)**. Schöpfer, L., et al. (2022). Microplastics persist in an arable soil but do not affect soil microbial biomass, enzyme activities, or crop yield. *Journal of Plant Nutrition and Soil Science*, 185, 836–849. <https://doi.org/10.1002/jpln.202200062>
- **(Moderate)**. Lin, D., Yang, G., Dou, P., Qian, S., Zhao, L., Yang, Y., & Fanin, N. (2020). Microplastics negatively affect soil fauna but stimulate microbial activity: Insights from a field-based microplastic addition experiment. *Proceedings of the Royal Society B*, 287, 20201268. <https://doi.org/10.1098/rspb.2020.1268>
- **(Moderate)**. Letwin, A., et al. (2026). Assessing the accumulation of microplastics in earthworms (*Eisenia fetida*) using traditional bioaccumulation modeling and synchrotron-based microcomputed tomography. *Environmental Toxicology and Chemistry*, 45, 1553–1566. <https://doi.org/10.1093/etojnl/vgag072>
- **(Very low)**. Li, M., Wu, D., Wu, D., Guo, H., & Han, S. (2021). Influence of polyethylene-microplastic on environmental behaviors of metals in soil. *Environmental Science and Pollution Research*, 28, 28329–28336. <https://doi.org/10.1007/s11356-021-12718-y>
- **(Moderate)**. Collin J. Weber. 2022. Investigating the dispersal of macro- and microplastics on agricultural fields 30 years after sewage sludge application. *Scientific Reports*, <https://doi.org/10.1038/s41598-022-10294-w>.
- **(Moderate)**. Stuart J. F. F. Ramage, et al. 2025. Microplastics in agricultural soils following sewage sludge applications: Evidence from a 25-year study. *Chemosphere* 376:144277.
- **(Not scored - secondary synthesis)**. Zhu, R., Zhang, Z., Zhang, N., Zhong, H., Zhou, F., Zhang, X., Liu, C., Huang, Y., Yuan, Y., Wang, Y., Li, C., Shi, H., Rillig, M. C., Dang, F., Ren, H., Zhang, Y., & Xing, B. (2025). A global estimate of multiecosystem photosynthesis losses under microplastic pollution. *Proceedings of the National Academy of Sciences*, 122(11), e2423957122. <https://doi.org/10.1073/pnas.2423957122>

21. Aquatic Systems

Aquatic occurrence is documented from rivers and coasts to open ocean and deep sea. Distribution is controlled by buoyancy, biofouling, aggregation, ingestion, sinking, resuspension and fragmentation. Surface-net datasets observe only a size- and density-selected fraction and cannot be treated as complete ocean inventories.

Wildlife ingestion is common in some taxa, but ingestion frequency is not equivalent to tissue absorption or adverse population effect. Sea-turtle studies illustrate both frequent occurrence and explicit null findings for gross pathology or body condition in selected samples. Ecological risk must integrate concentration, particle properties, species sensitivity and functional outcome. (Egger et al. 2020); (Zhao 2023); (Woodall et al. 2014); (Law et al. 2014); (Duncan et al. 2019); (Clukey et al. 2017)

Integrated evidence synthesis

Aquatic particles distribute among the surface microlayer, water column, shoreline, biota and sediment. Density alone does not determine position because biofouling, aggregation, shape and turbulence change buoyancy and settling. Surface trawls therefore sample a selected fraction and cannot represent the full water column or small-particle inventory (Egger et al. 2020; Zhao 2023).

Ingestion is common in laboratory and field studies, but gut content is not equivalent to assimilation. Many organisms can egest particles, and residence time depends on size, feeding mode and food availability. Apparent trophic transfer may reflect prey gut contents rather than transfer into consumer tissues. Demonstrating biomagnification requires concentration normalized to trophic level and evidence of retained internal burden.

Laboratory effects often occur under static or semi-static conditions with concentrations maintained above those measured in the field. Sediment species may encounter higher local concentrations, but exposure should be based on the relevant sediment mass or porewater rather than a water-column count. Temperature, salinity, food, pathogens and natural suspended solids can modify responses.

The evidence supports specific risks where exposure and organism sensitivity align, not a universal aquatic hazard level. Stronger studies combine field concentration, controlled dose-response, matched particles and ecologically adverse endpoints such as survival, growth or reproduction. Biomarker changes alone should not be equated with population injury.

Evidence-weighted interpretation

Surface scarcity, water-column occurrence and deep-sea deposition can all be true within the same ocean system (Egger et al. 2020; Zhao 2023; Woodall et al. 2014). Buoyancy is modified by biofouling, aggregation, shape and turbulence, while sampling nets exclude smaller and deeper material. A surface survey is therefore not a complete stock assessment, and a deep-sea detection is not a measured global sink fraction.

Wildlife gut occurrence is an exposure endpoint. It does not establish tissue translocation, biomagnification or adverse population effect. Turtle surveys reported widespread ingestion (Duncan et al. 2019), while a bycatch study found plastic in 91% of sampled turtles without attributing gastrointestinal blockage, lesions or poorer body condition to the debris in that sample (Clukey et al. 2017). Both findings should be retained.

Population-level ecological risk requires evidence that individual effects alter survival, reproduction or recruitment at field exposure. Laboratory biomarker changes and high local concentrations can identify plausible mechanisms or hotspots, but they should not be generalized across species, habitats and particle mixtures without field validation.

Selected evidence records

- **(Moderate)**. Egger, M., Nijhof, R., Quiros, L., et al. (2020). A spatially variable scarcity of floating microplastics in the eastern North Pacific Ocean. *Environmental Research Letters*, 15, 114056. <https://doi.org/10.1088/1748-9326/abbb4f>
- **(Moderate)**. Shiye Zhao. 2023. Pelagic microplastics in the North Pacific Subtropical Gyre: A prevalent anthropogenic component of the particulate organic carbon pool.
- **(Moderate)**. Woodall, L. C., Sanchez-Vidal, A., Canals, M., Paterson, G. L. J., Coppock, R., Sleight, V., Calafat, A., Rogers, A. D., Narayanaswamy, B. E., & Thompson, R. C. (2014). The deep sea is a major sink for microplastic debris. *Royal Society Open Science*, 1, 140317. <https://doi.org/10.1098/rsos.140317>

- **(Moderate)**. Law, K. L., et al. (2014). Distribution of Surface Plastic Debris in the Eastern Pacific Ocean from an 11-Year Data Set. *Environmental Science & Technology*. <https://doi.org/10.1021/es4053076>
- **(Moderate)**. Duncan, E. M., Broderick, A. C., Fuller, W. J., Galloway, T. S., Godfrey, M. H., Hamann, M., Limpus, C. J., Lindeque, P. K., Mayes, A. G., Omeyer, L. C. M., Santillo, D., Snape, R. T. E., & Godley, B. J. (2019). Microplastic ingestion ubiquitous in marine turtles. *Global Change Biology*, 25(2), 744–752. <https://doi.org/10.1111/gcb.14519>
- **(Moderate)**. Clukey, K. E., Lepczyk, C. A., Balazs, G. H., Work, T. M., & Lynch, J. M. (2017). Investigation of plastic debris ingestion by four species of sea turtles collected as bycatch in pelagic Pacific longline fisheries. *Marine Pollution Bulletin*, 120(1–2), 117–125. <https://doi.org/10.1016/j.marpolbul.2017.04.064>

22. Wastewater and Treatment

Wastewater treatment plants can substantially reduce particles in final effluent, but removal depends on unit process, particle size, density and identification method. Captured material often transfers to sludge, creating a terrestrial pathway when biosolids are applied to land.

Treatment studies should report complete mass balance, influent and effluent recovery, size distribution and fate of residual streams. Count-based removal can be misleading if fragmentation changes the number of particles or if the analytical window excludes smaller material. (Talvitie et al. 2017); (Michielssen et al. 2016); (Edo et al. 2020); (Kärkkäinen 2025); (Akarsu et al. 2021); (Tanaka et al. 2023)

The corrected Tanaka et al. 2023 laundry experiment reported 46.2 mg/person/day and 394,000 fibres/person/day for a pulsator-type machine versus 210 mg/person/day and 2,180,000 fibres/person/day for a drum-type machine. The experiment used one approximately 1.7 kg clothing ensemble containing 85% synthetic and 15% natural/other fibres, a 10 micrometre net and fluorescence-staining microscopy. The drum condition also included machine drying while the pulsator condition was air-dried. These limitations prevent the values from being treated as polymer-confirmed total emissions or generalized environmental exposure.

Integrated evidence synthesis

Wastewater treatment plants receive particles from household dust, laundering, personal-care and industrial sources, road runoff and sewer infiltration. Influent and effluent counts depend on sampling volume, temporal variability, solids handling and the lower size cutoff. Composite sampling is generally more representative than a single grab sample.

Conventional treatment can capture a large proportion of particles through screening, settling, biological flocs and filtration. High percentage removal does not mean destruction; most captured material enters sludge or another residual stream (Talvitie et al. 2017; Michielssen et al. 2016). Assessment should report both water and solids, account for changing flow and solids concentration, and avoid interpreting fragmentation below the method cutoff as removal.

Advanced membranes, electrocoagulation and other technologies can achieve high removal in model systems (Akarsu et al. 2021). Full-scale relevance depends on fouling, energy, chemical use, concentrate handling, nanoscale performance and secondary waste. A technology should be evaluated on total-system mass balance rather than only the treated-water endpoint.

The corrected Tanaka et al. 2023 is an original laundry source-release experiment by Tanaka and Shimatani (2023). It reports higher emissions from the tested drum-type washing/drying condition than from the pulsator condition, but fluorescence and microscopy without polymer-specific confirmation, mixed natural and synthetic fibres, confounded drying conditions and limited replication constrain quantitative attribution. The study supports laundry as a source pathway, not a universal emission factor.

Wastewater policy should distinguish source reduction, capture, transfer to sludge and verified destruction. Measures that reduce one discharge pathway may relocate material unless the residual stream and ultimate fate are included.

Evidence-weighted interpretation

A wastewater mass balance should follow influent water, treated effluent, screenings, grit, sludge, backwash and any concentrate. Count-based removal alone can be biased by fragmentation, aggregation or the analytical lower cutoff. A high percentage removal can coexist with substantial residual discharge when flow is large, and it usually represents transfer to a solids stream rather than destruction (Talvitie et al. 2017; Michielssen et al. 2016; Edo et al. 2020).

The corrected laundry experiment is useful as a source-pathway demonstration, not a universal emission factor. Its two washing conditions differed in machine type and drying, the clothing load included natural/other fibres, and identification relied on fluorescence and microscopy without polymer-specific confirmation (Tanaka et al. 2023). Its reported mass and fibre counts should therefore remain within the study design and not be converted directly to population exposure.

Table 15. Source, treatment and fate endpoints

Process	Evidence	Measured endpoint	What must not be claimed
Laundry release	Tanaka and Shimatani 2023	Study-specific fibre/mass release under two conditions	Universal polymer-confirmed emission factor
Conventional/advanced treatment	Talvitie et al. 2017; Michielssen et al. 2016	Reduced water-phase counts	Destruction or zero discharge
Sludge transfer	Edo et al. 2020; Kärkkäinen 2025	Captured material in residual solids	Elimination from environment
Electrocoagulation/membrane	Akarsu et al. 2021	High model-system removal	Full-scale performance without fouling/residual assessment
Weathering/biodegradable materials	Bagheri et al. 2017	Deterioration or persistence by environment	Mineralization without carbon balance

Selected evidence records

- **(Moderate)**. Talvitie, J., Mikola, A., Koistinen, A., & Setälä, O. (2017). Solutions to microplastic pollution – Removal of microplastics from wastewater effluent with advanced wastewater treatment technologies. *Water Research*, 123, 401–407. <https://doi.org/10.1016/j.watres.2017.07.005>
- **(Moderate)**. Michielssen, M. R., Michielssen, E. R., Ni, J., & Duhaime, M. B. (2016). Fate of microplastics and other small anthropogenic litter (SAL) in wastewater treatment plants depends on unit processes employed. *Environmental Science: Water Research & Technology*, 2, 1064–1073. <https://doi.org/10.1039/C6EW00207B>
- **(Moderate)**. Edo, C., González-Pleiter, M., Leganés, F., Fernández-Piñas, F. & Rosal, R. 2020. Fate of Microplastics in Wastewater Treatment Plants and Their Environmental Dispersion with Effluent and Sludge. *Environmental Pollution* 259:113837. <https://doi.org/10.1016/j.envpol.2019.113837>
- **(Moderate)**. Niina Kärkkäinen. 2025. Microplastic Pollution via Wastewater Effluent and Sewage Sludge: Special Focus on Microplastic Fibres in Compost. *Water, Air, & Soil Pollution*.
- **(Moderate)**. Akarsu, C., et al. (2021). Removal of microplastics from wastewater through electrocoagulation–electroflotation and membrane filtration processes. *Water Science and Technology*, 84(7), 1648–1662. <https://doi.org/10.2166/wst.2021.356>
- **(Low)**. Tanaka, S., & Shimatani, S. (2023). Microplastics Derived from Chemical Fibers. *Material Cycles and Waste Management Research*, 34(3), 178–182. <https://doi.org/10.3985/mcwmr.34.178>

23. Fragmentation, Weathering and Fate

Weathering and mechanical stress can embrittle plastic and generate smaller particles. Rates depend on polymer grade, stabilizer package, geometry, temperature, ultraviolet exposure, oxygen, abrasion and biological colonization. Accelerated laboratory weathering is useful for mechanism comparison but does not directly establish field generation rates.

Fragmentation increases particle number and surface area while conserving polymer mass unless chemical degradation or mineralization occurs. Biodegradable, compostable and oxo-degradable labels do not guarantee rapid disappearance in marine or soil environments. (Weinstein et al. 2016); (Chowreddy et al. 2026); (Song et al. 2020); (Napper et al. 2019); (Bagheri et al. 2017)

Integrated evidence synthesis

Weathering changes surface chemistry, brittleness, density, biofilm and fragmentation behaviour. Ultraviolet exposure, oxygen, heat, abrasion and mechanical stress act differently across polymers and formulations. Laboratory acceleration can identify mechanisms but does not automatically provide an environmental half-life because field intensity, geometry and exposure history differ (Weinstein et al. 2016; Chowreddy et al. 2026).

Fragmentation increases particle number while conserving most polymer mass. A large object can generate many smaller particles without creating new mass, and a count-based trend can rise as mass declines or moves below the analytical cutoff. Disappearance from a visible size class is therefore not evidence of biodegradation.

Transport models require emissions, size distribution, density, fragmentation, aggregation, settling, resuspension and degradation. Uncertainty in any of these can produce a wide range of residence-time or sink estimates. Observed surface stocks are not a complete mass balance because material may strand, sink, be buried, remain in larger debris or occur below the sampling window.

Verified destruction requires chemical conversion. Depolymerization, oxidation or mineralization should be demonstrated with carbon balance, products and by-products. Surface cracking, weight loss, loss of tensile strength or inability to recover fragments may indicate deterioration but not complete conversion (Napper et al. 2019; Bagheri et al. 2017).

Evidence-weighted interpretation

Weathering changes surface chemistry, brittleness and fragment production, but it does not necessarily reduce polymer mass or environmental persistence. Salt-marsh and sunlight studies demonstrate fragmentation under defined conditions (Weinstein et al. 2016; Song et al. 2020). Accelerated tests can compare formulations, but field rates depend on ultraviolet exposure, temperature, oxygen, abrasion, biofouling and geometry (Chowreddy et al. 2026).

Disintegration, weight loss and surface erosion are not synonymous with mineralization. Materials marketed as biodegradable can remain functional or generate fragments in soil and aquatic environments (Napper et al. 2019; Bagheri et al. 2017). Verified destruction requires carbon balance, carbon-dioxide or methane evolution where relevant, residual-polymer analysis and accounting for dissolved products.

Selected evidence records

- **(Moderate)**. Weinstein, J. E., Crocker, B. K., & Gray, A. D. (2016). From macroplastic to microplastic: Degradation of high-density polyethylene, polypropylene, and polystyrene in a salt marsh habitat. *Environmental Toxicology and Chemistry*, 35(7), 1632–1640. <https://doi.org/10.1002/etc.3432>
- **(Moderate)**. Chowreddy, R. R., Fredriksen, S. B., Anwar, H., Mylvaganam, B., Iveland, A., & Kamffjord, T. (2026). Degradation behaviour of different polyethylene and polypropylene materials under long-term accelerated weathering conditions. *Journal of Polymer Research*, 33, 45. <https://doi.org/10.1007/s10965-026-04777-x>
- **(Moderate)**. Song, Y. K., Hong, S. H., Jang, M., Han, G. M., Jung, S. W., & Shim, W. J. (2020). Rapid Production of Micro- and Nanoplastics by Fragmentation of Expanded Polystyrene Exposed to Sunlight. *Environmental Science & Technology*, 54(18), 11191–11200. <https://doi.org/10.1021/acs.est.0c02288>

- **(Moderate)**. Napper, I. E., & Thompson, R. C. (2019). Environmental deterioration of biodegradable, oxo-biodegradable, compostable and conventional plastic carrier bags in the sea, soil and open-air over a 3-year period. *Environmental Science & Technology*, 53(9), 4775–4783. <https://doi.org/10.1021/acs.est.8b06984>
- **(Moderate)**. Bagheri, A. R., Laforsch, C., Greiner, A., & Agarwal, S. (2017). Fate of so-called biodegradable polymers in seawater and freshwater. *Global Challenges*, 1, 1700048. <https://doi.org/10.1002/gch2.201700048>

24. Natural and Nonplastic Particle Comparisons

Every environment and every human exposure pathway contains particles. Humans and ecosystems encounter a much larger mixture of mineral, biological, cellulosic, combustion-derived and other anthropogenic particles than plastic alone. A particle-free control therefore establishes the effect of adding particles, not the effect uniquely attributable to plastic. Matched natural or alternative-particle controls and a denominator for the total particle mixture are central to plastic-specific causal inference.

Comparator studies show a mixed pattern. Some plastic preparations differ from silica, diatomite, cellulose or biobased fibres; in other cases, effects are similar or dominated by particle size, shape and concentration. Comparator quality depends on matching surface area, morphology, density and dose. (Gerdes 2019); (Straub et al. 2017); (Ogonowski et al. 2016); (Courtene-Jones et al. 2024); (Yardy et al. 2021)

In 916 ocean-surface samples, approximately 8% of characterized fibres were synthetic and about 92% were cellulosic or animal-derived (Suaria et al. 2020). The Paris airborne study similarly found roughly two-thirds natural or cellulosic fibres (Dris et al. 2017). These findings demonstrate why visual fibre counts and particle-free controls cannot establish plastic-specific exposure or effects, while not implying identical composition in every matrix.

Integrated evidence synthesis

The background mixture includes mineral dust, soil, soot, combustion particles, pollen, fungal spores, plant and animal fibres, tyre and brake wear, cellulose, skin debris, food particles, pigments, metals and other industrial particles. This continuous exposure is normal and unavoidable. Plastic-specific risk cannot be inferred from its mere presence or from a particle-free control when the proposed mechanism is generic inflammation, obstruction, sorption or surface interaction.

Particle comparison does not mean all dusts are equally hazardous. The evidence distinguishes ordinary low-toxicity background dust from specific high-hazard constituents. Respirable crystalline silica or quartz has an established exposure-response relationship with lung cancer; occupational wood dust is strongly associated with sinonasal cancer; and arsenic, cadmium, chromium, lead, mercury and related compounds are systemic toxicants, with several species carcinogenic (Steenland et al. 2001; Demers et al. 1995; Tchounwou et al. 2012). Quartz is therefore not an appropriate low-toxicity comparator. These substances warrant special controls because their hazards are demonstrated. Comparable human disease evidence is absent for ordinary plastic dust.

Environmental studies should report the plastic fraction of the total particle burden where feasible. A method that preselects hydrophobic or visually plastic-like objects cannot determine this denominator. Without the denominator, plastic detection cannot be interpreted as dominance, and effects observed in a naturally particle-rich environment cannot automatically be attributed to the plastic minority.

The purpose of comparison is not to declare every particle harmless. It is to identify the actual causal agent and place its dose and potency in context. Shape, persistence, chemistry and surface properties can produce different responses, but decision relevance requires a demonstrated effect at realistic exposure large enough to matter against the total particle background. On the current evidence, ordinary plastic dust has not demonstrated the exceptional potency needed to justify special treatment.

Direct human-lung context: numerous and diverse nonplastic particles

Hayden et al. 2023 provides an important reality check for interpreting reports of "particles in the human lung." Using automated in-situ FE-SEM/EDS, the investigators characterized 124,018 inorganic particles in just 38 surgical lung-biopsy specimens, assigning them to 45 particle or mineral classes. The material was extraordinarily diverse: clays such as illite, chlorite, kaolinite and talc; feldspars; quartz; titanium dioxide; iron-rich aluminosilicates; aluminum oxide; zircon and zirconia; and numerous metal- or metal-oxide particles. These included cerium oxide, rare-earth oxides containing La, Ce and Nd, tin oxide, antimony oxide, copper, zinc and Cr/Mn/Fe/Ni-rich steel-like particles, with rare lead-, uranium- and thorium-containing particles also identified. More than 90% of the analyzed particles were below 2.5 micrometres in greatest dimension, and most titanium-rich and trace-metal particles were below 1 micrometre. The central lesson is therefore not simply that foreign particles can be found in lung tissue; it is that any plastic detection occurs within a large and chemically diverse background of nonplastic particulate matter.

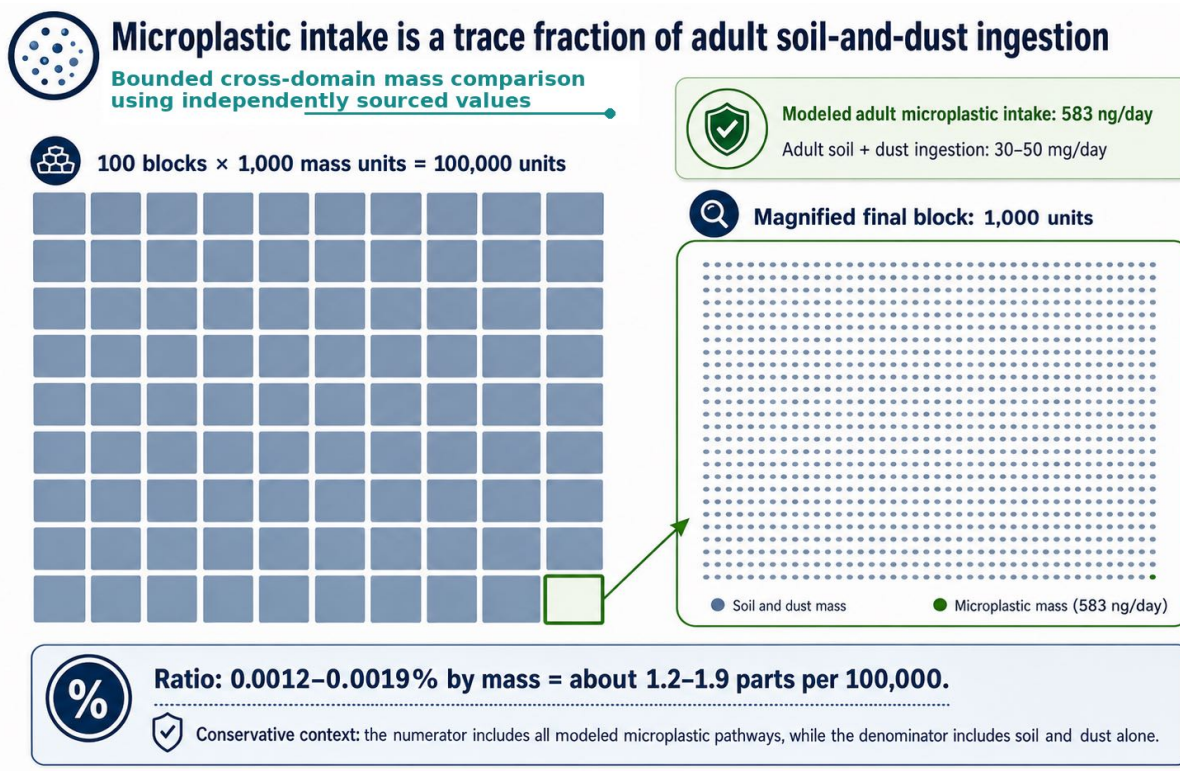
Hayden et al. should not be used to calculate a plastic-versus-nonplastic lung fraction. The biopsies came from selected military clinical cohorts, only a fixed 1 x 1 mm tissue area was analyzed per specimen, and the authors described the method as qualitative rather than a quantitative measure of total lung burden. In addition, backscattered-electron detection preferentially highlights material with average atomic number greater than carbon, so the method was inherently better suited to inorganic particles than to plastics; clustering and overlapping particles could also produce mixed spectra or undercounting. Those limitations define what Hayden can quantify, but they do not diminish its contextual importance. Human lung tissue can contain very large numbers of particles from many nonplastic materials, including some with well-established toxic or carcinogenic hazards. A discussion that singles out plastic particles while ignoring this broader particulate background is scientifically incomplete and can distort risk interpretation. The relevant questions are not merely "Was plastic detected?" but "What fraction of the relevant particle burden is plastic, how do its dose and potency compare with the other particles present, and is there evidence that plastic contributes disproportionate harm?" Hayden et al. shows why that broader particulate context is essential for scientifically meaningful assessment.

Ultrasmall nonplastic nanoparticle kinetic context

Miller et al. 2017 used gold as a relatively inert, highly traceable nonplastic nanoparticle to test whether inhaled particles can cross the pulmonary barrier. The study demonstrates that some ultrasmall particles can translocate, but also shows why particle size cannot be ignored: systemic appearance was substantially greater for approximately 4 nm than approximately 34 nm primary particles, and the animal series showed markedly less transfer above approximately 10-30 nm. The corrected illustrative human estimate was approximately 0.02% of inhaled mass over the defined 24-hour calculation. Thus, translocation is a generic nanoparticle phenomenon under some conditions, not a plastic-specific property, and evidence from a few-nanometre tracer should not be projected quantitatively to micrometre-scale plastics.

Comparative particle-burden perspective

An independent mass comparison places modeled adult microplastic intake at approximately 0.0012-0.0019% of adult soil-plus-dust ingestion alone - about one to two parts per 100,000 (Nor et al. 2021; U.S. EPA 2017). This should not be mistaken for a universal composition measurement. Matrix-specific same-sample studies report different fractions because they use different metrics, size windows and methods: Maurizi et al. 2024 found a mean indoor microplastic-to-nonplastic particle ratio of 0.2% by count (range 0-1.6%); Paris fibre studies found natural or cellulosic fibres dominant (Dris et al. 2017; Dris et al. 2016); and Jüngling et al. 2026 identified plastics as $0.36 \pm 0.13\%$ of particles in a proof-of-principle 0.5-10 micrometre potable-water analysis. The exact fraction varies, but every compatible comparison places plastic as a minority and the broad mass comparison places it at trace level.



Sources: Nor et al. 2021; U.S. EPA Exposure Factors Handbook, Chapter 5 (2017).

Figure 16. Bounded cross-domain mass comparison. Modeled adult microplastic intake of 583 ng/day is approximately 0.0012–0.0019% of EPA adult soil-plus-dust ingestion values of 30–50 mg/day – about 1.2–1.9 parts per 100,000. The calculation is contextual rather than a universal measured particle composition: numerator and denominator are independently derived and cover different pathways.

The hazard mechanisms most often invoked for microplastics are not unique. Poorly soluble low-toxicity particles can cause inflammation or impaired clearance at overload doses; clay, silica and metal oxides carry surface charge; soot, black carbon, organic matter and sediment sorb hydrophobic chemicals; microbes colonize glass, metal, minerals and natural fibres; and many particle types can be inhaled, ingested, translocated or retained under selected conditions. Plastic particles can therefore produce effects at very high, atypical doses, just as other dust particles can. Polymer chemistry is distinctive, but distinct chemistry is not equivalent to distinctive toxicity at realistic dose (Borm & Driscoll 2019).

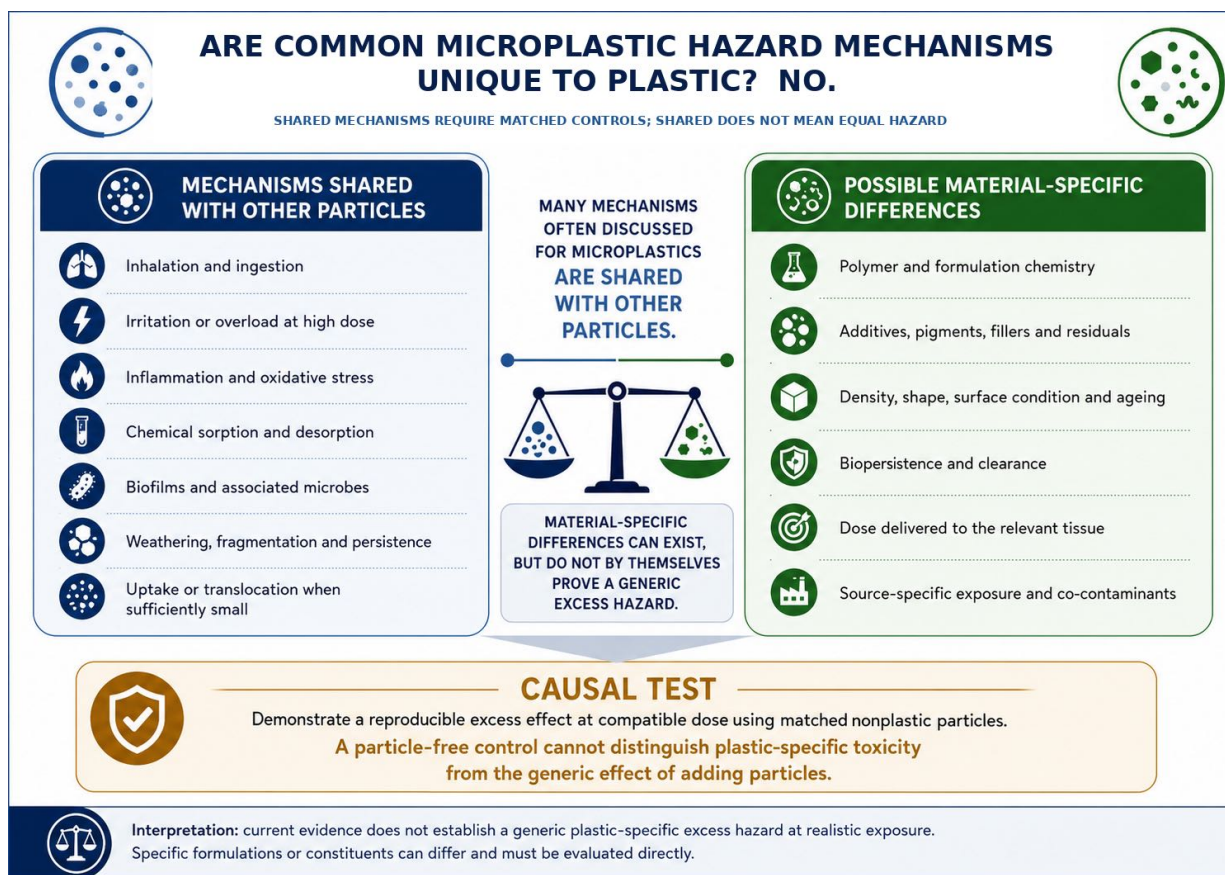


Figure 17. Are commonly proposed generic microplastic hazard mechanisms unique to plastic? No. High-dose particle effects and the generic mechanisms invoked for microplastics occur across many particle classes. Shared mechanisms do not prove equal hazard, but they do require matched comparators and realistic dose before a plastic-specific excess effect is claimed. Material- or formulation-specific differences remain possible and should be evaluated directly.

Ogonowski 2018 emphasized that many microplastic experiments cannot distinguish a plastic-specific response from ordinary suspended-particle effects (Ogonowski 2018). Matched-comparator work supports the same methodological conclusion (Gerdes 2019; Ogonowski et al. 2016). A 2023 meta-analysis reported only marginally greater average experimental hazard for microplastics than natural suspended solids, with substantial uncertainty, heterogeneous endpoints and limited direct comparability (Ogonowski et al. 2023). Even that result concerns laboratory hazard under selected conditions, not demonstrated risk at ordinary exposure and not exceptional potency relative to known hazardous dusts.

As a transparent heuristic, if ordinary plastic particles have the same potency per unit mass as the remaining particle mixture, an independently derived mass share of approximately 0.0012-0.0019% would contribute the same trace fraction of the generic particle burden. To contribute 1% of total burden, plastic would require approximately 520-870-fold greater potency per unit mass; to contribute 10%, approximately 5,700-9,500-fold; and to equal the remaining burden, approximately 51,000-86,000-fold. This is not a formal risk estimate because size, deposition, shape, chemistry, route and clearance can change potency. No evidence demonstrates anything approaching this exceptional generic potency for ordinary plastic particles at realistic exposure. Known high-hazard agents such as asbestos, crystalline silica, combustion soot, occupational wood dust and reactive or toxic metal compounds require substance-specific assessment; hazardous additives, leachates, pigments or fillers should likewise be assessed as those constituents.

Bottom line: ordinary plastic dust is one small component of ubiquitous dust. Its presence is expected; its ability to cause effects at extreme loading is not unique; and current evidence provides no scientific basis for treating it as a special public-health hazard. Where a specific formulation, additive, combustion product, occupational exposure or local hotspot is hazardous, that agent and dose should be assessed directly.

Route-specific comparator framework

Comparator choice must follow the route, deposition metric and endpoint. Respirable silica is relevant to inhalation toxicology but not automatically to oral ingestion or aquatic food dilution. Soil and food-associated insoluble solids

are more relevant oral comparators; suspended sediment and detritus are more relevant aquatic comparators; pigments, minerals, metal oxides, biological debris and laboratory contamination are relevant to tissue-detection claims.

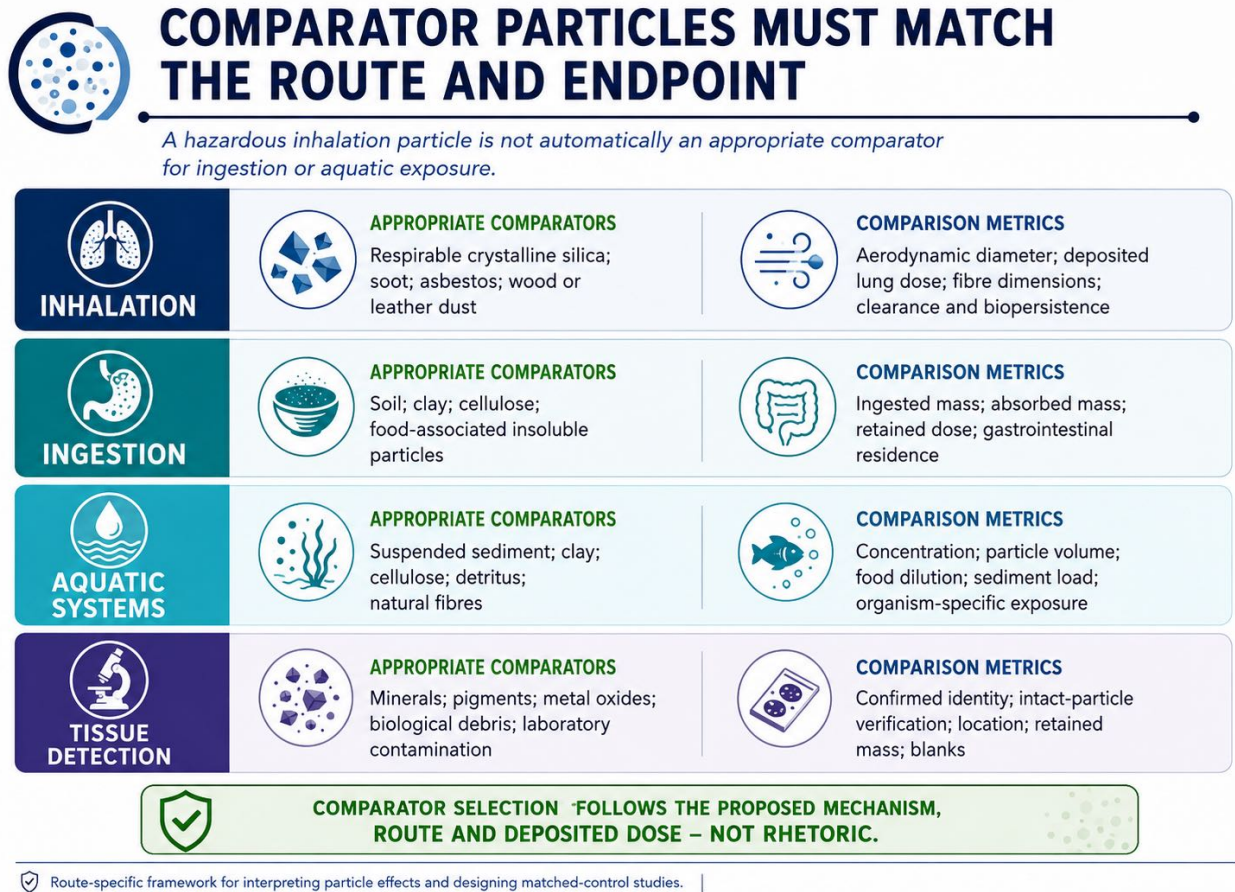


Figure 18. Route-specific particle comparators. Scientific comparability depends on route, particle dimensions, deposited or ingested dose, biopersistence, matrix and endpoint; hazardous inhalation particles should not be used rhetorically as generic comparators for every route.

Table 16. Comparator selection by route and evidentiary question

Route/question	Appropriate comparators	Decision metrics	Required interpretation
Inhalation	Respirable crystalline silica, soot, asbestos, wood dust, leather dust, occupational polymer dust	Aerodynamic diameter; fibre length/diameter; deposited dose; clearance; biopersistence; co-exposures	Use route-matched inhalation evidence. Do not infer oral or aquatic hazard from inhalation classification.
Ingestion	Soil, clay, cellulose, food-associated insoluble particles and other ordinary ingested solids	Ingested mass; size distribution; gastrointestinal persistence; absorbed and retained dose	Compare external intake separately from uptake. A particle-free control cannot test plastic specificity.
Aquatic systems	Suspended sediment, clay, cellulose, detritus and natural fibres	Concentration, particle volume, food dilution, sediment load, exposure duration, organism and life stage	Match environmental concentration and food availability; separate particle effects from additives and starvation.
Tissue detection	Pigments, minerals, metal oxides, biological debris, procedural contamination and polymer-associated markers	Confirmed identity; intact-particle verification; location; blanks; recovery; retained mass	A destructive marker or image alone does not establish an intact particle, origin, accumulation or pathology.

Selected evidence records

- **(High).** Zandra Gerdes. 2019. A novel method for assessing microplastic effect in suspension through mixing test and reference materials. Scientific Reports.
- **(Moderate).** Sandrine Straub, Philipp E. Hirsch and Patricia Burkhardt-Holm. 2017. Biodegradable and Petroleum-Based Microplastics Do Not Differ in Their Ingestion and Excretion but in Their Biological Effects in a Freshwater Invertebrate Gammarus fossarum.

- **(Moderate)**. Ogonowski, M., Schür, C., Jarsén, Å., & Gorokhova, E. (2016). The Effects of Natural and Anthropogenic Microparticles on Individual Fitness in *Daphnia magna*. PLOS ONE, 11(5), e0155063. <https://doi.org/10.1371/journal.pone.0155063>
- **(Moderate)**. W. Courteney-Jones, F. De Falco, F. Burgevin, et al. 2024. Are Biobased Microfibers Less Harmful than Conventional Plastic Microfibers: Evidence from Earthworms. Environmental Science & Technology.
- **(Moderate)**. Yardy, L., & Callaghan, A. (2021). Microplastic and Organic Fibres in Feeding, Growth and Mortality of *Gammarus pulex*. Environments, 8(8), 74. <https://doi.org/10.3390/environments8080074>
- **(Supplementary primary context outside frozen baseline)**. Hayden, L., Strausborger, S., & Lewin-Smith, M. (2023). 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), 235-243. <https://doi.org/10.1093/micmic/ozac015>.
- **(Supplementary primary kinetic/context publication lineage outside frozen baseline)**. Miller, M. R., Raftis, J. B., Langrish, J. P., et al. (2017). Inhaled Nanoparticles Accumulate at Sites of Vascular Disease. ACS Nano, 11(5), 4542-4552; correction ACS Nano 11, 10623-10624.

- (Supplementary comparative-particle context outside frozen baseline). Borm, P. J. A., & Driscoll, K. E. (2019). The hazards and risks of inhaled poorly soluble particles - where do we stand after 30 years of research? *Particle and Fibre Toxicology*, 16, 11. <https://doi.org/10.1186/s12989-019-0294-4>
- (Supplementary established-hazard context outside frozen baseline). Steenland, K., 't Mannetje, A., Boffetta, P., et al. (2001). Pooled exposure-response analyses and risk assessment for lung cancer in 10 cohorts of silica-exposed workers: an IARC multicentre study. *Cancer Causes & Control*, 12(9), 773-784. <https://doi.org/10.1023/A:1012214102061>
- (Supplementary established-hazard context outside frozen baseline). Demers, P. A., Kogevinas, M., Boffetta, P., et al. (1995). Wood dust and sino-nasal cancer: pooled reanalysis of twelve case-control studies. *American Journal of Industrial Medicine*, 28(2), 151-166. <https://doi.org/10.1002/ajim.4700280202>
- (Supplementary established-hazard context outside frozen baseline). Tchounwou, P. B., Yedjou, C. G., Patlolla, A. K., & Sutton, D. J. (2012). Heavy metal toxicity and the environment. *EXS*, 101, 133-164. https://doi.org/10.1007/978-3-7643-8340-4_6

25. Additives, Leachates and Sorbed Chemicals

Plastic debris can contain formulation additives and sorb environmental chemicals. Field and laboratory studies demonstrate chemical presence, partitioning and transfer under selected conditions. These endpoints are chemically distinct from intact-particle toxicity and should not be attributed automatically to the polymer particle itself.

Vector risk depends on incremental biological dose relative to water, sediment, food and natural particles. Field fish evidence can show no relationship between microplastic uptake and contaminant burden, while prepared co-exposure studies demonstrate that transfer can occur. Both findings are compatible: transfer is possible, but its net importance is scenario-dependent. (Hirai et al. 2011); (Rochman et al. 2014); (Ogonowski et al. 2018); (Wardrop et al. 2016); (Lee et al. 2018); (Bejgarn et al. 2015)

Integrated evidence synthesis

Plastic products are formulations, not pure polymers. Residual monomers, catalysts, stabilizers, plasticizers, pigments, flame retardants and processing aids can migrate or leach depending on product, temperature, solvent, ageing and surface area. A biological effect from a particle preparation may reflect the particle, an extractable chemical, the dispersant or a combination.

Leachate studies answer a different question from intact-particle studies. They can be directly relevant to product chemistry, but they should not be described as evidence that the polymer particle itself caused the effect. Conversely, washing particles to remove extractables may create an artificial preparation that underrepresents a real product. Fractionated designs testing particles, leachate and recombined exposure are the most informative (Hirai et al. 2011; Almroth et al. 2023).

Otaka et al. (1978) illustrates this distinction. Rats received chloroform extracts of titanium- or germanium-catalyzed PET resin for 90 days at doses up to 400 or 180 mg/kg/day, respectively, without treatment-related toxicity. The study is informative for aggressively extracted PET-associated low-molecular-mass material, but it does not establish a PET-particle or PET-macromolecule NOAEL and is excluded from the direct-polymer dose comparison.

Sorbed environmental chemicals can associate with plastic surfaces, but risk depends on the chemical gradient, competing natural particles, food and sediment, desorption kinetics and the contribution of plastic relative to other exposure pathways. Demonstrating sorption is not the same as demonstrating increased organism dose. In some conditions particles may carry chemicals; in others they may reduce bioavailability.

Alternatives should be evaluated on their own composition and lifecycle. Paper, natural fibres, glass, metals and biodegradable polymers can release particles or chemicals and are not automatically inert (Almroth et al. 2023). Comparative assessment should consider function, total material use, energy, emissions, waste, litter and toxicity rather than assuming that the absence of conventional plastic eliminates exposure.

Selected evidence records

- **(Moderate)**. Hirai, H., Takada, H., Ogata, Y., Yamashita, R., Mizukawa, K., Saha, M., Kwan, C., Moore, C., Gray, H., Laursen, D., Zettler, E. R., Farrington, J. W., Reddy, C. M., Peacock, E. E., & Ward, M. W. (2011). Organic micropollutants in marine plastics debris from the open ocean and remote and urban beaches. *Marine Pollution Bulletin*, 62(8), 1683–1692. <https://doi.org/10.1016/j.marpolbul.2011.06.004>
- **(Moderate)**. Rochman, C. M., Hoh, E., Kurobe, T., & Teh, S. J. (2014). Long-Term Sorption of Metals Is Similar among Plastic Types: Implications for Plastic Debris in Aquatic Environments. *PLOS ONE*, 9(1), e85433. <https://doi.org/10.1371/journal.pone.0085433>
- **(Moderate)**. Ogonowski, M., Wenman, V., Danielsson, S., & Gorokhova, E. (2018). Hydrophobic organic contaminants are not linked to microplastic uptake in Baltic Sea herring. *bioRxiv*. <https://doi.org/10.1101/363127>
- **(Low)**. Wardrop, P., Shimeta, J., Nuggeoda, D., Morrison, P. D., Miranda, A., Tang, M., & Clarke, B. O. (2016). Chemical pollutants sorbed to ingested microbeads from personal care products accumulate in fish. *Environmental Science & Technology*, 50(7), 4037–4044. <https://doi.org/10.1021/acs.est.5b06280>
- **(Moderate)**. Lee, H., Byun, D.-E., Kim, J. M., & Kwon, J.-H. (2018). Desorption of hydrophobic organic chemicals from fragment-type microplastics. *Ocean Science Journal*, 53, 631–639. <https://doi.org/10.1007/s12601-018-0051-x>

- **(Low).** Bejgarn, S., MacLeod, M., Bogdal, C., & Breitholtz, M. (2015). Toxicity of leachate from weathering plastics: An exploratory screening study with *Nitocra spinipes*. *Chemosphere*, 132, 114–119. <https://doi.org/10.1016/j.chemosphere.2015.03.010>
- **(Supplementary primary source outside the frozen baseline).** Otaka, T., Miyoshi, K., Komai, Y., & Nakayoshi, H. (1978). Safety evaluation of polyethylene terephthalate resins (I): Studies on the acute and subacute (3 months) toxicities by oral administration in rats. *Journal of the Food Hygienic Society of Japan*, 19(5), 431-442. <https://doi.org/10.3358/shokueishi.19.431>

Boundary case: tire rubber is not plastic - proven 6PPD-quinone leachate toxicity

This report evaluates plastics and microplastics. Tire rubber is an elastomeric material, not a plastic, and tire-wear particles are excluded from the microplastic category used here. This boundary case is included because tire wear is sometimes grouped with microplastics in environmental discussions and because it provides a strong example of why product-leachate toxicity must be attributed to the identified causal chemical rather than to a different material class.

Tian et al. 2021 chemically fractionated toxic roadway-runoff and tire-wear-particle leachate and identified 6PPD-quinone (6PPD-Q), an oxidation product of the tire antiozonant 6PPD, as the principal causal toxicant for acute coho salmon mortality. This is strong causal evidence that a chemical released from tire rubber can be acutely toxic to a sensitive aquatic species.

The absolute concentration scale in the original paper was subsequently revised after a commercial 6PPD-Q standard became available. Tian et al. 2022 re-tested juvenile coho and estimated an LC50 of 95 ng/L (95% CI 80-110 ng/L), approximately 8.3-fold lower than the original estimate. Revised roadway-runoff concentrations commonly remained at or above lethal levels. The recalibration therefore strengthened, rather than removed, the conclusion that 6PPD-Q is a potent aquatic toxicant for coho salmon.

Interpretive boundary: this evidence concerns a specific nonplastic tire-rubber additive transformation product in an aquatic exposure pathway. It does not demonstrate that rubber particles themselves have the same toxicity, does not establish human-health risk, and provides no evidence that plastic or microplastic particles are inherently toxic. In this report, the 6PPD-Q case is treated only as nonplastic boundary/context evidence and is excluded from the microplastic evidence count and plastic-toxicity conclusions. Importantly, the rubber additive 6PPD is not used in plastics.

- **(Nonplastic boundary/context source - excluded from microplastic evidence).** Tian, Z., Zhao, H., Peter, K. T., et al. (2021). A ubiquitous tire rubber-derived chemical induces acute mortality in coho salmon. *Science*, 371(6525), 185-189. <https://doi.org/10.1126/science.abd6951>
- **(Nonplastic boundary/context source - excluded from microplastic evidence).** Tian, Z., Gonzalez, M., Rideout, C. A., et al. (2022). 6PPD-Quinone: Revised Toxicity Assessment and Quantification with a Commercial Standard. *Environmental Science & Technology Letters*, 9(2), 140-146. <https://doi.org/10.1021/acs.estlett.1c00910>

26. Modeling Versus Measured Evidence

Models are essential for integrating incomplete observations, but their outputs are not measurements. Source inventories, river-emission models, ocean mass budgets, exposure models and lifetime-retention models depend on sparse inputs, size distributions, transport coefficients, uptake assumptions and boundary conditions.

Modeled estimates should be labeled as modeled, presented with sensitivity ranges and checked against measured mass balance. Apparent precision is not evidence of accuracy. Models are most valuable for identifying influential assumptions and designing measurements, not for replacing them. (Nor et al. 2021); (Meijer et al. 2021); (Borrelle et al. 2020); (Lebreton et al. 2019); (Weiss et al. 2021); (Eriksen et al. 2014)

Integrated evidence synthesis

Models are essential for integrating sparse measurements, testing scenarios and identifying influential parameters. Their outputs remain conditional on inputs and structure. A modelled global flux, stock or exposure estimate should be labelled as such and should not be described as a direct observation.

Microplastic models are particularly sensitive to particle-to-mass conversion. Count data require assumptions about size distribution, density and shape, while trawl or filter cutoffs omit part of the distribution. River-emission and ocean-stock estimates can differ substantially when those assumptions change (Borrelle et al. 2020; Weiss et al. 2021; Eriksen et al. 2014).

Scenario projections add uncertainty in waste generation, collection, informal disposal, behaviour, policy implementation and technology. They are useful for comparing assumptions but do not predict a single inevitable future. Uncertainty ranges should reflect data and structural uncertainty rather than only variation in fitted parameters.

Validation requires independent measurements at the scale and matrix of the prediction. Agreement with the same data used for calibration is not an independent test. Models should publish equations, parameter distributions and sensitivity analyses so that conclusions can be updated when measurements improve.

Evangelou et al. 2026 provides a direct global validation example. Three atmospheric inventories overpredicted size-aligned median measurements by two to four orders of magnitude. Measurement-constrained scaling reduced estimated global 5-100 μm emissions to 0.0045 Tg/year (90% interval 0.0006-0.0089), about two orders below a recent 0.32 Tg/year estimate. Land dominated particle-number emissions, while inferred mass depended strongly on size assumptions. Sparse ocean data, incomplete sources and heterogeneous methods remain important limitations.

Case study: optical measurements versus atmospheric burden

Liu et al. 2026 combined experimentally derived optical constants for selected coloured particles with FLEXPART-SBDART modeling and reported $0.039 \pm 0.019 \text{ W/m}^2$ of direct radiative forcing, equivalent to 16.2% of the modeled black-carbon forcing. The estimate was driven primarily by nanoplastics (0.033 W/m^2 versus 0.006 W/m^2 for microplastics) and assumed global column burdens of 0.017 and 0.075 mg/m^2 , respectively.

Those burden assumptions are conclusion-controlling. Maintaining the modeled 0.075 mg/m^2 microplastic burden with the paper's prescribed eight-day lifetime implies an annual global microplastic emission on the order of 1.75 Tg/year, whereas Evangelou et al. obtained a measurement-constrained 5-100 μm estimate of 0.0045 Tg/year. The size ranges are not identical and the comparison does not directly solve the nanoplastic term, but it demonstrates that the headline warming result is highly sensitive to an atmospheric inventory hundreds of times larger than the newer measurement-scaled estimate. The paper's optical measurements remain informative; the claimed real-world global warming contribution is not validated.

The evidence hierarchy used here gives measured data priority for occurrence and exposure, while using models to connect compartments or estimate quantities that cannot yet be measured. The two evidence types are complementary when their boundaries remain explicit.

Strong optical absorption does not establish a large climate effect

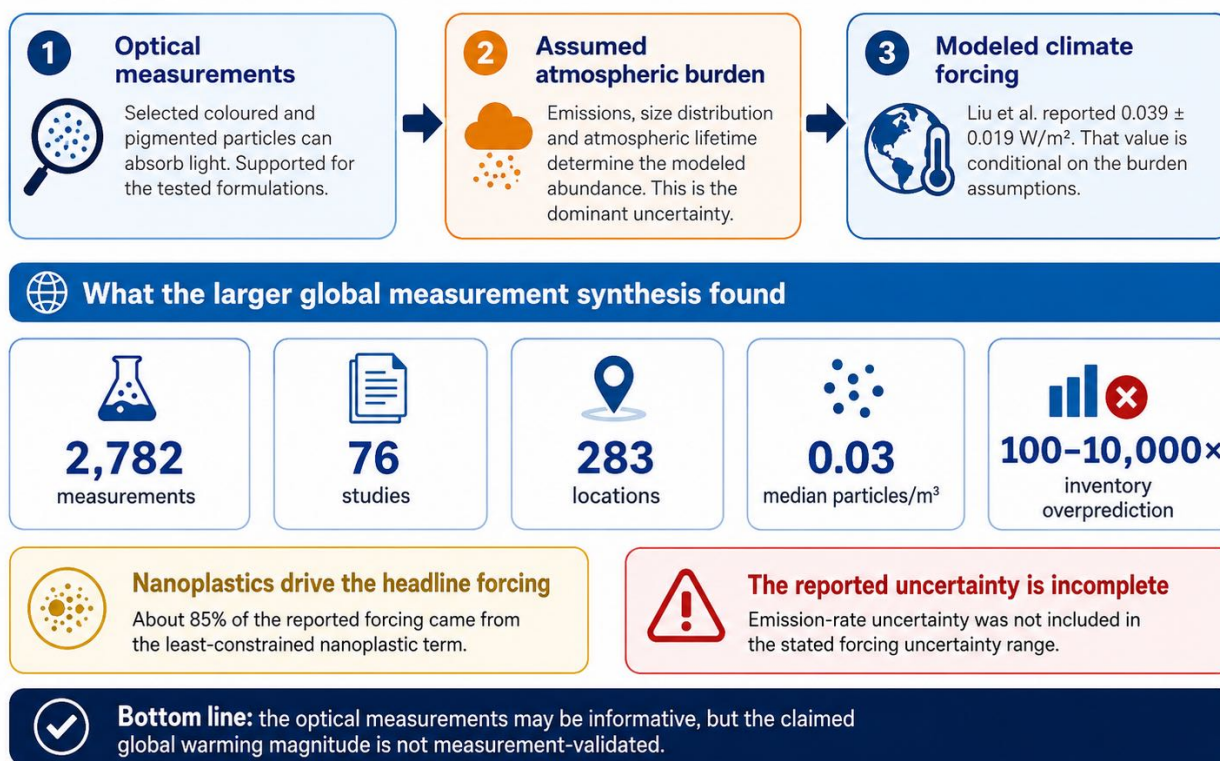


Figure 18A. Strong optical absorption does not by itself establish a large climate effect. The forcing estimate remains conditional on the modeled atmospheric burden. Evangelou et al. 2026 compiled 2,782 measurements from 76 studies at 283 locations, reported a global median of 0.03 particles/ m^3 , and found existing inventories two to four orders of magnitude above size-aligned observations.

Selected evidence records

- **(Supplementary source outside the frozen baseline).** Evangelou, I., Bucci, S., & Stohl, A. (2026). Atmospheric microplastic emissions from land and ocean. *Nature*, 649, 1186–1189. <https://doi.org/10.1038/s41586-025-09998-6>
- **(Moderate).** Liu, Y., Fu, H., Zhang, H., et al. (2026). Atmospheric Warming Contributions from Airborne Microplastics and Nanoplastics. *Nature Climate Change*. <https://doi.org/10.1038/s41558-026-02620-1>
- **(Not scored - secondary synthesis).** Nur Hazimah Mohamed Nor, Merel Kooi, Noël J. Diepens, and Albert A. Koelmans. 2021. Lifetime Accumulation of Microplastic in Children and Adults.
- **(Moderate).** Meijer, L.J.J., van Emmerik, T., van der Ent, R., Schmidt, C., & Lebreton, L. (2021). More than 1000 rivers account for 80% of global riverine plastic emissions into the ocean. *Science Advances*, 7, eaaz5803. <https://doi.org/10.1126/sciadv.aaz5803>
- **(Moderate).** Borrelle, S. B., Ringma, J., Law, K. L., et al. (2020). Predicted growth in plastic waste exceeds efforts to mitigate plastic pollution. *Science*, 369, 1515–1518. <https://doi.org/10.1126/science.aba3656>
- **(Moderate).** Lebreton, L. C. M., van der Zwet, J., Damsteeg, J.-W., Slat, B., Andrady, A., & Reisser, J. (2019). A Global Mass Budget for Positively Buoyant Macroplastic Debris in the Ocean. *Scientific Reports*. <https://doi.org/10.1038/s41598-019-49413-5>
- **(Moderate).** Weiss, L., Ludwig, W., Heussner, S., et al. (2021). The missing ocean plastic sink: Gone with the rivers. *Science*, 373, 107–111. <https://doi.org/10.1126/science.abe0290>
- **(Moderate).** Eriksen, M., Lebreton, L. C. M., Carson, H. S., et al. (2014). 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. <https://doi.org/10.1371/journal.pone.0111913>

27. Removal Versus Destruction

Removal means transfer from one stream or compartment to another. Filtration, sedimentation, flotation, coagulation and sludge capture may reduce effluent concentration without destroying polymer. Destruction requires verified depolymerization, oxidation or mineralization with an explicit carbon mass balance and identification of by-products.

Claims of remediation should therefore specify capture efficiency, residual size distribution, final destination and whether particle number changed through fragmentation. Biodegradation claims require evidence of chemical conversion rather than loss from the visible or measured size window. (Talvitie et al. 2017); (Michielssen et al. 2016); (Akarsu et al. 2021); (Napper et al. 2019); (Bagheri et al. 2017)

Integrated evidence synthesis

Treatment performance should be expressed as a mass balance whenever possible. Percentage removal can be inflated by low influent counts, selective sampling or loss below the detection limit. Reporting influent, effluent, sludge, concentrate and uncertainty is more informative than a single percentage (Talvitie et al. 2017; Michielssen et al. 2016).

Capture technologies transfer particles to another medium. Filters require replacement or regeneration; coagulation generates sludge; membranes generate concentrate; flotation and electrocoagulation create residuals. Environmental benefit depends on whether the captured material is securely managed and on the energy and chemical burden of the process.

Biodegradation claims require evidence of polymer-chain cleavage and ultimate conversion. Weight loss, surface pitting, embrittlement or reduced molecular weight may indicate deterioration but can coexist with production of smaller persistent fragments. Carbon dioxide evolution, dissolved products and carbon recovery are needed to distinguish mineralization from fragmentation (Napper et al. 2019; Bagheri et al. 2017).

Source prevention may be preferable when a release pathway is well defined, but substitution can create other material or lifecycle burdens. Engineering decisions should compare total environmental performance, including function and unintended consequences, rather than optimize only a microplastic count at one outlet.

Evidence-weighted interpretation

Engineering studies often report removal from a sampled water phase. That endpoint includes capture, settling, adsorption, membrane retention and transfer to sludge or concentrate. It should not be described as destruction unless the polymer carbon is shown to be mineralized or converted into identified products with a closed mass balance (Talvitie et al. 2017; Michielssen et al. 2016; Akarsu et al. 2021).

The same distinction applies to biodegradation claims. Visual deterioration, loss of tensile strength, surface pitting or smaller fragments can indicate ageing while increasing the number of microplastic particles. Long-duration environmental studies of conventional and marketed biodegradable materials show that receiving environment and formulation govern persistence (Napper et al. 2019; Bagheri et al. 2017).

Selected evidence records

- **(Moderate)**. Talvitie, J., Mikola, A., Koistinen, A., & Setälä, O. (2017). Solutions to microplastic pollution – Removal of microplastics from wastewater effluent with advanced wastewater treatment technologies. *Water Research*, 123, 401–407. <https://doi.org/10.1016/j.watres.2017.07.005>
- **(Moderate)**. Michielssen, M. R., Michielssen, E. R., Ni, J., & Duhaime, M. B. (2016). Fate of microplastics and other small anthropogenic litter (SAL) in wastewater treatment plants depends on unit processes employed. *Environmental Science: Water Research & Technology*, 2, 1064–1073. <https://doi.org/10.1039/C6EW00207B>
- **(Moderate)**. Akarsu, C., et al. (2021). Removal of microplastics from wastewater through electrocoagulation–electroflotation and membrane filtration processes. *Water Science and Technology*, 84(7), 1648–1662. <https://doi.org/10.2166/wst.2021.356>
- **(Moderate)**. Napper, I. E., & Thompson, R. C. (2019). Environmental deterioration of biodegradable, oxo-biodegradable, compostable and conventional plastic carrier bags in the sea, soil and open-air over a 3-year period. *Environmental Science & Technology*, 53(9), 4775–4783. <https://doi.org/10.1021/acs.est.8b06984>

- **(Moderate)**. Bagheri, A. R., Laforsch, C., Greiner, A., & Agarwal, S. (2017). Fate of so-called biodegradable polymers in seawater and freshwater. *Global Challenges*, 1, 1700048. <https://doi.org/10.1002/gch2.201700048>

28. Risk Characterization

Risk characterization integrates occurrence, external exposure, internal dose, hazard, adversity and causal certainty. Plastic particles are found widely because dust and particles are found widely; ubiquity is expected and does not establish exceptional hazard. The version-controlled evidence supports occurrence and selected high-dose experimental effects, but no reliable causal evidence demonstrates adverse human health outcomes from ordinary general-population microplastic exposure. The integrated evidence weighs against a material public-health risk and against treating ordinary plastic dust as a special category.

Confidence is highest for methodological conclusions and for the distinction between detection and harm. Confidence is moderate for the conclusion that most ingested mass is cleared, because human kinetics are sparse. Confidence is limited for universal temporal trends and for quantitative population risk. Specific ecological risks may be higher in local hotspots or for susceptible species, but they should not be generalized across all polymers, sizes and environments.

The conclusion stress test identifies 15 major conclusions. Two are graded Established, ten Strongly supported, two Moderately supported and one Limited evidence. No conclusion is graded as proof of zero risk. The central finding is evidentiary proportionality: claims should not exceed the strength, dose relevance and causal reach of the underlying studies.

The quantitative layer strengthens the conclusion that ordinary modeled external mass exposure is remote from selected administered toxicology doses, extending to approximately 600 million-fold for the formal 5,000 mg/kg/day cellulose-acetate NOAEL, while preserving the limitations that prevent a universal safety claim. Quantitative human absorption, size-resolved internal dose and long-term retention remain insufficiently measured. Measurement-constrained atmospheric evidence also shows that available inventories can overpredict observed concentrations by two to four orders of magnitude. No conclusion-strength grade changed after the quantitative reconciliation, thermoplastics source recovery, atmospheric validation or Lane et al. human-biomonitoring audit.

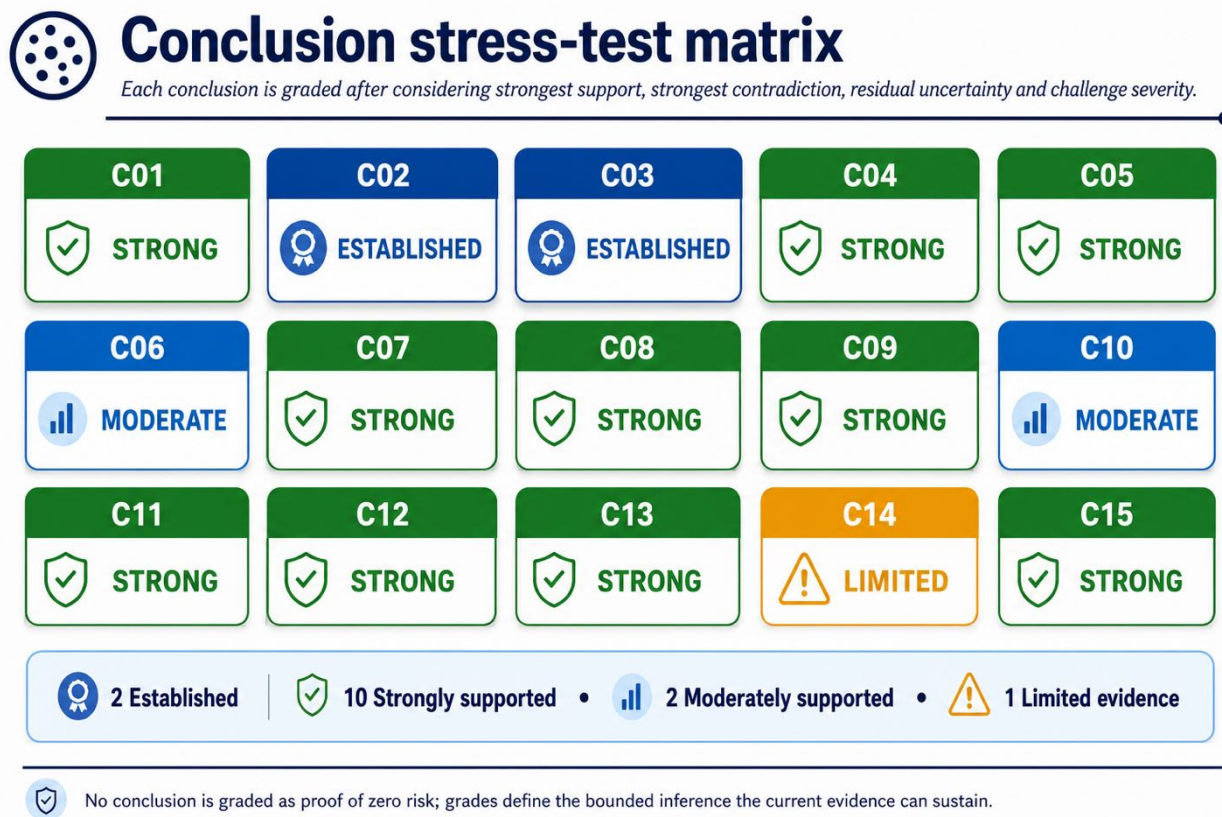


Figure 19. Conclusion-strength grades after stress testing.

The independent Lane et al. 2026 audit strengthens the human-evidence boundary: none of 133 human tissue or bodily-fluid datasets met all essential QA/QC criteria for direct quantitative risk assessment. This does not invalidate every occurrence finding or make risk unknowable; it means current biomonitoring cannot by itself define representative population body burden, organ accumulation or a particle-specific human dose-response.

Atmospheric model validation strengthens a parallel boundary. Evangelou et al. found a global measured median of 0.03 particles/m³ and inventories two to four orders of magnitude above size-aligned measurements. Liu et al. provides useful colour-dependent optical measurements, but its 0.039 W/m² forcing estimate depends on atmospheric plastic burdens comparable with two-thirds of black-carbon mass and is dominated by extrapolated nanoplastics. It is therefore retained as a model-dependent scenario, not as a demonstrated material climate forcing.

Integrated evidence synthesis

Risk characterization follows a causal sequence: reliable identification, external exposure, uptake or deposition, internal dose, biological interaction, adverse effect and population consequence. Confidence decreases when a link is inferred rather than measured. The corpus is strongest on occurrence and analytical limitations, less complete on external dose, and weakest on quantitative human internal dose and causal disease outcomes.

For human health, the evidence supports exposure and possible limited uptake but not a general causal disease conclusion. High-dose laboratory effects establish biological possibility, as they do for many poorly soluble particles. Standardized no-adverse-effect evidence and the large separation between modeled external mass exposure and selected administered doses strongly constrain broad high-risk claims. The contrast with established particulate hazards is informative: quartz, occupational wood dust and certain toxic metal compounds have demonstrated human disease evidence, whereas ordinary plastic dust does not. Uncertainty about smaller particles, chronic exposure or formulation details does not itself create a high-risk signal.

For ecology, risk is more context-dependent. Local concentrations, organism feeding mode, fibres, larger debris and whole-product chemistry can create credible hazards. The main limitation is extrapolation from laboratory concentration and individual biomarkers to field populations. Risk should therefore be assessed by matrix, species and source rather than assigned to microplastics as a single substance.

Uncertainty is not a neutral placeholder that automatically favors danger. Method error can produce false positives and false negatives, and limited data can conceal a small effect or overstate one through marker artefacts. This report distinguishes three states: no relevant evidence, weak evidence unable to bound risk, and informative evidence that constrains the magnitude of a plausible effect. The current conclusion rests on the third state for a large or material ordinary-exposure effect, while remaining less certain for subtle effects, the smallest particles, chronic inhalation and unusual source-specific exposure.

A proportionate risk-management interpretation is to rank particulate hazards by validated exposure and demonstrated toxicity. Detectability, analytical uncertainty or material novelty alone is insufficient to establish high priority. Special controls are justified for demonstrated hazards, specific toxic constituents, occupational exposures and local hotspots; they are not justified merely because a particle is plastic. Interventions should also be evaluated against source reduction, substitution, lifecycle and residual-stream trade-offs.

The Hayden et al. 2023 lung-particle study does not alter the microplastics risk conclusion because it neither measures plastic exposure nor tests microplastic health effects. Its importance is contextual: 124,018 inorganic particles spanning 45 classes were characterized in 38 selected lung-biopsy specimens, demonstrating directly that material-specific tissue detections occur within a large and chemically diverse nonplastic particulate background. The study cannot supply a plastic-to-nonplastic ratio because its FE-SEM/EDS method was qualitative and preferentially detected inorganic particles. It nevertheless shows why plastic detections should not be interpreted in isolation. A scientifically meaningful assessment must ask how the plastic fraction, dose and potency compare with the broader particle mixture and whether plastic contributes disproportionate harm, rather than treating detection of one material as intrinsically exceptional.

Miller et al. 2017 likewise does not overturn the ordinary-exposure conclusion. It supplies high-value positive kinetic evidence that deliberately inhaled ultrasmall nanoparticles can enter the circulation and localize preferentially in diseased vascular tissue. Importantly, this localization is not evidence that the particles caused the disease: in the human arm, gold was administered only 24 hours before removal of long-established carotid plaques, and in the mouse arm repeated inert-gold dosing did not increase plaque size. The same paper also

constrains overinterpretation: the corrected illustrative systemic-transfer estimate was approximately 0.02%, transfer fell strongly with increasing particle size, and the human plaque arm included only three exposed patients. For microplastics, the defensible inference is that translocation and deposition at diseased sites cannot be categorically excluded for sufficiently small particles, but finding particles in plaque or another diseased vascular structure is evidence of co-location, not causation; quantitative extrapolation from 4-5 nm gold to micrometre-scale plastics is not supported.

Risk communication is not biologically neutral. Negative expectations and alarming framing can generate nocebo symptoms and distress, including adverse experiences without a corresponding toxic exposure mechanism (Colloca & Barsky 2020). This does not mean that every reported symptom is psychological or that uncertainty should be concealed. It means that communication should be accurate and proportionate: when the evidence weighs against material risk, repeatedly presenting ordinary plastic-particle detection as a hidden threat can itself create avoidable harm.

Table 17. Evidence basis for the ordinary-exposure human-health conclusion

Evidence line	Current result	Inference supported	Main boundary / strongest contradiction
External exposure	Nor et al. provides an integrated modeled mass/count estimate; Heo et al. documents extreme heterogeneity among 76 count-based dietary studies.	Exposure estimates are method- and pathway-bounded and cannot be collapsed into one universal value.	Incomplete pathways and lower-size fractions; Yadav and Song use incompatible mass constructs.
Kinetics and clearance	Most ingested material is eliminated; retained human microplastic burden is not well quantified and is expected to be size-dependent. Controlled inhalation of ~4-5 nm gold nanoparticles demonstrates that a very small fraction of ultrasmall particles can cross the pulmonary barrier; the corrected Miller et al. illustrative 24-h estimate is ~0.02% of inhaled mass.	External intake should not be equated with absorbed or retained dose. Barrier crossing is biologically plausible for ultrasmall nanoparticles and is strongly size-dependent.	Gold was an engineered inert tracer under controlled exposure; the estimate was not a closed mass balance, and transfer fell sharply with size. It is not a microplastic absorption fraction and does not justify extrapolation to micrometre-scale particles.
Animal toxicology	Selected stronger studies report no detected adverse effects at a formal study NOAEL of 25 mg/kg/day for weathered fragmented PP, 1,000 mg/kg/day for PA-6, approximately 1,600-2,000 mg/kg/day of each PE/PET component administered together, and at least 2,000 mg/kg/day for fragmented PP and PTFE. Additional archival/control-diet evidence reports standalone PE at 5% of diet for 90 days and VCM-free PVC at 10% of diet across a lifespan design, but neither establishes a verified formal polymer NOAEL. No verified repeated-dose oral NOAEL was identified for real fragmented/weathered PS. Positive studies occur under selected conditions.	Current evidence does not demonstrate exceptional generic potency near ordinary exposure.	Sánchez 2026 reports intestinal effects at 1 mg/kg/day for one PVC preparation; Lu 2025 reports PET liver effects at 50-500 µg/day; Lewis 2026 reports mild acute effects after 5 or 50 mg/kg; lower PE/PET lung findings remain study-limited and discordant. PS lacks a source-verified repeated-dose point of departure.
Human evidence	No study demonstrates ordinary-exposure disease causation. Tran et al. found 25 living-human studies, including 11 rated low ROBINS-E risk, but confounding and exposure measurement remain major concerns. Lane et al. independently found that none of 133 human tissue or bodily-fluid datasets met all 14 essential QA/QC criteria for direct quantitative risk assessment.	Current evidence does not support classifying ordinary general-population exposure as a material public-health risk.	Recent CRC, cardiovascular, CSF and brain associations require replication with validated exposure and temporality. The Lane framework is deliberately strict and does not imply that every detection is false.
Plastic specificity	Generic particle mechanisms are shared; matched realistic-dose comparator evidence of a generic plastic excess is absent.	A plastic label alone does not establish excess hazard beyond other particles.	Formulation, additives, shape, ageing and biopersistence can produce source-specific differences.
Integrated conclusion	Occurrence is established; causal evidence at ordinary exposure is not. Positive associations and experimental hazards remain method-, dose- and design-bounded.	No study has established a causal relationship between ordinary general-population microplastic exposure and disease in humans.	Conditional conclusion; replicated prospective exposure-response evidence with validated exposure and temporality would be conclusion-changing if it emerges.

Current agency positions — scope and evidence-base limitations

Agency statements are reported within their exact scope and date. They are not collapsed into a claim of unanimous agreement. A research-prioritization or contaminant-listing action is not equivalent to a finding that harm has been demonstrated; conversely, a conclusion that current evidence does not demonstrate risk is not a claim that every uncertainty is resolved.

Table 18. Current official positions on microplastics and human health

Organization and document	Current position and scope	Main limitation and pending work
U.S. FDA Microplastics and Nanoplastics in Foods; current page 24 July 2024; 2026 Human Foods Program priorities	Scope: Foods and food exposure. Position: Current scientific evidence does not demonstrate that detected food levels pose a human-health risk. In 2026 FDA prioritized accurate, reproducible detection, quantification and characterization methods.	Limitation: Method and occurrence uncertainty remain; the position is not a conclusion about every route. Method-development priority is not itself a finding of harm or a completed quantitative risk assessment.
WHO Dietary and inhalation exposure to nano- and microplastic particles; 30 August 2022	Scope: Food, water and air; evidence through December 2021 Position: Reviewed available evidence and identified major uncertainties and research needs.	Limitation: Evidence and methods were insufficient for broad definitive conclusions across all particles and routes. Pending: Future work and research priorities defined.
EFSA European Parliament request for scientific advice; 9 December 2025	Scope: Potential health risks from microplastics in food, water and air Position: A new scientific opinion is underway; current evidence remains marked by major knowledge gaps.	Limitation: No current complete assessment should be represented as final. Pending: Scientific opinion expected by end of 2027.
U.S. EPA draft CCL 6; announced 2 April 2026; CCL pages current through June 2026	Scope: Drinking-water information collection and regulatory prioritization. Position: Microplastics are included as a group in draft CCL 6; EPA is developing methods and filling health/occurrence data gaps.	Limitation: A CCL listing imposes no requirements and is not a regulatory determination. Pending: SAB/public process, final CCL 6 and later regulatory-determination decisions for contaminants with sufficient data.
Health Canada / ECCC Science Assessment of Plastic Pollution; 2020	Scope: Environmental and human-health state of science Position: No human-health concern identified at that time, but data were insufficient for robust quantitative assessment.	Limitation: Evidence base predates many recent studies and was not a formal chemical risk assessment. Pending: Further exposure, toxicology and method research recommended.
German BfR current communications; 11 November and 2 December 2025	Scope: Consumer human-health risk. Position: Current scientific knowledge indicates relatively low risk; tissue detections and disease correlations do not establish a causal link.	Limitation: Small samples, method uncertainty, lack of harmonized protocols and uncertain long-term nanoplastic effects. Pending: Continued method, kinetic and mechanistic research.
UK COT Statement on potential risks from inhalation of microplastics; 2024	Scope: Inhaled micro- and nanoplastics Position: A full inhalation risk assessment could not be performed from the available evidence.	Limitation: No epidemiological or controlled-dose human inhalation studies; limited kinetics and non-standardized methods. A full quantitative inhalation risk assessment could not be completed; this is not a finding that ordinary exposure causes harm. Pending: Harmonized methods, representative exposure, toxicokinetics and route-relevant toxicology.

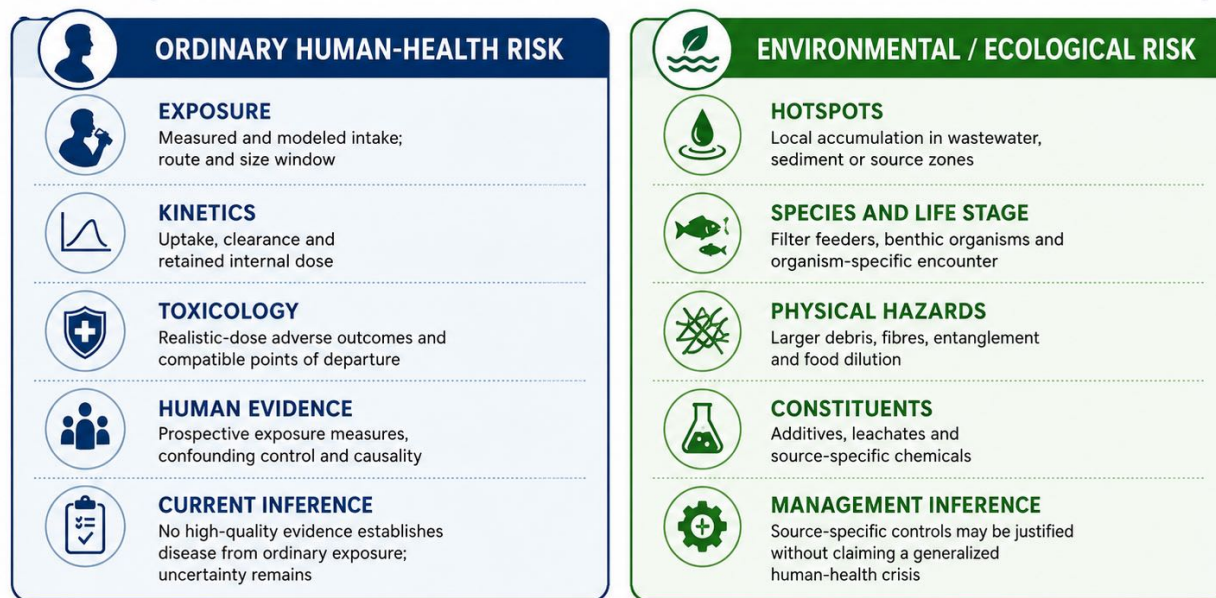
Two distinct risk questions

Ordinary human-health exposure and environmental/ecological management are related but not interchangeable. Evidence from one question must not silently stand in for the other.



TWO DISTINCT RISK QUESTIONS

HUMAN-HEALTH CONCLUSIONS AND ECOLOGICAL MANAGEMENT QUESTIONS
REQUIRE DIFFERENT EVIDENCE



**DO NOT USE HUMAN ORAL-DOSE COMPARISONS
AS A SUBSTITUTE FOR ECOLOGICAL RISK ANALYSIS.**

The questions overlap in occurrence and source control,
but their endpoints, comparators, dose metrics and
decision criteria are not interchangeable.

Figure 20. Two distinct risk questions. Human-health conclusions require exposure, uptake, retained dose and causal evidence. Ecological decisions require species-, habitat-, concentration- and source-specific analysis. A bounded human-health conclusion does not dismiss local ecological or larger-debris harms.

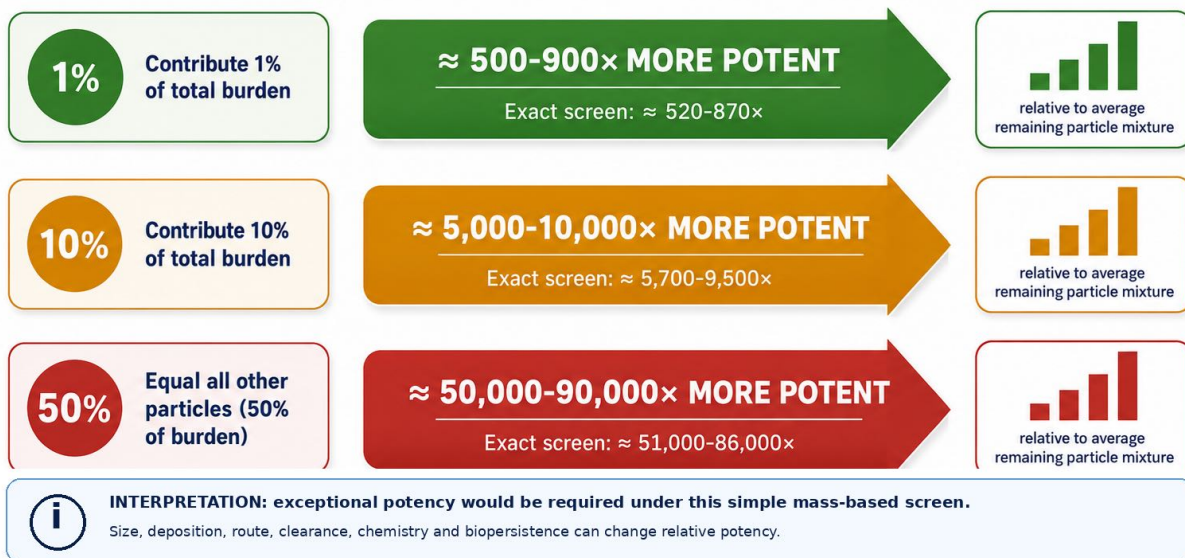
Cumulative weight-of-evidence interpretation

The intensity of scientific and public attention should be proportional to demonstrated risk. In the present evidence system, plastic is a small to trace fraction in the selected compatible particle comparisons, the evaluated common-polymer preparations generally show low toxicity under the tested conditions, selected study-specific highest doses without detected adverse effects extend from 25 to 5,000 mg/kg/day and are remote from modeled ordinary exposure, typical measured outdoor atmospheric occurrence is very low within the included analytical windows, and no High-quality human study demonstrates realistic-dose disease causation. More than five decades of evidence therefore support a stable conclusion: ordinary plastic dust is not a demonstrated special public-health hazard.



How potent would plastic need to be to materially affect total particle burden?

SCREENING HEURISTIC — assumes equal potency per unit mass; NOT A FORMAL RISK ESTIMATE



Derived from the Mohamed Nor/EPA 0.0012-0.0019% comparison; exact values depend on the burden-share formulation.

Figure 21. Screening heuristic using the bounded 0.0012-0.0019% mass comparison. Under the explicit simplifying assumption of equal potency per unit mass, plastic would require approximately 520-870-fold greater potency to contribute 1% of total burden, 5,700-9,500-fold to contribute 10%, and 51,000-86,000-fold to equal all other particles combined. This is not a formal risk estimate; size, deposition, route, clearance, chemistry and biopersistence can alter relative potency.

Cumulative weight-of-evidence updating clarifies why the conclusion should be stable but revisable. Relevant null findings, kinetic evidence and study-specific high-dose findings without detected adverse effects reduce the plausibility of a large hidden ordinary-exposure effect. A new high-dose cell study, detection report or cross-sectional association has limited power to reverse the integrated conclusion. A replicated study demonstrating confirmed intact particles, realistic exposure, measured internal dose and causal adverse outcomes would have high evidentiary weight and would appropriately change that conclusion.

CUMULATIVE WEIGHT-OF-EVIDENCE UPDATING

INFERENCE IS BUILT ON ACCUMULATING RELEVANT, HIGH-QUALITY EVIDENCE — NOT PUBLICATION COUNT

Evidence quality, relevance and independence determine how much a new result changes the conclusion.
Each line of evidence is assessed against key scientific criteria and integrated to reach a conclusion.

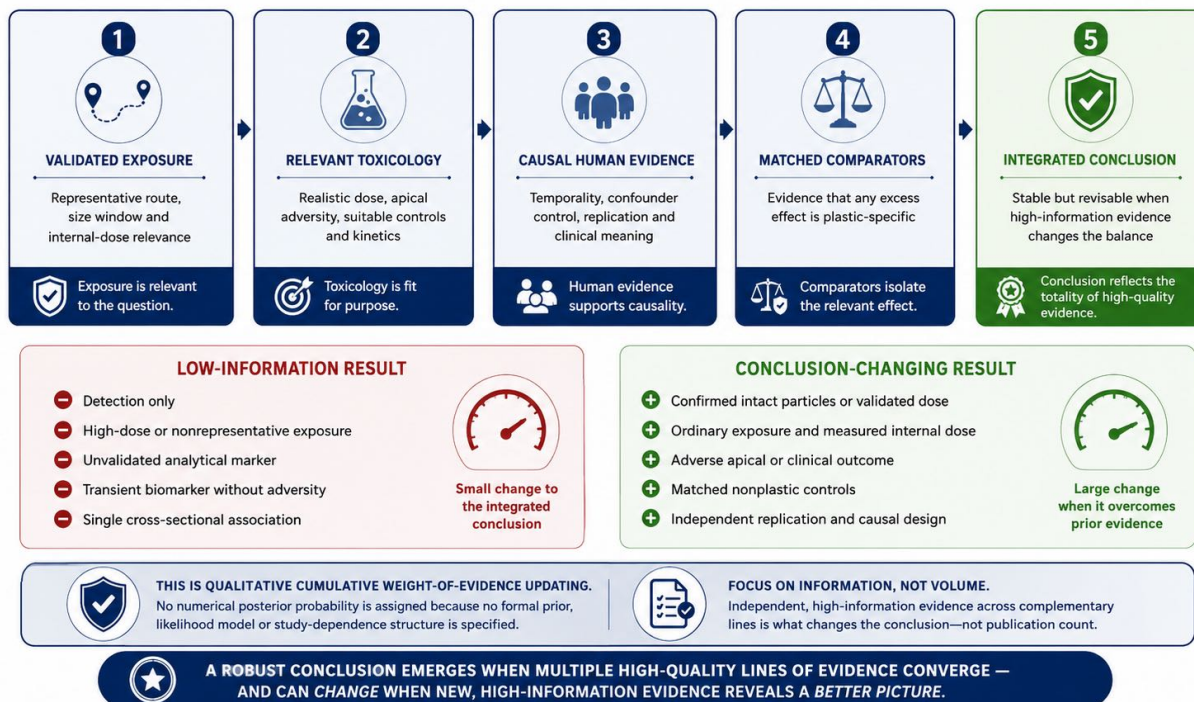


Figure 22. Cumulative weight-of-evidence updating. Relevant evidence is weighted by analytical validity, exposure realism, causal informativeness and independence. Publication count alone is not evidence, and no numerical probability is assigned. A large hidden ordinary-exposure effect becomes less plausible as sufficiently sensitive evidence accumulates without detecting it.

The current risk characterization is therefore stronger than 'unknown.' The evidence is sufficient to reject the claim that ordinary microplastic exposure should presently be treated as a demonstrated, material or uniquely plastic public-health threat. Plastic is found widely because particles are found widely; very high-dose effects are possible because high-dose particle effects are generic; and no evidence establishes exceptional potency at ordinary exposure. The residual possibility of a subtle, subgroup-specific or currently unmeasured effect is not equivalent to evidence of material danger and does not justify disproportionate worry.

Evidence-weighted interpretation

Risk characterization integrates a source, an exposure pathway, an internal or target-site dose, a dose-response relationship and an adverse outcome. The present evidence is strongest at the first step - occurrence - and progressively weaker through total human exposure, absorption, retained dose and disease causation. A large occurrence literature cannot substitute for missing kinetic and causal links.

For human health, the database supports ordinary ingestion and inhalation, selected plausible tissue detections, experimental hazard at some doses and important no-adverse-effect findings at far higher administered masses. No reliable causal evidence demonstrates adverse human health outcomes from ordinary general-population exposure. A precise upper bound for every possible subtle effect cannot be calculated, but the integrated evidence weighs against a material public-health risk under the defined conditions.

For environmental management, source controls can be justified by persistence, litter prevention, operational efficiency or local ecological evidence without claiming universal toxicological catastrophe. The most defensible interventions are those with measurable source reduction, low substitution burden and a complete accounting of residual streams.

29. Major Evidence Gaps

The most decision-relevant gaps are representative route-specific exposure measurements, validated methods for the smallest particles and biological matrices, quantitative human kinetics, realistic chronic inhalation studies, causal epidemiology, matched-comparator toxicology and field ecological studies. Research priority should reflect plausible exposure, hazard, uncertainty reduction and ability to change a decision rather than analytical novelty alone. The Nor et al. 2021/EPA comparison indicates a small modeled ordinary mass relative to one independently derived particulate denominator, but that cross-domain calculation does not determine research priorities by itself. Targeted work remains justified for credible source-specific high exposures, susceptible populations or mechanisms capable of exceptional potency.

A newly identified study should interrupt report production only if it plausibly changes a major conclusion: credible causal evidence of harm at realistic exposure; a materially revised exposure estimate large enough to alter the proportionality analysis; a validated toxicity threshold near real exposure; a reproducible plastic-specific effect materially greater than matched nonplastic particles; a major method validation or invalidation that changes a substantive conclusion; or a correction or retraction. Mere detection at a smaller size or characterization of an unusual formulation is not a stopping condition.

Integrated evidence synthesis

The highest decision value comes from research that places plastic particles within the complete particulate mixture, identifies dominant contributors to exposure and disease, and tests whether the plastic fraction has an effect beyond matched particles. Established particulate hazards provide a comparative benchmark, not a reason to discontinue microplastic research. Human kinetics, chronic inhalation and nanoplastic studies are most informative when linked to validated exposure, a plausible internal dose and a decision-relevant adverse endpoint.

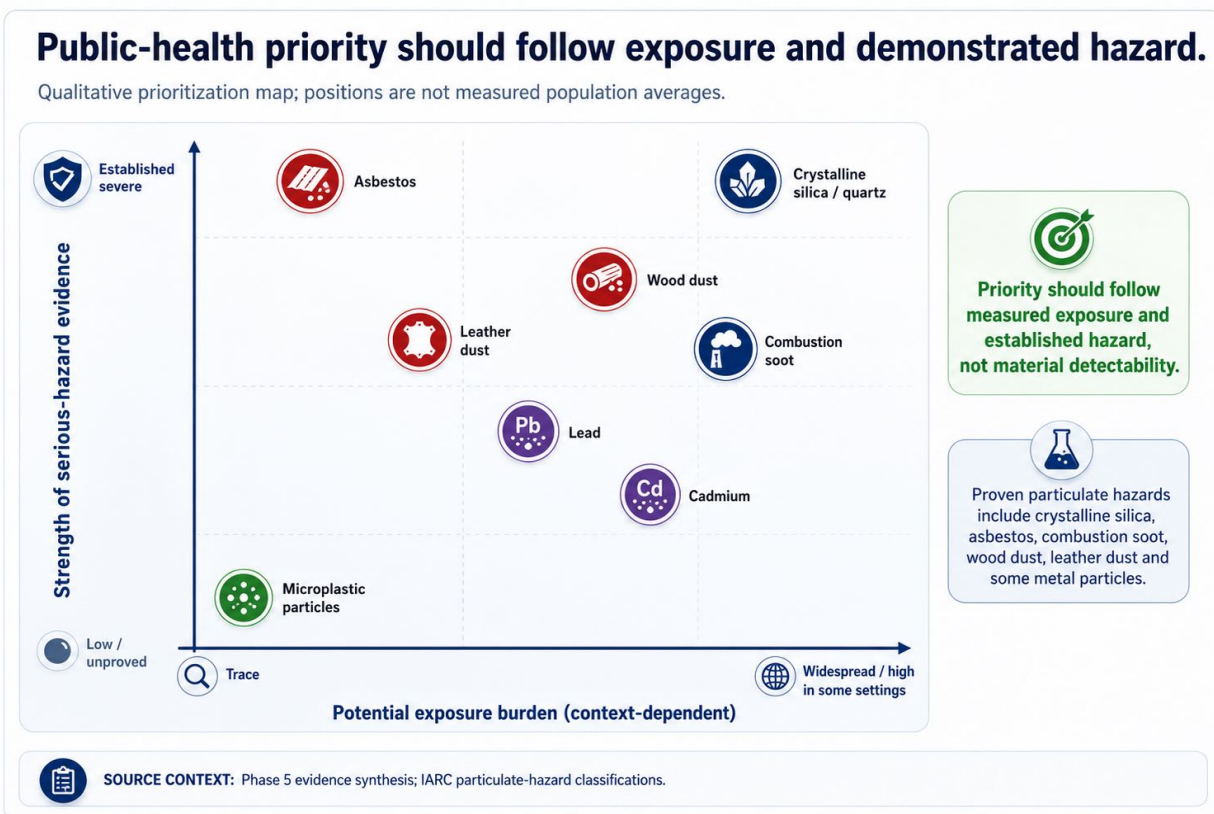


Figure 23. Conceptual prioritization map: public-health attention should follow measured exposure and established serious hazard. Positions are qualitative and context-dependent, not measured cross-population averages.

Analytical validation is useful when it tests a consequential claim and prevents false positives. Blind interlaboratory proficiency testing, matrix-specific recovery and interference studies, complete blanks and orthogonal confirmation can determine whether reported tissue or nanoscale signals are real. The purpose is evidentiary adjudication, not

an assumption that detecting ever-smaller plastic particles creates a health priority. Nanoplastic claims require chemical specificity and demonstrated matrix performance; uncertainty or a DLS size peak is not a risk signal.

Plastic-specific toxicology is most informative when it begins with a defined material, credible exposure pathway and mechanistic hypothesis capable of producing a material effect at realistic dose. Where concern is driven by an additive, leachate, pigment, filler or reactive degradation product, the constituent and its realistic dose should be evaluated separately. Tire-rubber chemicals are outside the plastics scope and should be assessed explicitly as nonplastic constituents. Any experiment intended to support particle-specific causation requires matched nonplastic comparators, complete characterization, route-appropriate dosing and independent replication.

Microplastic epidemiology requires validated exposure markers and should not assume that analytical detectability implies clinical importance. Prospective studies become decision-relevant when exposure precedes outcome, the metric has demonstrated specificity and reproducibility, confounding is controlled, and the expected effect is biologically plausible at the measured dose. Cross-sectional biomarker associations remain hypothesis-generating and should not be used alone for quantitative risk estimates.

Decision-relevance stopping rule for evidence updates

The objective is not to accumulate an unlimited number of plastic-specific occurrence, tissue-detection or high-dose screening studies. Under a decision-relevance stopping rule, routine additions are logged for the next version-controlled database update unless they plausibly change a major exposure estimate, invalidate or validate a conclusion-controlling method, establish an adverse effect near ordinary internal dose, demonstrate reproducible plastic-specific excess potency using matched comparators, or materially alter causal human evidence. This prevents publication volume from substituting for information value.

Evidence-weighted interpretation

Evidence gaps should be ranked by their expected reduction of consequential uncertainty and their capacity to prevent human disease or ecological harm. High-value work includes representative total-particle exposure characterization, validated biological-matrix methods, realistic route-specific kinetics and toxicology, evaluation of specific hazardous constituents or unusual source exposures, matched-particle tests of plastic specificity and field studies capable of resolving population-level ecological effects. Incomplete data alone do not establish priority, but neither should uncertainty be treated as evidence that further work lacks value.

A useful new study should state in advance what conclusion it could strengthen or overturn. It should also report null results, complete methods, blanks, recoveries, particle characterization, raw data and predefined analysis. Without these elements, a larger literature may increase publication count without reducing uncertainty.

Table 19. Decision-relevant allocation of research priority

Priority	Study design needed	Decision improved	Minimum control
1. Total particulate burden and composition	Compatible mass-, count- and size-resolved measurement of the whole particle mixture	Identifies dominant exposure and hazard contributors	Same matrix, size window and denominator for plastic and nonplastic particles
2. Established particulate hazards	Exposure-response and intervention studies for silica, asbestos, combustion soot, hazardous metals, wood dust, leather dust and other proven hazards	Reduces demonstrated disease burden	Substance-specific exposure and clinically relevant endpoints
3. Extraordinary plastic-specific hypothesis	Study only after credible preliminary evidence of realistic exposure and exceptional potency	Tests whether a plastic effect could be material despite trace exposure	Realistic dose, matched particles, constituent separation and independent replication

Priority	Study design needed	Decision improved	Minimum control
4. Specific hazardous constituent or high-exposure source	Targeted chemistry and dosimetry for additives, leachates, pigments, fillers, tyre-rubber chemicals or unusual occupational releases	Attributes risk to the actual agent and source	Measured constituent dose and appropriate substance-specific comparator
5. Analytical adjudication of consequential claims	Blind interlaboratory testing and orthogonal confirmation for tissue or nanoscale claims	Prevents false-positive evidence from driving policy	Full blanks, recovery, raw data and no inference from detection to harm
6. Full-system intervention analysis	Compare source control, substitution, lifecycle burden and residual streams	Prevents risk transfer and misallocation of resources	Total environmental and health impact, not plastic reduction alone

30. Conclusions

Dust and particles are ubiquitous and unavoidable. Plastic particles are found in air, water, food, soil, household dust and selected human samples for the same basic reason that mineral, biological, cellulosic, combustion-derived and metal-containing particles are found there: the world is particle-rich. Across compatible comparisons, plastic is a small to trace component of that background. No study has established a causal relationship between ordinary general-population microplastic exposure and disease in humans, and current evidence does not support classifying that exposure as a material public-health risk. This conclusion does not cover occupational dusts, unusual source hotspots, damaged medical devices, fires, intentional administration or extreme experimental doses.

Recent studies strengthen the evidence that particles or polymer-associated signals can be detected in human matrices and can correlate with disease or biomarkers. Tran et al. 2026, Xu et al. 2025, Nihart et al. 2025, Monikh et al. 2025 and the Campen et al. 2025 reply, He et al. 2025, Li et al. 2026a-b, Chen et al. 2026, Fang et al. 2026, Lu et al. 2026 and Paolisso et al. 2026 deserve explicit weight. They do not, however, establish that ordinary exposure preceded and caused the observed disease. Exposure measurement, reverse causation, confounding, selected clinical populations and matrix-specific analytical validity remain the decisive boundaries.

1. Plastic is a minor component of the total particle burden. Nor et al. 2021 provides the principal integrated particle-resolved human benchmark identified in this review: 883 particles/day and 583 ng/day over its modeled 1-5,000-micrometre domain. The corresponding 0.0012-0.0019% comparison is limited to EPA adult soil-plus-dust ingestion alone and is not a universal composition measurement, but same-sample indoor, fibre and water studies independently place plastic as a minority. Yadav's geometric apparent mass and Song's polymer-marker mass are incompatible stress cases and must not be pooled with the benchmark. For air, Evangelou et al. reported a global median of 0.03 particles/m³ and found that existing inventories overpredicted measurements by two to four orders of magnitude.

2. External exposure is not internal dose. Available kinetic evidence indicates that most orally administered particle mass is cleared, while the absorbed and retained fractions at ordinary exposure remain incompletely quantified. Reports of particles or polymer-associated signals in tissues can establish occurrence only when analytical identity and contamination control are adequate; they do not by themselves establish accumulation, pathology or disease causation. An independent QA/QC audit of 133 datasets from 101 studies found that none met all 14 essential criteria for direct quantitative risk assessment. This does not invalidate every occurrence finding or make health risk unknowable; it means the current literature cannot be used as a representative measure of human body burden, organ accumulation or particle-specific dose-response.

3. Plastic particles can produce effects at very high, atypical doses, as can many other poorly soluble particles. Such findings are generic dose-dependent hazard evidence, not proof that ordinary plastic dust is uniquely toxic. Stronger mammalian studies include formal study NOAELs of 25 mg/kg/day for weathered fragmented PP and 5,000 mg/kg/day for cellulose acetate, 1,000 mg/kg/day for PA-6, approximately 1,600-2,000 mg/kg/day of each PE and PET component administered together, and author-reported values of at least 2,000 mg/kg/day for fragmented PP and PTFE. These doses are remote from modeled ordinary exposure and do not create universal safety thresholds, but they strongly constrain claims of exceptional potency at trace exposure.

4. There is no scientific basis for treating ordinary plastic dust as a special case. Generic particle mechanisms cannot establish plastic-specific causation, and no reliable evidence shows ordinary common-polymer particles to be materially more harmful than the broader low-toxicity dust mixture at compatible dose. In contrast, respirable crystalline silica or quartz, occupational wood dust and certain toxic metal or metalloid compounds have established human toxic or carcinogenic hazards. Those agents merit special controls because their hazards are demonstrated; ordinary plastic dust has not met that evidentiary threshold.

5. No reliable human evidence demonstrates adverse health effects at ordinary exposure. The strongest contrary studies are important signals for scientific scrutiny, but exposure misclassification, cross-sectional design, reverse causality, confounding and analytical uncertainty remain conclusion-controlling. After more than five decades and 1,661 canonical evidence records, the conclusion is solid, not a placeholder awaiting the first meaningful studies. A prospective, replicated exposure-response relationship with orthogonal particle confirmation, quantified internal dose and credible causal evidence would materially change it.

6. Human-health and ecological questions must remain separate. Laboratory and field evidence supports concern for particular species, matrices, concentrations and local accumulation settings, and targeted source management may be justified without asserting a generalized human-health crisis. Human oral toxicology and the EPA particulate-context calculation are not ecological-risk assessments.

The conclusion should therefore be stated plainly: ordinary plastic-particle exposure has not been demonstrated to cause adverse human health effects, and current evidence indicates that it does not constitute a material public-health risk. Plastic is found everywhere because dust is found everywhere. It can produce effects under extreme, atypical loading because high-dose particle effects are not unique to plastic. Neither fact provides a scientific or logical justification for treating plastic dust as uniquely dangerous. The evidence supports perspective and proportionate management of specific sources or constituents, not disproportionate focus on plastic particles as a category.

Established nocebo research shows that negative expectations and threat-focused communication can generate symptoms and distress (Colloca & Barsky 2020). This does not imply that any particular reported symptom is psychogenic or that genuine hazards should be minimized. Public communication should therefore be accurate, calm and proportional to demonstrated risk while retaining the clear boundary that occupational, medical, fire, hotspot and extreme-dose scenarios require their own assessment.

31. Recommendations for Researchers, Regulators and Communicators

Researchers should report polymer formulation, size distribution, shape, particle number, mass, surface-area dose, agglomeration, route, vehicle and delivery method; use matrix-specific blanks, recovery and orthogonal identity confirmation; include route- and mechanism-matched nonplastic particle comparators where a plastic-specific biological effect or mechanism is claimed, while recognizing that occurrence, method-validation and physical-fate questions may require different controls; separate intact-particle effects from additives, leachates and analytical markers; report null findings; and make spectra, chromatograms and classification rules available where feasible. Atmospheric and climate models should state emission inventories, size distributions, particle lifetimes and pigment assumptions, propagate emission uncertainty, and be validated against size-aligned measurements before model output is communicated as a real-world burden or forcing.

Human biomonitoring studies should prospectively apply the Lane et al. framework or an equivalent standard: representative donor and anatomically defined sampling; complete collection-material and date information; validated digestion conditions that preserve particles; filtered reagents and clean-air controls; sufficient field and procedural blanks with LOD/LOQ and explicit correction; replicated positive recoveries across relevant polymers and sizes; and individual particle size, count, polymer and shape data. Unusual particle dimensions should be discussed against physiological barriers, sample geometry and contamination pathways.

Exposure studies should distinguish measured particle mass, modeled mass, geometry-derived mass and polymer-marker-equivalent mass. Lower and upper size limits, count-to-mass assumptions and uncertainty propagation should be explicit. Toxicology studies should compare administered dose with plausible internal dose and should not label the highest tested dose without detected adverse effects as a formal NOAEL unless the primary source supports that terminology.

Regulators should distinguish occurrence monitoring from risk assessment, identify the specific source, route, population and endpoint, and compare proposed intervention benefits with substitution and lifecycle consequences. Ordinary plastic dust should not be given special hazard status merely because it is detectable or synthetic; exceptional controls require demonstrated exceptional exposure, potency or a hazardous constituent. Human-health conclusions should not be generalized to ecological settings, and ecological hotspot management should not be presented as proof of generalized human disease risk. Treatment policies should distinguish capture, transfer and destruction.

Communicators should begin with the physical reality: particles are everywhere, plastic is a small to trace component of the total dust background, and detection is expected. They should then state the dose boundary: plastic particles can produce effects at very high, atypical doses, as can many dusts, but no reliable evidence establishes causal adverse human-health effects from ordinary real-world exposure or exceptional plastic-specific potency. Known hazardous particles such as quartz, occupational wood dust and certain toxic metal compounds should not be blurred together with ordinary plastic dust. Communicators should avoid equating detection with injury, ingestion with uptake, tissue occurrence with pathology, association with causation, particle count with mass, administered dose with ordinary exposure or model output with measurement. Disproportionate threat framing can produce nocebo symptoms and distress, so accuracy requires neither alarmism nor claims of absolute zero risk (Colloca & Barsky 2020). Proposed substitutions should be assessed using complete material and life-cycle consequences rather than plastic removal alone.

Appendix A. Methodology and Audit Boundaries

Database field definitions

The authoritative field definitions are preserved in the workbook Data Dictionary and the master TSV. Core operational counts are role-based and do not imply equal quality.

Study selection

The corpus preserves the canonical frozen order and includes primary studies, scientific syntheses, formal context, commentary, supporting information, duplicates and provenance records. Excluded records remain visible for auditability.

Quality scoring

Method validity, exposure realism, control quality and plastic specificity are scored on provisional 0-3 scales and may be overridden by expert adjudication.

Analytical hierarchy

Highest confidence generally combines chemical specificity, matrix validation, blanks, recovery, an explicit validated identification/quantification window and, when the claimed measurand or matrix complexity warrants it, orthogonal confirmation. A single rigorously validated particle-resolved method can support a bounded finding within its demonstrated matrix and size range. Nominal instrumental detection capability is not the same as the validated lower identification or quantification cutoff achieved after matrix preparation, recovery and spectral-quality rules. Lower-confidence methods include unconfirmed optical identification, staining alone, SEM-EDS carbon morphology, DLS without polymer identity and thermal-marker mass interpreted as intact particles.

Exposure realism

Measured real-world occurrence is separated from exposure-relevant controlled studies, scenario models, high-dose proof-of-concept studies and unquantified exposure.

Publication lineage

Preprints, final articles, corrections, supplements and derivative communications are linked to prevent double counting.

Search limitation

The database is extensive but not a claim to contain every publication. The stopping rule prioritizes sources capable of changing conclusions.

Controlled grading and adjudication

Records were initially structured through controlled data extraction and rule-based fields, followed by expert adjudication of evidence role, design, analytical validity, exposure realism, controls, plastic specificity and permitted inference. Automated scoring was used as an organizational aid, not as an autonomous scientific verdict. The adjudicator knew study results; outcome blinding was not implemented and is therefore a potential review-level bias. Positive, null and adverse findings were retained under the same inclusion and quality rules. Expert overrides required a documented conflict between the mechanical score and source-level evidence, with the reason recorded in controlled notes.

Duplicate and publication-lineage control

Conference abstracts, preprints, journal articles, corrections, supplements and later analyses from the same experiment were linked where identifiable. The independent-primary count is not intended to count the same dataset repeatedly. Remaining lineage uncertainty is disclosed rather than resolved by assumption.

Search and corpus limitations

The database is extensive but is not a claim to contain every publication. Versioned acquisition, source-verification, publication-lineage and adjudication records are retained, but the earliest corpus-building search dates, verbatim strings, raw-result counts and pre-canonical deduplication totals were not preserved in one version-controlled log. Appendix I therefore separates exact records from reconstructed descriptions and does not invent PRISMA counts. English-language and accessible-source bias, evolving terminology, incomplete indexing, inaccessible supplements and rapid publication growth can affect recall. A newly identified source is added only through controlled correction rather than silent alteration of the version-controlled baseline.

Auditability and reproducibility

This report is released as an author-final critical evidence synthesis. External specialist review and independent second-rater grading are not part of the release design. Auditability is provided through source-level citations, explicit appraisal domains, preservation of contrary and null findings, publication-lineage control, quantitative compatibility rules, study-summary verification codes and a version-controlled correction process. Any credible error or conclusion-changing new evidence should be documented, adjudicated at source level and propagated to affected text, tables, figures and traceability records.

Source-level verification checklist

1. Verify polymer, formulation, size distribution, route, duration, dose and endpoint against the primary source.
2. Verify that particle identity is established by a method capable of supporting the claimed inference.
3. Confirm sample representativeness, blank correction, recovery and positive controls, matrix validation and lower size cutoff.
4. Separate intact particles from additives, leachates, pigments, fillers, sorbed chemicals and destructive analytical markers.
5. Determine whether matched nonplastic particle controls are required and present.
6. Check whether the study dose and delivery method are compatible with ordinary exposure.
7. Identify the strongest supporting and strongest contrary evidence and whether either changes the conclusion.
8. Record the exact permitted inference and the evidence that would materially change it.

Reproducibility status and living-update protocol

This evidence programme is database-driven but is not represented as a completed PRISMA systematic review. Appendix I consolidates the recoverable search history, exact version-controlled corpus accounting, screening and exclusion rules, official-agency refresh queries and a prospective update protocol. Historical raw search-result and deduplication counts that were not retained remain explicitly unavailable. The programme includes the targeted current-evidence, common-polymer toxicology, thermoplastics source-recovery, atmospheric and human-biomonitoring QA/QC updates consolidated through 29 July 2026, internal database reconciliation, a mechanically generated High/Moderate register, a 115-source internal development audit set prepared during manuscript development and an internal AI-assisted challenge review. The 115-source set is retained as historical internal QA provenance and is not represented as external validation.

Crosswalk between custom grades and established appraisal tools

Evidence class	Custom domains	Closest established tool	Important non-equivalence
Human observational / clinical	Exposure validity, temporality, confounding, selection, outcomes, reporting, causal reach	ROBINS-E	ROBINS-E low risk indicates lower bias in a non-randomized exposure study; it does not validate the microplastic analyte or establish causality.
Human tissue / bodily-fluid measurement	Sample representativeness, donor and anatomical context, collection/digestion, contamination controls, blanks/LOD/LOQ, blank correction, positive recovery, particle size/count/type/shape	Lane et al. 2026 14-criterion QA/QC framework	A zero on any essential criterion means not fit for direct quantitative risk assessment within that framework; it does not mean every detection is false or the study has no scientific value.
Animal toxicology	Randomization, allocation, blinding, dose, route, controls, attrition, adversity, kinetics	SYRCLE / OHAT	Custom grade adds exposure realism, plastic specificity and administered-to-internal-dose interpretation.

Evidence class	Custom domains	Closest established tool	Important non-equivalence
Analytical methods	Identity specificity, blanks, recovery, matrix effects, limit of detection, orthogonal confirmation	QUADAS-2 adapted / method-validation principles	QUADAS-2 was designed for diagnostic accuracy; adaptation is conceptual rather than a formal QUADAS score.
Systematic reviews	Search completeness, duplicate screening, appraisal, synthesis, publication bias	AMSTAR 2 / PRISMA reporting	Secondary-synthesis quality is separate from the quality and causal reach of included studies.

Table A1. Crosswalk of the custom appraisal framework to established tools

Appendix B. Conclusion Stress-Test Matrix

Reader-facing stress-test summary

The table below identifies the strongest important challenge to each major conclusion. Detailed source-level stress tests follow. A contrary study is not dismissed because its direction differs; it is assessed for analytical validity, exposure relevance, causal design, independence and whether it changes the bounded conclusion.

Table B1. Major conclusions versus strongest contrary evidence

Major conclusion	Strongest support	Strongest important contradiction	Does it change the conclusion?	Evidence that would change it
Ordinary exposure is small within the best integrated model	Nor et al. 2021 integrated synthesis	Yadav 2022 and Song 2024 report mg/day apparent masses	No: incompatible geometric and marker-mass constructs	Validated representative total-exposure study with measured particle-resolved mass
No demonstrated human disease causation at ordinary exposure	Human studies lack validated ordinary-exposure dose and causal designs; Tran et al. 2026 concludes heterogeneity and bias constrain causality. Lane et al. 2026 found that none of 133 human tissue or bodily-fluid datasets met all essential QA/QC criteria for direct quantitative risk assessment.	Marfella 2024, Xu 2025, Nihart 2025, He 2025, Li 2026a, Chen 2026, Fang 2026, Lu 2026 and Paolisso 2026 report important associations or disease-state occurrence findings.	No: none establishes ordinary exposure as the cause; several materially strengthen the case for prospective research.	Replicated prospective exposure-response with orthogonal particle confirmation, internal dose, temporality and robust confounder control.
Generic mechanisms are not plastic-specific	Critical reviews and matched comparator experiments	Formulation-specific effects and 2023 meta-analysis suggesting marginal excess hazard	Narrows wording but does not overturn: differences are possible, exceptional generic potency not established	Matched particles showing reproducible excess effect at realistic dose and equivalent surface/size
High-dose animal effects do not define ordinary human risk	Dose-realism audit; Merski 2008 PE/PET mixture; Buesen 2025 PA-6; Lee 2022 PTFE; Kim 2021 and Lee 2023 PP no-adverse-effect findings; archival 5% PE and lifespan 10% VCM-free PVC context.	Sánchez 2026 reports intestinal changes at 1 mg/kg/day; Lu 2025 reports chronic PET liver effects at 50-500 µg/day; Lewis 2026 reports mild acute PET effects; Lee 2022 PE and Kim 2025 PET report pulmonary findings at lower doses.	No for ordinary human exposure: positive studies remain remote from ordinary external mass exposure and have comparator, endpoint-adversity, lesion-identity or study-design limitations. The added evidence increases the priority of PET replication but does not create a compatible ordinary-exposure point of departure.	Replicated chronic adverse outcome at validated ordinary internal dose with route-relevant dosing and matched nonplastic particles.
Tissue detection does not establish pathology	Analytical validation, blanks, causal criteria and absence of a complete kinetic chain. Lane et al. 2026 independently found that none of 133 human tissue or bodily-fluid datasets met all 14 essential QA/QC criteria for direct quantitative use in risk assessment.	Nihart 2025 reports high brain mass; He 2025 and Fang 2026 report CSF associations; Li 2026a reports living brain-tumour occurrence; Li 2026b reports matched blood-bone-disc occurrence; Chen 2026 and Marfella 2024 report disease-tissue associations.	No causal conclusion change; these studies strengthen occurrence/association evidence and require direct analytical replication.	Validated intact-particle identity and location, quantitative burden, temporality, dose-response and mechanism.
Ecological risk is context-specific	Environmentally realistic studies and exposure-distribution analyses	Laboratory effects and hotspot measurements	No universal conclusion; supports targeted hotspot and species-specific management	Field-replicated population or community effects at measured concentrations
Current ordinary human risk is not established as high	Integrated exposure, kinetics, toxicology and causal evidence	Large apparent exposure estimates and selected human associations, including the expanded 2025-2026 tissue, cardiovascular, neurological, cancer and metabolic literature	No at present; method and causal limitations are conclusion-controlling	Convergent validated exposure, internal dose and prospective causal evidence

C01: Strongly supported

Conclusion: Reported microplastics are widespread across environmental matrices, but absolute abundance estimates are strongly conditioned by sampling design, analytical size window, recovery and identification rules.

Strongest support: Ciornii et al. 2025; Lares 2019; Peneva 2025; Jüngling 2026

Strongest contrary evidence: Validated matrix-specific methods demonstrate that reliable measurement is possible within defined size and polymer windows (Mukotaka 2021; Akhbarizadeh et al. 2025).

Highest-quality contradiction: Mukotaka 2021

Required assumptions: The term widespread refers to repeated detection, not uniform concentration or increasing trend.

Residual uncertainty: Cross-study concentration comparisons remain uncertain without harmonized methods.

Evidence that would change the conclusion: Large harmonized interlaboratory datasets showing low between-method variance across matrices.

C02: Established

Conclusion: Analytical false positives and contamination are material risks, especially for biological matrices, small particles and thermal-marker methods.

Strongest support: Rauert et al. 2025; Rauert et al. 2022; Liu et al. 2024; Bissonnette et al. 2025; Lane et al. 2026

Strongest contrary evidence: Improved cleanup, isotope standards and orthogonal methods can reduce these errors (Almeida et al. 2026; Kim et al. 2026).

Highest-quality contradiction: Kim et al. 2026

Required assumptions: Evidence of a false-positive pathway does not invalidate every result from the affected method.

Residual uncertainty: Laboratory-specific false-positive rates are not consistently reported.

Evidence that would change the conclusion: Blind proficiency testing demonstrating consistently low false-positive rates in complex tissues.

C03: Established

Conclusion: Detection of polymer-associated signal or particles in human tissues does not, by itself, establish absorption rate, long-term retention, pathology or disease causation.

Strongest support: Lane et al. 2026; Rauert et al. 2025; Bissonnette et al. 2025; Dzierżyński et al. 2025; Marfella et al. 2024

Strongest contrary evidence: Several studies report particle-resolved occurrence or clinical associations (Amato-Lourenço et al. 2024; Jenner et al. 2022; Marfella et al. 2024).

Highest-quality contradiction: Marfella et al. 2024

Required assumptions: Causal inference requires validated exposure, temporality, confounder control and biological plausibility at relevant dose.

Residual uncertainty: Some specific tissue detections may be valid even when causal claims are not.

Evidence that would change the conclusion: Prospective replicated studies with orthogonal particle confirmation, internal-dose quantification and causal intervention evidence.

C04: Strongly supported

Conclusion: No reliable evidence demonstrates that ordinary realistic microplastic exposure causes adverse human health effects.

Strongest support: Quality distribution: 63 human observational/clinical records comprise 2 Moderate, 50 Low and 11 Very low; no High-quality causal study. Lane et al. 2026 independently screened 133 human tissue or bodily-fluid datasets and found that none met all 14 essential QA/QC criteria for direct quantitative risk assessment. Key examples: Marfella et al. 2024; Lee et al. 2024; Kong et al. 2025.

Strongest contrary evidence: Associations have been reported for cardiovascular, reproductive, inflammatory and developmental endpoints.

Highest-quality contradiction: Marfella et al. 2024

Required assumptions: Association studies can generate hypotheses but cannot resolve reverse causation, confounding and measurement error.

Residual uncertainty: A causal effect cannot be ruled out; it is not demonstrated.

Evidence that would change the conclusion: Prospective exposure-validated cohorts or randomized exposure-reduction interventions with clinically meaningful outcomes.

C05: Strongly supported

Conclusion: Animal and cell studies demonstrate biological hazard potential under some experimental conditions, but many use doses, pristine particles and exposure systems not representative of typical human exposure.

Strongest support: Fatima et al. 2026; Lin et al. 2025; Deng et al. 2017; Stock 2021

Strongest contrary evidence: Some studies report effects at concentrations described as environmentally relevant (Horn et al. 2020; Bacchetta et al. 2024).

Highest-quality contradiction: Horn et al. 2020

Required assumptions: Risk inference requires quantitative comparison of administered dose with measured exposure and internal dose.

Residual uncertainty: Environmental realism varies by matrix, particle type and organism.

Evidence that would change the conclusion: Replicated adverse apical effects at independently measured real-world doses with matched natural-particle controls.

C06: Moderately supported

Conclusion: Standardized and repeated-dose mammalian studies include important null findings and high doses without detected adverse effects for specific PP, PA-6, PTFE and PE/PET preparations. The PP evidence includes a GLP 28-day formal NOAEL of 25 mg/kg/day for weathered fragments. Supplementary archival evidence adds standalone PE at 5% of the diet for 90 days and a lifespan 10% VCM-free PVC control diet, but neither is treated as a formal standalone polymer NOAEL. No verified repeated-dose oral NOAEL was identified for fragmented/weathered PS.

Strongest support: Merski et al. 2008; Buesen et al. 2025; Lee et al. 2022 (PTFE); Kim et al. 2021 PP full paper and supporting information; Lee et al. 2023; Welle and Franz 2018/CIR 2007 archival PE context; Feron et al. 1981 PVC control-diet context.

Strongest contrary evidence: Lee et al. 2022 (PE) and Kim et al. 2025 (PET) report pulmonary findings at lower doses; Lu et al. 2025 reports chronic PET liver effects at 50-500 micrograms/day; Lewis et al. 2026 reports mild acute PET effects; Sánchez et al. 2026 reports PVC effects at 1 mg/kg/day; Fatima et al. 2026 reports high-dose PVC/PS effects. The Kim et al. 2020 PS conference abstract cannot be quantitatively assessed from the accessible source.

Highest-quality contradiction: Lu et al. 2025 provides the most informative longer-duration PET contradiction; Lee et al. 2022 PE remains the principal direct pulmonary contradiction, while Kim et al. 2025 and Lewis et al. 2026 remain lower-confidence or acute evidence.

Required assumptions: Polymer, mixture status, particle size, shape, preparation, aging, additives, duration and lesion verification limit cross-study generalization.

Residual uncertainty: PE and PET evidence is discordant. The 13-week 50:50 mixture supports high component exposure without apparent target-organ toxicity, but particle dimensions were not measured and the polymers were not tested independently. The standalone PE source remains secondary-cited. PVC and fragmented/weathered PS lack dedicated guideline-quality repeated-dose oral studies establishing formal NOAELs. PET studies span acute biomarkers, chronic liver effects and disputed pulmonary pathology and require independent replication.

Evidence that would change the conclusion: Well-powered guideline studies showing reproducible adverse effects at materially lower doses with chemically co-localized lesion particles, quantitative blinded pathology and complete vehicle, dye and particle controls.

C07: Strongly supported

Conclusion: A particle-free control is insufficient to establish plastic-specific toxicity; matched natural or alternative-particle comparators materially improve causal attribution.

Strongest support: Gerdes 2019; Straub et al. 2017; comparative-particle evidence across the database

Strongest contrary evidence: Some polymer-specific material or chemical effects can be established without a natural-particle comparator.

Highest-quality contradiction: Hirai et al. 2011

Required assumptions: Comparator must be matched for size, shape, surface area, density and dose.

Residual uncertainty: Perfect matching is technically difficult.

Evidence that would change the conclusion: Consistent polymer-specific effects across well-matched comparator studies.

C08: Strongly supported

Conclusion: Drinking-water concentrations are method- and system-dependent; well-controlled studies can find very low confirmed particle counts, and distribution systems can both remove and introduce particles.

Strongest support: Feld et al. 2021; Adediran et al. 2024; Sun et al. 2024; Mukotaka 2021

Strongest contrary evidence: Other surveys report higher counts, especially at smaller size cutoffs.

Highest-quality contradiction: Sun et al. 2024

Required assumptions: Counts from different lower size limits are not directly comparable.

Residual uncertainty: Sub-spectroscopic particles are incompletely measured, but analytical uncertainty alone does not establish material risk or justify a plastic-specific research priority.

Evidence that would change the conclusion: Validated source-to-tap evidence showing a materially larger exposure plus harm at realistic dose beyond matched nonplastic particles.

C09: Strongly supported

Conclusion: Food and beverage studies demonstrate external ingestion pathways, but they generally do not measure absorbed or retained dose and cannot establish health risk from occurrence alone.

Strongest support: Milne 2024; Zhou 2023; Traylor et al. 2024; Schwabl et al. 2019

Strongest contrary evidence: Some product-use studies report high particle counts under specific preparation conditions.

Highest-quality contradiction: Zhou 2023

Required assumptions: Particle count must not be treated as mass or retained dose without validated conversion.

Residual uncertainty: Total diet exposure remains sensitive to analytical cutoff and food handling.

Evidence that would change the conclusion: Validated total-diet mass balance linked to internal dose and health-relevant thresholds.

C10: Moderately supported

Conclusion: Most ingested microplastic mass is expected to be excreted rather than retained, but quantitative human absorption and clearance remain incompletely measured.

Strongest support: Lee et al. 2023; Straub et al. 2017; Letwin et al. 2026; Nor et al. 2021

Strongest contrary evidence: Controlled animal studies report distant-tissue signals under some conditions (Garcia et al. 2024; Deng et al. 2017).

Highest-quality contradiction: Garcia et al. 2024

Required assumptions: Animal tracer signals accurately represent intact particles and are transferable to humans.

Residual uncertainty: Human particle-size-specific absorption fractions are uncertain.

Evidence that would change the conclusion: Validated human kinetic studies with mass balance, orthogonal identification and repeated sampling.

C11: Strongly supported

Conclusion: Environmental fragmentation and weathering generate secondary particles but do not create additional polymer mass; material formulation and stabilization strongly affect rates.

Strongest support: Weinstein et al. 2016; Chowreddy et al. 2026; degradation/weathering corpus

Strongest contrary evidence: Some models or accelerated tests extrapolate rapid generation under selected conditions.

Highest-quality contradiction: Chowreddy et al. 2026

Required assumptions: Mass conservation is maintained and fragmentation is distinguished from mineralization.

Residual uncertainty: Field rates across products and environments remain uncertain.

Evidence that would change the conclusion: Long-term field mass-balance studies showing substantial mineralization or unexpected source rates.

C12: Strongly supported

Conclusion: Wastewater treatment can remove a large fraction of particles from effluent, but removal commonly transfers particles to sludge or residual streams and is not equivalent to destruction.

Strongest support: Talvitie et al. 2017; Michielssen et al. 2016; wastewater/treatment corpus

Strongest contrary evidence: Some advanced processes may degrade or mineralize selected polymers under controlled conditions.

Highest-quality contradiction: No single conclusion-changing study identified in the version-controlled corpus.

Required assumptions: Capture efficiency and destruction are reported separately.

Residual uncertainty: Downstream fate of captured particles is often poorly quantified.

Evidence that would change the conclusion: Full-system mass balances demonstrating verified mineralization at scale.

C13: Strongly supported

Conclusion: Plastic can carry additives and sorbed chemicals, but vector risk depends on net bioavailability relative to water, food, sediment and natural particles; detection on plastic is not sufficient.

Strongest support: Hirai et al. 2011; Rochman et al. 2014; Ogonowski et al. 2018; Wardrop et al. 2016

Strongest contrary evidence: Prepared co-exposure experiments demonstrate chemical transfer can occur (Wardrop et al. 2016).

Highest-quality contradiction: Wardrop et al. 2016

Required assumptions: Risk is determined by incremental dose, not merely chemical presence on plastic.

Residual uncertainty: Localized scenarios may differ.

Evidence that would change the conclusion: Field evidence showing plastic ingestion materially increases total internal chemical dose and adverse outcome.

C14: Limited evidence

Conclusion: Evidence for temporal increase is matrix- and method-specific; the version-controlled database does not justify a universal claim that environmental microplastic concentrations are increasing everywhere.

Strongest support: Historical and repeated monitoring records show heterogeneous trends and substantial method changes.

Strongest contrary evidence: Some local time series and archived-sample studies report increases.

Highest-quality contradiction: No globally representative harmonized time series identified.

Required assumptions: Trend requires comparable sampling and analysis over time.

Residual uncertainty: Many environments lack consistent long-term datasets.

Evidence that would change the conclusion: Global harmonized multi-decadal monitoring with stable methods.

C15: Strongly supported

Conclusion: Public communication should state the evidence-based particle context directly: dust is ubiquitous; plastic is a small to trace component; very high-dose effects are generic to many particles; known hazards such as quartz, occupational wood dust and toxic metal compounds are materially more concerning; and no reliable evidence demonstrates adverse human health effects from ordinary plastic-particle exposure. Current evidence provides no basis for special-case alarm, and uncertainty does not itself imply danger.

Strongest support: Integrated evidence-quality distribution, method studies, toxicology and epidemiology conclusions C01-C14.

Strongest contrary evidence: Some studies report adverse endpoints or associations.

Highest-quality contradiction: Marfella et al. 2024

Required assumptions: Risk communication should reflect evidence quality, dose and causal certainty.

Residual uncertainty: Specific high-exposure or susceptible subgroups remain under-studied.

Evidence that would change the conclusion: Replicated causal human evidence at measured realistic exposure.

Appendix C. Source Provenance and Unresolved Records

Purpose and counting rule. The frozen 1,661-record corpus contains one record classified as an unresolved source-identification record: STUDY-01095. Separately, Appendix H retains 13 technical, review, context or provenance records whose bibliographic identity is substantially known but whose authoritative public copy, date, authorship or version lineage remains incomplete. These are different audit categories and should not be combined into one unresolved-source count.

The 13 provenance-incomplete context records are retained for transparency. They are not counted as independent primary evidence, do not independently control any major conclusion, and should not be used to validate the current report. Their unresolved fields can be corrected in a future version without changing the frozen 1,661-record corpus unless a separate database adjudication is performed.

Table C1. Source-provenance records requiring future bibliographic cleanup

Catalogue ID	Record	Outstanding provenance issue
STUDY-01095	Unresolved source-identification record	Source identity remains unresolved in the frozen corpus. This is the single record counted in the corpus category "unresolved source-identification."
STUDY-01199	ACC Microplastic Reference Materials workshop summary (2022)	Authoritative public copy, exact version date, named report authorship and stable provenance remain unverified.
STUDY-01172	Jambeck Was Wrong: Reanalysis of Updated Data on Plastic and Ocean Inputs (2023)	Unpublished local analysis; author, version date, underlying data/equations, uncertainty analysis and institutional or peer-review provenance remain unverified.
STUDY-01427	Ball Corporation - Global Plastics and Recycling one-pager	Authoritative source, publication date, named authorship and stable version provenance remain unverified.
STUDY-01373	Chesapeake Bay Program - Microplastics in the Chesapeake Bay one-pager	Author/date/stable source and exact derivative relationship to the formal STAC assessment remain unresolved.
STUDY-01134	DeArmitt / Phantom Plastics - Microplastics and Tap Water (2026)	Two local technical-review PDFs remain without a designated public/final version, stable URL or verified source-level version lineage.
STUDY-01193	DeArmitt - Microplastic Ingestion & Accumulation (2024)	Verified public version, stable version date, reproducible source-level data package and equivalence to related public material remain unresolved.
STUDY-01216	DeArmitt - Ridgeway Rockbags: Scientific Assessment (2024)	Multiple local draft/final PDFs remain; exact commissioned final version, release date and stable public provenance remain unverified.
STUDY-01244	Guilloux - WHO Activities on Microplastics (2024)	Exact venue, presentation date, public archival record and authoritative final slide-deck version remain unresolved.
STUDY-01231	Lemstra - Dutch-language book extract/proof (2024)	Final book title, publisher, edition, publication date and stable final-publication source remain unverified.
STUDY-01364	Papatheodorou & Karapanagioti - Degradation of Plastic Debris... (2018)	Conference contribution is identifiable, but the authoritative abstract/presentation, final version and sufficient method detail remain unresolved.
STUDY-01119	Plastics Research Council / DeArmitt - Microplastics Evidence Review (2026)	Multiple local drafts/copies remain and the exact finalized public version and canonical version lineage have not been fully reconciled.
STUDY-01308	Royal Society of Chemistry workshop/presentation materials (circa 2017)	Exact title, presenter, event date and authoritative archived source remain unresolved; the previous Envirotech link was not equivalent.
STUDY-01192	Santiago v. Boston Scientific - Marlex polypropylene mesh expert opinion (2024)	Expert author, court, docket, filing date, public authentication and definitive version status remain insufficiently established.

Release interpretation. The presence of these provenance records is an auditability feature, not an evidentiary dependency. The central findings of this report are supported by separately identified primary, secondary and formal technical sources with the limitations stated in the relevant sections.

Appendix D. Cited Record Bibliography

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Appendix E. Quantitative Compatibility and Conversion Rules

The quantitative layer preserves original metrics and permits conversion only when the formula and assumptions are explicit. Particle count, mass, surface area and volume are not treated as interchangeable. Measured, modeled and administered quantities remain separate, and every comparison is assigned a compatibility class.

The key rules are: (1) do not convert count to mass without measured size, shape and density; (2) do not pool studies with materially different lower size cutoffs; (3) do not equate thermal polymer-marker mass with intact-particle count or morphology; (4) do not equate external intake with absorbed or retained dose; (5) do not treat animal administered dose divided by modeled human exposure as a formal margin without compatible toxicokinetics; (6) do not compare airborne fibre counts directly with occupational dust mass limits; and (7) do not describe water-phase removal as destruction.

The selected values below are anchors for interpretation, not a pooled exposure distribution. Their uncertainty and compatibility boundaries remain part of the result.

Table E1. Key quantitative anchors and required boundaries

Metric	Value	Source	Required interpretation boundary
Principal modeled adult external intake	883 particles/day; 583 ng/day	Nor et al. 2021	Best available integrated probabilistic synthesis identified; not measured total personal exposure
Illustrative 70-year external mass	14.91 mg/person	Report derivative from Nor et al. 2021	Constant-intake scenario; not internal dose
Modeled retained mass at age 70	40.7 ng/person	Nor et al. 2021	Narrower 1-10-micrometre model; not measured burden
Spanish bottled water	0.73 MPs/L; 1.61 micrograms/L	Gálvez-Blanca et al. 2024	Below 15 micrometres excluded; mass geometry-based
Danish tap water	7 confirmed total; about 0.2/L in 10-100 micrometres	Feld et al. 2021	Very low count with detection-limit uncertainty
Proof-of-principle potable-water particle composition	0.36 ± 0.13% of 0.5-10 micrometre particles identified as plastic; 18% of identified plastics below 1 micrometre	Jüngling et al. 2026	Three samples from one low-particle-load water source; automated Raman proof of principle, not a general water composition estimate
Global atmospheric concentration	Median 0.03 particles/m³; land 0.08; ocean 0.003	Evangelou et al. 2026	Global compilation of 925 concentration measurements; heterogeneous methods and size windows; not personal dose; model comparison focused on 5-100 µm
Scaled global atmospheric emissions	0.0045 Tg/year (90% interval 0.0006-0.0089) for 5-100 µm	Evangelou et al. 2026	Measurement-constrained model estimate; incomplete sources, sparse ocean observations and size-distribution assumptions remain
Indoor/outdoor airborne fibres	1-60 / 0.3-1.5 fibres/m³	Dris et al. 2017	Total fibres not synonymous with plastics; not mass
Human lung occurrence	0.69 particles/g blank-adjusted mean	Jenner et al. 2022	Selected surgical tissue; occurrence only

Metric	Value	Source	Required interpretation boundary
Selected oral highest doses without detected adverse effects	25; 1,000; approximately 1,600-2,000 per PE/PET component; at least 2,000; 5,000 mg/kg/day	Kim 2021; Buesen 2025; Merski 2008; Lee 2022 PTFE; Lee 2023; Thomas 1991	PE/PET components were co-administered in a 50:50 mixture; cellulose acetate is a semi-synthetic thermoplastic; all values are study-specific scale anchors, not universal thresholds or formal margins.
OSHA particulate context	15 mg/m ³ total; 5 mg/m ³ respirable	External regulatory context	Not microplastic-specific and not directly comparable with fibre counts
Yadav geometric apparent mass	122-203 mg/day	Supplementary method-stress source	Calculated from assumed geometry; incompatible with particle-resolved benchmark
Song polymer-marker apparent mass	27.1 mg/day at median 58.9 kg	Supplementary method-stress source	Py-GC/MS polymer-equivalent mass; intact particles not verified; high matrix-interference risk

Appendix F. Comparative Particle Context and Cumulative Weight-of-Evidence Updating

This appendix documents the independent derivation and evidentiary limits of the report's total-particle context and cumulative weight-of-evidence interpretation. The version-controlled database remains the 1,661-record baseline and the final-release manuscript does not alter the calculation or its evidentiary limits. The calculation uses Nor et al. 2021's modeled adult microplastic intake and U.S. EPA adult soil-plus-dust ingestion factors; matrix-specific comparisons remain separate. The purpose is to restore a relevant particulate denominator without misrepresenting the calculation as a direct measurement of total particle composition.

Nor et al. 2021 modeled median adult microplastic intake at 583 ng/day (0.000583 mg/day) for particles from 1 to 5,000 µm (Nor et al. 2021). The U.S. EPA recommends 30 mg/day as the central-tendency adult soil-plus-dust ingestion rate and summarizes key adult estimates around 50 mg/day. Division gives a microplastic-to-soil-plus-dust mass ratio of 1.17×10^{-5} to 1.94×10^{-5} , equivalent to approximately 0.0012–0.0019%, or 1.2–1.9 parts per 100,000. This is conservative as a total-particle context because the microplastic numerator includes all modeled pathways whereas the denominator includes soil and dust alone. It is not a claim that every air, water, food, blood or dust sample has the same composition.

The proportional-risk heuristic assumes equal potency per unit mass between ordinary plastic particles and the remaining particle mixture. Under the independently derived 0.0012–0.0019% mass comparison, plastic would require approximately 520–870-fold greater potency per unit mass to contribute 1% of total burden, 5,700–9,500-fold to contribute 10%, and 51,000–86,000-fold to equal the remaining burden. These are screening calculations rather than formal risk estimates because deposition, size, route, clearance, surface area and biopersistence can differ. They identify the extraordinary potency needed before a trace mass fraction becomes a major risk. No such exceptional potency is demonstrated for ordinary common-polymer particles at realistic exposure. Where a product contains a hazardous additive, pigment, filler, leachate or other constituent, the relevant comparison is the exposure and toxicity of that constituent, not the label 'microplastic'.

The cumulative weight-of-evidence statement is qualitative, not a formal quantitative probability model. The absence of reliable realistic-dose human causation, study-specific high-dose no-adverse-effect findings, kinetic evidence and the lack of a demonstrated generic plastic-specific excess mechanism collectively reduce the plausibility of a large hidden ordinary-exposure effect. A numerical probability would require prespecified endpoint definitions, study sensitivity, evidence dependence, publication-bias correction and an explicit evidence-to-probability model; this report does not invent such a number.

Route-matched kinetic context is also size-bounded. Miller et al. 2017 demonstrates systemic translocation of a small fraction of deliberately inhaled, few-nanometre gold nanoparticles in humans. After correction, the authors' illustrative 24-hour transfer estimate was approximately 0.02%, and smaller particles translocated much more readily than larger particles. This supports the biological plausibility of translocation for ultrasmall nanoparticles while arguing against treating all particles below 5 mm as one kinetic class. The result is not used to calculate a microplastic retention fraction.

Table F1. Contextual proportionality and cumulative-evidence rules

Proposition	Evidence or calculation	Permitted interpretation	Boundary
Nor et al. 2021/EPA contextual fraction	Approximately 0.0012-0.0019% relative to adult soil-plus-dust ingestion alone	Modeled microplastic mass is very small relative to this independently derived particulate denominator	Not a universal composition, direct risk measurement or complete inhaled-plus-ingested denominator
Matrix-specific fractions	Selected outdoor comparisons around 0.01%; selected indoor studies below 1% to a few percent	The exact fraction varies, but plastic is generally a minority	Count and mass fractions are not interchangeable
Potency needed to overcome trace mass	At 0.0012–0.0019% mass share: about 520–870-fold potency for 1% of burden; 5,700–9,500-fold for 10%; 51,000–86,000-fold to equal the remainder	A trace fraction becomes important only with exceptional potency or exposure	Screening calculation, not a formal risk estimate; deposition and toxicokinetics can differ

Proposition	Evidence or calculation	Permitted interpretation	Boundary
Plastic specificity	Critical review and matched-comparator studies (Ogonowski 2018; Gerdes 2019; Ogonowski et al. 2016)	Generic mechanisms cannot establish plastic-specific causation	Shared mechanisms do not imply equal hazard; formulation-specific differences require matched testing at compatible dose
Comparative hazard	2023 meta-analysis found only marginally greater average hazard with substantial uncertainty (Ogonowski et al. 2023)	No evidence of orders-of-magnitude exceptional potency	Heterogeneous experimental concentrations and endpoints
Dose separation	Selected oral values, including PE/PET mixture components and PTFE, are approximately 3 million to 240 million times above modeled ordinary external mass exposure.	Trace ordinary mass is remote from the selected study-specific no-adverse-effect dose scale.	Cross-study scale comparison, not a formal margin of safety
Exposure sensitivity	At 1,000-fold higher exposure, remaining separation approximately 3,000 to 240,000-fold	Conclusion is not sensitive to modest exposure-estimation error	Still depends on compatible toxicokinetics and endpoints
Cumulative weight of evidence	More than five decades of relevant evidence (1973-2026) without High-quality realistic-dose human causation, considered with kinetics and study-specific no-adverse-effect findings	A large hidden ordinary-exposure effect becomes progressively less plausible	Inference depends on quality, relevance, independence and sensitivity; no numerical posterior without a formal model
Human-lung inorganic particle background	Hayden et al. 2023: 124,018 particles characterized in 38 surgical biopsy specimens; 45 classes; >90% below 2.5 micrometres in greatest dimension	Direct evidence that lung tissue can contain a chemically diverse mixture of mineral, metal and metal-oxide particles within a submicrometre-to-micrometre size range	Selected military clinical biopsies; fixed-area qualitative FE-SEM/EDS; counts are lower limits and not particles/g; method favors inorganic/high-atomic-number contrast and cannot define a plastic fraction or population burden
Ultrasmall inhaled nanoparticle translocation	Miller et al. 2017 + correction: controlled ~4-5 nm gold inhalation; corrected illustrative 24-h transfer ~0.02%; strong size dependence	Pulmonary barrier crossing is possible for a small fraction of ultrasmall nanoparticles	Engineered gold tracer, controlled exposure and non-closed mass balance; not a microplastic uptake fraction and not transferable to micrometre-scale particles

Independent contextual sources

U.S. Environmental Protection Agency. Exposure Factors Handbook, Chapter 5: Soil and Dust Ingestion, 2017 update. <https://www.epa.gov/expobox/exposure-factors-handbook-chapter-5>

Maurizi, L., Simon-Sánchez, L., Vianello, A., Nielsen, A. H., & Vollertsen, J. (2024). Every breath you take: High concentration of breathable microplastics in indoor environments. *Chemosphere*, 361, 142553. <https://doi.org/10.1016/j.chemosphere.2024.142553>

Hayden, L., Strausborger, S., & Lewin-Smith, M. (2023). 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), 235-243. <https://doi.org/10.1093/micmic/ozac015>.

Miller, M. R., Raftis, J. B., Langrish, J. P., et al. (2017). Inhaled Nanoparticles Accumulate at Sites of Vascular Disease. *ACS Nano*, 11(5), 4542-4552. <https://doi.org/10.1021/acsnano.6b08551>. Correction: *ACS Nano*, 11, 10623-10624. <https://doi.org/10.1021/acsnano.7b06327>.

Appendix G. Current Agency Positions and Review Status

This appendix records official agency sources refreshed through 25 July 2026 and the review status at publication. The exact official-domain discovery queries are recorded in Appendix I. Agency statements are scope-specific and should be refreshed when the report is next updated.

Official agency sources

U.S. Food and Drug Administration. Microplastics and Nanoplastics in Foods. Content current 24 July 2024. <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 new finding that current food levels cause harm. <https://www.fda.gov/about-fda/human-foods-program/human-foods-program-2026-priority-deliverables>

U.S. Food and Drug Administration. Understanding How the FDA Regulates Substances that Come into Contact with Food. Content current 8 May 2025. FDA states that safety requires a reasonable certainty of no harm under intended conditions and that science can never be absolutely certain of the absence of any risk from any substance. <https://www.fda.gov/food/food-packaging-other-substances-come-contact-food-information-consumers/understanding-how-fda-regulates-substances-come-contact-food>

World Health Organization. Dietary and inhalation exposure to nano- and microplastic particles and potential implications for human health. 30 August 2022. ISBN 978-92-4-005460-8. <https://www.who.int/publications/i/item/9789240054608>

European Food Safety Authority. Microplastics: European Parliament requests scientific advice from EFSA. 9 December 2025; opinion expected by end of 2027. <https://www.efsa.europa.eu/en/news/microplastics-european-parliament-requests-scientific-advice-efsa>

U.S. Environmental Protection Agency. Microplastics Research. Updated 19 March 2026. <https://www.epa.gov/water-research/microplastics-research>

Environment and Climate Change Canada and Health Canada. Science Assessment of Plastic Pollution. 2020. <https://www.canada.ca/en/environment-climate-change/services/evaluating-existing-substances/science-assessment-plastic-pollution.html>

German Federal Institute for Risk Assessment (BfR). Microplastics - What do we know today? 11 November 2025. <https://www.bfr.bund.de/en/press-release/microplastics-what-do-we-know-today/>

German Federal Institute for Risk Assessment (BfR). Microplastics: Discrepancy between scientific knowledge and public perception. 2 December 2025. BfR states that current knowledge indicates relatively low human-health risk, that detections and correlations do not establish causation, and that validated methods and long-term data remain necessary. <https://www.bfr.bund.de/en/notification/microplastics-discrepancy-between-scientific-knowledge-and-public-perception/>

U.S. Environmental Protection Agency. Draft Contaminant Candidate List 6 - CCL 6. Announced 2 April 2026; page updated 7 April 2026. Microplastics are included as a contaminant group for research and possible future regulatory consideration; listing is not a risk determination. <https://www.epa.gov/ccl/draft-contaminant-candidate-list-6-ccl-6>

U.S. Environmental Protection Agency. Basic Information on the CCL and Regulatory Determination. Updated 2026. EPA states that CCL publication imposes no requirements; a later regulatory determination is a separate decision requiring sufficient health and occurrence information. <https://www.epa.gov/ccl/basic-information-ccl-and-regulatory-determination>

UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment. Statement on the potential risks from inhalation exposure to microplastics. 2024. The Committee concluded that available data were insufficient for a full risk assessment and identified major method, exposure, kinetic and human-data gaps. <https://cot.food.gov.uk/sites/default/files/2024-02/Microplastics%20inhalation%20final%20draft%20statement%20Acc%20V%20SO.pdf>

Context sources for conditional safety examples

Joo, M. A., Kim, E. Y., et al. (2013). Hyponatremia caused by excessive intake of water as a form of child abuse. *Annals of Pediatric Endocrinology & Metabolism*, 18(2), 95-98. <https://doi.org/10.6065/apem.2013.18.2.95>

Alva, R., Mirza, M., Baiton, A., et al. (2023). Oxygen toxicity: cellular mechanisms in normobaric hyperoxia. *Cellular Biology and Toxicology*, 39(1), 111-143. <https://doi.org/10.1007/s10565-022-09773-7>

Appendix H. Cited and Conclusion-Controlling Study Summaries

This appendix summarizes catalogue sources explicitly cited in the narrative, conclusion-controlling records and supplementary sources outside the frozen baseline retained for this edition. It is not a summary of every record in the 1,661-record evidence database. Each entry gives the citation, design, principal finding, risk relevance and key limitation. Verification codes are: V1 = official publisher full text, article page or primary institutional source reopened; V2 = authoritative abstract and bibliographic metadata verified; V3 = legacy version-controlled database record whose citation and identifier were checked but whose primary source was not independently reopened during the cited-source audit. Study IDs remain visible here for traceability but are not shown in the main narrative.

Environmental occurrence and transport

Brahney, J., Hallerud, M., Heim, E., Hahnenberger, M., & Sukumaran, S. (2020). Plastic rain in protected areas of the United States. *Science*, 368, 1257–1260. <https://doi.org/10.1126/science.aaz5819>.

Design: Field study measuring atmospheric deposition of plastic particles in protected areas of the western United States using wet and dry deposition sampling.

Findings: Plastic particles and fibres were reported in deposition samples, supporting long-range atmospheric transport into remote and protected environments.

Risk relevance: Establishes an atmospheric deposition pathway and regional occurrence; it does not establish a human-health or ecological effect.

Key limitations: Regional stations, contamination-sensitive sampling, method-defined size range and incomplete chemical confirmation constrain flux estimates and source attribution.

Catalogue ID: STUDY-00655. DOI/link: 10.1126/science.aaz5819 Verification: V3.

Egger, M., Nijhof, R., Quiros, L., et al. (2020). A spatially variable scarcity of floating microplastics in the eastern North Pacific Ocean. *Environmental Research Letters*, 15, 114056. <https://doi.org/10.1088/1748-9326/abbb4f>.

Design: Eastern North Pacific field survey comparing floating microplastic abundance across oceanographic regions.

Findings: Floating microplastics were spatially heterogeneous and comparatively scarce in some sampled areas, demonstrating that surface inventories are not uniform.

Risk relevance: Provides measured ocean-occurrence and distribution evidence and tests assumptions of homogeneous surface accumulation.

Key limitations: Surface-net sampling omits smaller and submerged particles, and cruise track, season, wind mixing and ocean circulation limit extrapolation to total ocean mass.

Catalogue ID: STUDY-00619. DOI/link: 10.1088/1748-9326/abbb4f Verification: V3.

Law, K. L., et al. (2014). Distribution of Surface Plastic Debris in the Eastern Pacific Ocean from an 11-Year Data Set. *Environmental Science & Technology*. <https://doi.org/10.1021/es4053076>.

Design: Eleven-year field dataset of surface plastic debris in the eastern Pacific Ocean.

Findings: The study mapped broad spatial distribution and temporal variability of surface debris across repeated sampling years.

Risk relevance: Provides long-term measured surface-ocean occurrence evidence and a benchmark for evaluating transport models.

Key limitations: Net mesh, wind-driven vertical mixing, route coverage and focus on buoyant surface material prevent conversion to total-ocean inventory or harm.

Catalogue ID: STUDY-00504. DOI/link: 10.1021/es4053076 Verification: V3.

Shiye Zhao. 2023. Pelagic microplastics in the North Pacific Subtropical Gyre: A prevalent anthropogenic component of the particulate organic carbon pool.

Design: Field study quantifying pelagic microplastics and particulate organic carbon in the North Pacific Subtropical Gyre.

Findings: The study reported microplastics as a detectable anthropogenic component of the sampled particulate pool, while natural particulate organic carbon remained the relevant broader matrix.

Risk relevance: Provides mass and particulate-context evidence for a high-accumulation ocean region.

Key limitations: Results are location-, depth-, season-, cutoff- and method-specific and should not be generalized to the whole ocean or converted directly into ecological risk.

Catalogue ID: STUDY-00627. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Woodall, L. C., Sanchez-Vidal, A., Canals, M., Paterson, G. L. J., Coppock, R., Sleight, V., Calafat, A., Rogers, A. D., Narayanaswamy, B. E., & Thompson, R. C. (2014). The deep sea is a major sink for microplastic debris. *Royal Society Open Science*, 1, 140317. <https://doi.org/10.1098/rsos.140317>.

Design: Deep-sea sediment and benthic-environment occurrence study using microscopy and spectroscopy to identify microplastic debris.

Findings: Microplastics were reported in deep-sea samples, supporting the deep ocean as a sink for transported plastic debris.

Risk relevance: Extends occurrence and fate evidence beyond surface waters.

Key limitations: Limited sites, contamination sensitivity, restricted analytical cutoff and uncertain sediment accumulation rates prevent global mass or biological-effect inference.

Catalogue ID: STUDY-01397. DOI/link: 10.1098/rsos.140317 Verification: V3.

Temporal-trend and historical-monitoring studies

Beer, S., Garm, A., Huwer, B., Dierking, J., & Nielsen, T. G. (2018). No increase in marine microplastic concentration over the last three decades – A case study from the Baltic Sea. *Science of the Total Environment*, 621, 1272–1279. <https://doi.org/10.1016/j.scitotenv.2017.10.101>.

Design: Retrospective Baltic Sea case study comparing marine microplastic concentrations over approximately three decades.

Findings: The study found no detectable increase in the sampled concentration metric over the evaluated period.

Risk relevance: Important null trend evidence showing that increasing attention or production does not automatically imply an increasing measured concentration in every matrix.

Key limitations: The conclusion is specific to the Baltic samples, archived material, size range and analytical comparability and does not establish a universal global trend.

Catalogue ID: STUDY-00339. DOI/link: 10.1016/j.scitotenv.2017.10.101 Verification: V3.

Brandon, J. A., Jones, W., & Ohman, M. D. (2019). Multidecadal increase in plastic particles in coastal ocean sediments. *Science Advances*, 5(9), eaax0587. <https://doi.org/10.1126/sciadv.aax0587>.

Design: Sediment-core study reconstructing multidecadal changes in plastic particles in coastal ocean sediments.

Findings: Particle abundance increased through the dated sediment record, consistent with rising local inputs and sediment accumulation.

Risk relevance: Provides a historical trend for one depositional matrix.

Key limitations: Sediment mixing, dating, local source history, recovery and analytical cutoff constrain temporal resolution and prevent generalization to surface water or global abundance.

Catalogue ID: STUDY-01599. DOI/link: 10.1126/sciadv.aax0587 Verification: V3.

Ryan, P. G., & Moloney, C. L. (1990). Plastic and other artefacts on South African beaches: Temporal trends in abundance and composition. *South African Journal of Science*, 86, 450–452.

Design: Repeated beach-survey study evaluating temporal changes in abundance and composition of plastic and other artefacts on South African beaches.

Findings: The study documented matrix- and category-specific temporal changes in beach debris.

Risk relevance: Provides a long-term local trend record.

Key limitations: Beach accumulation reflects local use, cleanup, wind, currents and sampling protocol and cannot establish a global microplastic trend or health effect.

Catalogue ID: STUDY-01196. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Simon Turner, A. A. Horton, Neil L. Rose & Claire Hall. 2019. A temporal sediment record of microplastics in an urban lake, London, UK. *Journal of Paleolimnology* 61:449-462.

Design: Dated sediment-core study reconstructing a temporal microplastic record in an urban London lake.

Findings: The core recorded changing particle abundance through time, providing a local archive of urban inputs and sediment retention.

Risk relevance: Supports use of sediments for historical trend reconstruction.

Key limitations: Core chronology, sediment mixing, local catchment changes, polymer confirmation and size cutoff bound the inferred trend.

Catalogue ID: STUDY-01497. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Airborne occurrence and external exposure

Dris, R., Gasperi, J., Mirande, C., Mandin, C., Guerrouache, M., Langlois, V., & Tassin, B. (2017). A first overview of textile fibers, including microplastics, in indoor and outdoor environments. *Environmental Pollution*, 221, 453–458. <https://doi.org/10.1016/j.envpol.2016.12.013>.

Design: Early indoor/outdoor fibre occurrence study using deposition sampling and chemical characterization of selected fibres.

Findings: Fibres were reported in both indoor and outdoor environments, with indoor levels higher in the studied setting and most fibres not confirmed as synthetic plastic.

Risk relevance: Supports atmospheric and indoor-fibre occurrence and provides total-fibre context.

Key limitations: Deposition is not respirable concentration; natural and regenerated-cellulose fibres can dominate, and only chemically confirmed synthetic fibres should be counted as microplastics.

Catalogue ID: STUDY-00977. DOI/link: 10.1016/j.envpol.2016.12.013 Verification: V3.

Islam, M. S., et al. (2023). How microplastics are transported and deposited in realistic upper airways? *Physics of Fluids*, 35, 063319. <https://doi.org/10.1063/5.0150703>.

Design: Computational fluid-dynamics study modeling transport and deposition of microplastic particles in realistic upper-airway geometries.

Findings: Predicted deposition depended strongly on aerodynamic size, shape, flow and airway geometry.

Risk relevance: Improves route-specific inhalation and deposited-dose modeling.

Key limitations: Model output requires validation with representative particles and breathing conditions and does not by itself establish tissue dose or adverse effects.

Catalogue ID: STUDY-00605. DOI/link: 10.1063/5.0150703 Verification: V3.

Liu, Y., Fu, H., Zhang, H., et al. (2026). Atmospheric Warming Contributions from Airborne Microplastics and Nanoplastics. *Nature Climate Change*. <https://doi.org/10.1038/s41558-026-02620-1>.

Design: Experimental EELS characterization of selected coloured PS, PE and PET particles combined with assumed global MNP emissions, FLEXPART transport and SBDART radiative-transfer modeling.

Findings: The study reported 0.039 ± 0.019 W/m² of global direct radiative forcing, equivalent to 16.2% of its modeled black-carbon forcing. Microplastics contributed 0.006 W/m² and nanoplastics 0.033 W/m²; modeled column burdens were 0.075 and 0.017 mg/m², respectively.

Risk relevance: Demonstrates that pigment, colour and particle size can materially affect the optical properties of selected plastic formulations and provides a testable radiative-forcing scenario.

Key limitations: Approximately 85% of the forcing came from nanoplastics extrapolated from assumed size distributions and a prescribed 28-day lifetime. Emission-rate uncertainty was not included. The combined modeled plastic burden was about 66% of the modeled black-carbon burden, and a larger global measurement synthesis found that existing atmospheric inventories overpredicted size-aligned concentrations by two to four orders of magnitude. The global forcing magnitude and ocean-gyre hotspots are therefore not validated real-world effects.

Catalogue ID: STUDY-00527. DOI/link: 10.1038/s41558-026-02620-1 Verification: V3.

Evangelou, I., Bucci, S., & Stohl, A. (2026). Atmospheric microplastic emissions from land and ocean. *Nature*, 649, 1186-1189. <https://doi.org/10.1038/s41586-025-09998-6>.

Design: Global atmospheric synthesis compiling 2,782 microplastic concentration and deposition measurements from 76 studies at 283 locations, combined with FLEXPART source-receptor modeling and size-aligned comparison of three emission inventories.

Findings: The measured global median atmospheric concentration was 0.03 particles/m³, with medians of 0.08 particles/m³ over land and 0.003 particles/m³ over the ocean. Existing top-down and bottom-up inventories overpredicted measured concentrations by two to four orders of magnitude. Measurement-constrained scaling yielded total 5-100 µm emissions of 0.0045 Tg/year and showed that land dominated particle-number emissions.

Risk relevance: Provides the largest global atmospheric measurement synthesis identified in this review, constrains typical outdoor occurrence, directly tests model outputs against observations and strengthens the conclusion that unscaled atmospheric models can materially overstate exposure and emissions.

Key limitations: The underlying studies used heterogeneous sampling and analytical methods; ocean observations were sparse; several source sectors were missing; mass-to-number conversion depended on assumed size distributions and geometry; and the transport-model comparison focused on 5-100 µm, not the full nanoplastic range or personal breathing-zone dose.

Catalogue ID: Not assigned (supplementary source outside the frozen baseline). DOI/link: 10.1038/s41586-025-09998-6 Verification: V1.

Rachid Dris, Johnny Gasperi, Mohammed Saad, Cécile Mirande & Bruno Tassin. 2016. Synthetic fibers in atmospheric fallout: A source of microplastics in the environment? *Marine Pollution Bulletin* 104:290-293.

Design: Field study of synthetic fibres in atmospheric fallout using deposition sampling and particle characterization.

Findings: Fibres were reported in atmospheric fallout, supporting the atmosphere as a transport pathway and source to terrestrial and aquatic surfaces.

Risk relevance: Establishes occurrence and deposition, not respirable dose or adverse health effects.

Key limitations: Passive fallout favors larger settling fibres; early visual or limited spectroscopic classification, contamination and natural/regenerated fibres constrain plastic-specific counts.

Catalogue ID: STUDY-00296. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Song, S., van Dijk, F., Vasse, G. F., Liu, Q., Gosselink, I. F., Weltjens, E., et al. (2024). Inhalable textile microplastic fibers impair airway epithelial differentiation. *American Journal of Respiratory and Critical Care Medicine*, 209(4), 427-443. <https://doi.org/10.1164/rccm.202211-2099OC>.

Design: Airway epithelial culture study exposing developing airway cells to inhalable textile microplastic fibres.

Findings: The fibres impaired selected epithelial differentiation endpoints under direct experimental exposure.

Risk relevance: Mechanistic inhalation-hazard evidence for a defined textile-fibre preparation.

Key limitations: In-vitro delivered dose, fibre dimensions, formulation, absence of matched natural/regenerated fibres and lack of organism-level outcomes prevent inference to ordinary human inhalation risk.

Catalogue ID: STUDY-01590. DOI/link: 10.1164/rccm.202211-2099OC Verification: V3.

Stephanie L. Wright, Julia Ulke, Anais Font, Kelly L. A. Chan & Frank J. Kelly. 2020. Atmospheric microplastic deposition in an urban environment and an evaluation of transport. *Environment International* 136:105411.

Design: Urban atmospheric-deposition field study combining fallout sampling with particle characterization and transport evaluation.

Findings: Microplastic-associated fibres and fragments were reported in urban deposition, with transport influenced by meteorology and local sources.

Risk relevance: Supports external airborne deposition and source-transport characterization.

Key limitations: Deposition flux is not respirable concentration or deposited lung dose; local sampling, blanks, size range and polymer-confirmation fraction limit population exposure inference.

Catalogue ID: STUDY-00148. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Food, beverage and packaging-release studies

Asadi Qajarloo, Z., Arfaeinia, H., Jahanara, M., et al. (2026). Influence of temperature and storage time on microplastic release from packaged sausages. *Scientific Reports*. <https://doi.org/10.1038/s41598-026-53153-8>.

Design: Packaged-food release study testing effects of temperature and storage time on microplastic occurrence in sausages.

Findings: Reported particle release or food concentrations varied with storage duration and temperature under the tested packaging conditions.

Risk relevance: Supports source-specific consumer-exposure characterization.

Key limitations: Product selection, packaging formulation, sample handling, blanks, size cutoff and confirmation rate limit market-wide and health-risk inference.

Catalogue ID: STUDY-00571. DOI/link: 10.1038/s41598-026-53153-8 Verification: V3.

Dehaut, A., Cassone, A.-L., Frère, L., Hermabessiere, L., Himber, C., Rinnert, E., Rivière, G., Lambert, C., Soudant, P., Huvet, A., Duflos, G., & Paul-Pont, I. (2016). Microplastics in seafood: Benchmark protocol for their extraction and characterization. *Environmental Pollution*, 215, 223–233. <https://doi.org/10.1016/j.envpol.2016.05.018>.

Design: Seafood analytical-method study developing and testing a benchmark extraction and characterization protocol.

Findings: The protocol evaluated digestion, filtration and spectroscopic confirmation for recovering microplastics from seafood tissues.

Risk relevance: Supports more comparable dietary-occurrence measurement and identifies method-induced losses or contamination.

Key limitations: Recovery with selected standards and species does not guarantee equivalent performance for every tissue, polymer, weathering state or smaller particle size.

Catalogue ID: STUDY-01366. DOI/link: 10.1016/j.envpol.2016.05.018 Verification: V3.

Guanyu Zhou. 2023. How many microplastics do we ingest when using disposable drink cups? *Journal of Hazardous Materials*, 441 (2023) 129982.

Design: Consumer-product release experiment measuring particles generated during use of disposable drink cups and estimating ingestion.

Findings: Use conditions released particles from disposable cups, with the reported amount varying by cup material, liquid temperature and protocol.

Risk relevance: Supports product-source characterization. Release does not itself establish absorbed dose or adverse health effects.

Key limitations: Product selection, simulated use, analytical cutoff, blank correction and assumptions converting particle counts to intake limit generalization.

Catalogue ID: STUDY-00265. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Gálvez-Blanca, V., Edo, C., González-Pleiter, M., Fernández-Piñas, F., Leganés, F., & Rosal, R. (2024). Microplastics and non-natural cellulosic particles in Spanish bottled drinking water. *Scientific Reports*, 14, 11089. <https://doi.org/10.1038/s41598-024-62075-2>.

Design: Spanish bottled-water occurrence study measuring microplastics and non-natural cellulosic particles above a defined lower-size cutoff.

Findings: The study reported approximately 0.73 microplastic particles/L and 1.70 artificial cellulosic particles/L; its estimated plastic mass was geometry-derived.

Risk relevance: Provides low-concentration bottled-water evidence and directly shows that nonplastic anthropogenic particles can exceed confirmed plastic counts.

Key limitations: Particles below approximately 15 micrometres were excluded, and mass was calculated from geometry rather than directly measured.

Catalogue ID: STUDY-01229. DOI/link: 10.1038/s41598-024-62075-2 Verification: V3.

Madeleine H. Milne. 2024. Exposure of U.S. adults to microplastics from commonly-consumed proteins. *Environmental Pollution*, 343 (2024) 123233.

Design: Market-basket occurrence study measuring anthropogenic or microplastic particles in commonly consumed protein foods and estimating adult dietary exposure.

Findings: Particles were reported across sampled protein categories, with concentrations and estimated intake varying by food type and analytical assumptions.

Risk relevance: Supports dietary occurrence and route-specific exposure characterization.

Key limitations: The sample frame, lower size cutoff, contamination controls, polymer-confirmation fraction and count-to-intake assumptions limit population-level estimates; ingestion is not absorption.

Catalogue ID: STUDY-00194. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Qin, J., & Fauziah, S. H. (2026). Microplastic contamination in pelagic and coastal fish from Malaysian waters: Tissue-specific distribution, polymer characteristics, and environmental risk indicators. *Marine Pollution Bulletin*, 229, 119669. <https://doi.org/10.1016/j.marpolbul.2026.119669>.

Design: Field survey of pelagic and coastal fish from Malaysian waters, examining tissue distribution and polymer characteristics.

Findings: Microplastics were reported in sampled fish, while edible muscle occurrence above the study cutoff was negligible or very low.

Risk relevance: Provides regional fish-occurrence and seafood-exposure context.

Key limitations: Cross-sectional sampling, contamination controls, polymer-confirmation rate and exclusion of smaller particles limit inference; occurrence does not establish toxicity.

Catalogue ID: STUDY-01211. DOI/link: 10.1016/j.marpolbul.2026.119669 Verification: V3.

Traylor, S. D., Granek, E. F., Duncan, M., & Brander, S. M. (2024). From the ocean to our kitchen table: anthropogenic particles in the edible tissue of U.S. West Coast seafood species. *Frontiers in Toxicology*, 6, 1469995. <https://doi.org/10.3389/ftox.2024.1469995>.

Design: Field survey of anthropogenic particles in edible tissues of seafood species from the U.S. West Coast.

Findings: Particles were reported in selected edible-tissue samples, with occurrence varying among species and locations.

Risk relevance: Provides direct seafood-occurrence evidence for the sampled species.

Key limitations: Small or regional sample frames, contamination-sensitive fibres, uncertain source attribution, analytical cutoff and incomplete polymer confirmation prevent population exposure or harm inference.

Drinking-water, treatment and distribution studies

Arata Mukotaka. 2021. Rapid analytical method for characterization and quantification of microplastics in tap water using a Fourier-transform infrared microscope. Science of the Total Environment, 790 (2021) 148231.

Design: Tap-water analytical-method study using FTIR microscopy for rapid particle characterization and quantification.

Findings: The study demonstrated a workflow for identifying and counting microplastics in tap water within a defined size and spectral domain.

Risk relevance: Supports drinking-water method development and more reproducible occurrence measurement.

Key limitations: Method performance depends on filter, matrix, recovery, blanks, spectral thresholds and lower-size cutoff and does not establish toxicity.

Catalogue ID: STUDY-00361. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Feld, L., da Silva, V. H., Murphy, F., Hartmann, N. B., & Strand, J. (2021). A study of microplastic particles in Danish tap water. Water, 13(15), 2097. <https://doi.org/10.3390/w13152097>.

Design: Danish tap-water occurrence study using contamination-controlled spectroscopy in a defined 10-100 micrometre window.

Findings: Only seven confirmed particles were detected across the analyzed samples, corresponding to roughly 0.2 particle/L in the stated window.

Risk relevance: High-value low-concentration drinking-water evidence and a counterweight to much higher method-incompatible counts.

Key limitations: Very low counts carry detection-limit and sampling uncertainty, and particles below the analytical window were not measured.

Catalogue ID: STUDY-00863. DOI/link: 10.3390/w13152097 Verification: V3.

Gbotemi A. Adediran, et al. 2024. Fate and behaviour of microplastics (>25 µm) within the water distribution network, from water treatment works to service reservoirs and customer taps. Water Research 255:121508.

Design: Source-to-tap field study following microplastics larger than 25 micrometres through a drinking-water distribution network.

Findings: Particle abundance and composition changed from treatment works through reservoirs and customer taps, indicating both removal and possible distribution-system contributions.

Risk relevance: Provides direct distribution-network fate and source evidence.

Key limitations: Particles below 25 micrometres were not measured, and limited networks, sampling events, source attribution and count-based mass balance constrain generalization.

Catalogue ID: STUDY-00426. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Radityaningrum, A.D., Trihadiningrum, Y., & Soedjono, E.S. (2023). Performance of conventional drinking water treatment plants in removing microplastics in East Java, Indonesia. Journal of Ecological Engineering, 24(6), 129–143. <https://doi.org/10.12911/22998993/162785>.

Design: Field evaluation of conventional drinking-water treatment plants in East Java using influent/effluent microplastic measurements.

Findings: The plants reduced counted particles to varying degrees across treatment stages.

Risk relevance: Provides full-scale treatment-performance evidence.

Key limitations: Removal percentages depend on matched sampling, recovery, cutoff and polymer confirmation; captured material transfers to residuals and is not necessarily destroyed.

Catalogue ID: STUDY-01006. DOI/link: 10.12911/22998993/162785 Verification: V3.

Xiangxiang Li, Yihan Yu, Mei Yang, Shaobai Wen & Jun Zhang. 2024. Tracking Microplastics Contamination in Drinking Water Supply Chain in Haikou, China: From Source to Household Taps. Toxics 12:793.

Design: Source-to-household field study tracing microplastics across the drinking-water supply chain in Haikou, China.

Findings: The study reported stage-dependent changes from source water through treatment and distribution to household taps.

Risk relevance: Supports source attribution and identifies where particles may be removed or introduced.

Key limitations: One supply system, sampling period, size window, recovery and contamination control limit transfer to other networks; occurrence does not establish health effects.

Catalogue ID: STUDY-00857. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Xiangying Sun, Yunjie Zhu, Lihui An, Yan Liu, Yin Zhuang, Yubang Wang, Mingdong Sun & Qiuji Xu. 2024. Microplastic Transportation in a Typical Drinking Water Supply: From Raw Water to Household Water. Water 16(11):1567.

Design: Field study following microplastic transport from raw water through a drinking-water plant and household delivery.

Findings: Particle numbers and characteristics changed across treatment and distribution, showing that plant removal and downstream sources both influence household water.

Risk relevance: Provides integrated source-to-tap occurrence evidence.

Key limitations: System-specific treatment, pipe materials, sampling, cutoff and identification methods prevent a universal release or exposure estimate.

Catalogue ID: STUDY-00865. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Xu, et al. (2024). Mass Concentration and Removal Characteristics of Microplastics and Nanoplastics in a Drinking Water Treatment Plant. ACS ES&T Water. <https://doi.org/10.1021/acsestwater.4c00222>.

Design: Drinking-water treatment-plant study measuring polymer-associated mass concentration and removal characteristics of micro- and nanoplastics.

Findings: The study reported mass-oriented concentrations and treatment-stage changes, offering a different measurand from particle counts.

Risk relevance: Useful for mass-based exposure context and treatment evaluation.

Key limitations: Thermal or mass-marker analysis may not verify intact particle number, size or morphology; matrix interference, recovery and plant representativeness remain important.

Catalogue ID: STUDY-00492. DOI/link: 10.1021/acsestwater.4c00222 Verification: V3.

Jamison Hart, M. N., & Lenhart, J. J. (2026). What's in your water? A comparative analysis of micro- and nanoplastics in treated drinking water and bottled water. Science of the Total Environment, 1011, 181148.

<https://doi.org/10.1016/j.scitotenv.2025.181148>.

Design: Comparative occurrence study using scanning electron microscopy and optical photothermal infrared spectroscopy on samples from four drinking-water treatment plants and six bottled-water brands, covering approximately 300 nm to 42.3 micrometres.

Findings: Bottled water contained significantly higher micro- and nanoplastic particle concentrations than treated drinking water, particularly in smaller size classes. PA, PET and PE were prominent; rubber was also reported, but rubber is not classified as plastic in this report.

Risk relevance: Extends the observed drinking-water size window and provides a direct treated-versus-bottled comparison.

Key limitations: Limited plants, brands and sampling occasions; method-specific particle recovery and classification; occurrence does not establish absorbed dose, retained burden or health risk.

Catalogue ID: Not assigned (supplementary source outside the frozen baseline). DOI/link: 10.1016/j.scitotenv.2025.181148 Verification: V2.

Human ingestion and integrated exposure studies

Cox, K.D., Covernton, G.A., Davies, H.L., Dower, J.F., Juanes, F., and Dudas, S.E. (2020). Correction to Human Consumption of Microplastics. Environmental Science & Technology, 54, 10974.

Design: Exposure-modeling study estimating human intake and/or internal burden from compiled occurrence data.

Findings: The model produced route- and size-dependent exposure estimates that were sensitive to assumed concentrations, particle geometry and pathway coverage.

Risk relevance: Provides quantitative context when the assumptions and compatible size domain are explicit.

Key limitations: Modeled input is not measured personal exposure; heterogeneous source studies, missing pathways and count-to-mass assumptions can dominate the result.

Catalogue ID: STUDY-00445. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Nur Hazimah Mohamed Nor, Merel Kooi, Noël J. Diepens, and Albert A. Koelmans. 2021. Lifetime Accumulation of Microplastic in Children and Adults.

Design: Integrated probabilistic exposure model combining multiple food categories and inhalation across a harmonized 1-5,000 micrometre domain.

Findings: The model estimated median adult intake of 883 particles/day and 583 ng/day and modeled a much smaller retained burden for a narrower size domain.

Risk relevance: The report's principal integrated external-exposure benchmark and a basis for bounded scale comparisons.

Key limitations: Inputs are heterogeneous and modeled rather than measured total personal exposure; pathway coverage, lower size cutoff, geometry, density and retention assumptions remain important.

Catalogue ID: STUDY-00448. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Schwabl, P., Köppel, S., Königshofer, P., Bucsics, T., Trauner, M., Reiberger, T., & Liebmann, B. (2019). Detection of various microplastics in human stool: a prospective case series. Annals of Internal Medicine, 171(7), 453–457.

<https://doi.org/10.7326/M19-0618>.

Design: Prospective case series examining microplastics in stool from human participants using digestion and spectroscopic identification.

Findings: Multiple polymer types were reported in stool samples, demonstrating ingestion and gastrointestinal passage.

Risk relevance: Supports the narrow conclusion that ingested polymer particles can be excreted.

Key limitations: Very small sample, no representative exposure assessment, contamination sensitivity and restricted analytical window prevent estimates of absorption, retained dose or health effects.

Catalogue ID: STUDY-00904. DOI/link: 10.7326/M19-0618 Verification: V3.

University of Newcastle and World Wide Fund for Nature. 2019. Assessing Plastic Ingestion from Nature to People. Commissioned quantitative analysis.

Design: Commissioned synthesis estimating human plastic ingestion from published food, water and air studies.

Findings: The analysis produced the widely publicized credit-card-per-week estimate by combining heterogeneous counts and assumptions.

Risk relevance: Historically important exposure communication and a stress test for count-to-mass conversion.

Key limitations: Incompatible source methods, incomplete pathways, uncertain geometry and density and unvalidated conversion assumptions make the headline mass estimate unreliable as measured intake.

Catalogue ID: STUDY-01382. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Human inhalation and respiratory-deposition studies

Lauren C. Jenner, Jeanette M. Rotchell, Robert T. Bennett, Michael Cowen, Vasileios Tentzeris & Laura R. Sadofsky. 2022. Detection of microplastics in human lung tissue using micro-FTIR spectroscopy. Science of the Total Environment 831:154907.

Design: Human lung-tissue case series using digestion, filtration and micro-FTIR to identify suspected microplastics.

Findings: Polymer-assigned particles were detected in selected lung samples, with a blank-adjusted mean used in the report as an occurrence anchor.

Risk relevance: Supports a narrow statement that some human lung samples can contain spectroscopically assigned particles.

Key limitations: Selected surgical samples, small numbers, contamination sensitivity, no exposure history or disease comparator and no inference of retained dose, accumulation or causation.

Catalogue ID: STUDY-00369. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Luís Fernando Amato-Lourenço, Regiani Carvalho-Oliveira, Gabriel Ribeiro Júnior, et al. 2021. Presence of airborne microplastics in human lung tissue. Journal of Hazardous Materials 416:126124.

Design: Human lung-tissue occurrence study using microscopy and spectroscopic polymer identification on selected surgical samples.

Findings: Spectroscopically assigned polymer particles were reported in some lung samples, supporting a plausible inhalation-deposition pathway.

Risk relevance: Provides narrow human respiratory-occurrence evidence, not proof of accumulation, disease or causal pulmonary toxicity.

Key limitations: Small selected sample, contamination sensitivity, limited exposure histories, no healthy population comparator and a method-defined size window.

Catalogue ID: STUDY-00257. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Hayden, L., Strausborger, S., & Lewin-Smith, M. (2023). 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), 235-243.

<https://doi.org/10.1093/micmic/ozac015>.

Design: Primary human lung-tissue methods/occurrence study using automated in-situ FE-SEM with EDS on 38 surgical biopsy specimens from deployed and nondeployed U.S. military service members. Approximately 300 fields covering a 1 x 1 mm tissue area were analyzed per specimen with a practical feature cutoff of about 0.17 micrometres.

Findings: A total of 124,018 particles were characterized by chemistry and morphology and assigned to 45 particle or mineral classes and 12 groups. Common classes were clays, feldspars, quartz, titanium dioxide and metal/metal-oxide particles. Cerium, rare-earth, tin, antimony, copper, zinc and steel-like particles were identified, with rare lead, uranium and thorium. More than 90% of particles had a greatest dimension below 2.5 micrometres.

Risk relevance: Provides direct route-matched evidence that selected human lung tissue can contain very large numbers and a wide chemical diversity of nonplastic inorganic particles. This supplies essential context for interpreting any plastic lung detection: occurrence of one material should be evaluated against the broader particulate mixture rather than in isolation. The study strengthens comparative-particle and analytical-method context; it does not measure microplastics, quantify a plastic fraction or establish a health effect.

Key limitations: Selected clinical military cohort rather than a representative healthy population; standardless FE-SEM/EDS was described as qualitative rather than quantitative; fixed-area counts are not a tissue-mass burden; backscattered-electron thresholding favored particles with average atomic number above carbon; clusters and overlapping particles produced mixed spectra and lower-limit counts; EDS alone could not uniquely resolve geological versus manufactured source.

Catalogue ID: Not assigned (supplementary primary context outside the frozen baseline). DOI/link: 10.1093/micmic/ozac015. Verification: V1.

Miller, M. R., Raftis, J. B., Langrish, J. P., et al. (2017). Inhaled Nanoparticles Accumulate at Sites of Vascular Disease. ACS Nano, 11(5), 4542-4552. <https://doi.org/10.1021/acsnano.6b08551>. Correction: ACS Nano, 11, 10623-10624.

<https://doi.org/10.1021/acsnano.7b06327>.

Design: Controlled human inhalation and complementary mouse study using analytically distinctive gold nanoparticles. Fourteen healthy male volunteers inhaled approximately 3.8-5 nm primary gold particles for 2 hours with intermittent exercise; a separate size-comparison study contrasted approximately 4 nm with approximately 34 nm primary particles. Three carotid-endarterectomy patients inhaled approximately 5 nm gold for 4 hours the day before surgery. HR-ICPMS measured gold in blood, urine and tissues, while Raman microscopy and electron microscopy provided selected particle localization. Mice received 2-200 nm gold preparations to define size dependence.

Findings: Gold appeared in systemic samples after controlled inhalation and was detectable in blood in 12 of 14 volunteers at 24 hours. Systemic appearance was much greater for the smaller human exposure and fell strongly with increasing primary particle size in mice. Gold localized preferentially to atherosclerotic vascular regions in the mouse model, and Raman microscopy detected gold-associated signal in carotid plaques from the three exposed surgical patients but not non-exposed controls. The published correction changed the authors' illustrative 24-hour translocation estimate from approximately 0.2% to approximately 0.02% of inhaled mass after correcting units and exposure calculations.

Risk relevance: High-value route-specific kinetic evidence that a small fraction of ultrasmall inhaled nanoparticles can cross the pulmonary barrier in humans and that translocation is strongly size-dependent. It supplies a positive benchmark for biological plausibility while constraining extrapolation: few-nanometre particles are not kinetically equivalent to conventional micrometre-scale microplastics. The gold tracer was selected for low biological activity, so the study does not establish toxicity from translocation.

Key limitations: The corrected approximately 0.02% value is an illustrative 24-hour estimate rather than a closed mass balance; alveolar deposition, non-urinary clearance, hepatic uptake and continued slow transfer were not fully quantified. HR-ICPMS establishes systemic gold after tissue digestion but does not by itself prove intact-particle form, although Raman/electron microscopy support

particulate gold in selected tissues. The healthy-volunteer groups were small, the carotid-plaque human arm included only three exposed patients, exposure was deliberate and not ordinary environmental microplastic exposure, and repeated inert-gold dosing did not increase plaque size in the mouse experiment. No microplastic-specific health effect is demonstrated.

Catalogue ID: Not assigned (supplementary primary kinetic/context publication lineage outside the frozen baseline). DOI/link: 10.1021/acsnano.6b08551; correction 10.1021/acsnano.7b06327. Verification: V1. Main article, correction and supplied supporting information reviewed as one publication lineage.

Absorption, clearance and retention studies

Garcia, M. A., et al. (2024). In Vivo Tissue Distribution of Polystyrene or Mixed Polymer Microspheres and Metabolomic Analysis after Oral Exposure in Mice. Environmental Health Perspectives, 132. <https://doi.org/10.1289/EHP13435>.

Design: Controlled oral-exposure study in mice comparing tissue distribution of polystyrene or mixed-polymer microspheres and metabolomic responses.

Findings: Particle-associated signals and metabolomic changes varied by preparation and tissue under the administered conditions.

Risk relevance: Provides mechanistic distribution and hazard evidence.

Key limitations: Administered dose, model spheres, uncertain distinction between intact particles and labels, and absence of matched nonplastic controls limit relevance to ordinary exposure.

Catalogue ID: STUDY-00711. DOI/link: 10.1289/EHP13435 Verification: V3.

Letwin, A., et al. (2026). Assessing the accumulation of microplastics in earthworms (*Eisenia fetida*) using traditional bioaccumulation modeling and synchrotron-based microcomputed tomography. Environmental Toxicology and Chemistry, 45, 1553–1566. <https://doi.org/10.1093/etjnl/vgag072>.

Design: Earthworm uptake study combining conventional bioaccumulation modeling with synchrotron microcomputed tomography.

Findings: The study evaluated particle ingestion, distribution and elimination and tested whether conventional bioaccumulation concepts adequately describe microplastics.

Risk relevance: Provides terrestrial kinetic and method evidence.

Key limitations: Exposure concentration, soil matrix, imaging resolution and distinction between gut contents and tissue translocation constrain claims of bioaccumulation.

Catalogue ID: STUDY-00626. DOI/link: 10.1093/etjnl/vgag072 Verification: V3.

Sandrine Straub, Philipp E. Hirsch and Patricia Burkhardt-Holm. 2017. Biodegradable and Petroleum-Based Microplastics Do Not Differ in Their Ingestion and Excretion but in Their Biological Effects in a Freshwater Invertebrate *Gammarus fossarum*.

Design: Four-week freshwater amphipod study comparing biodegradable PHB, petroleum-based PMMA and silica reference particles.

Findings: Both plastics were ingested and egested; ingestion and excretion were similar, while some biological responses differed among particle types.

Risk relevance: Strong comparative design that helps separate polymer-dependent from generic particle responses.

Key limitations: Exposure level, one species, modest effects and imperfect particle matching limit field transfer.

Catalogue ID: STUDY-01281. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Sijoon Lee, Dongseon Kim, Kyung-Ku Kang, et al. 2023. Toxicity and biodistribution of fragmented polypropylene microplastics in ICR mice. International Journal of Molecular Sciences 24:8463.

Design: Repeated oral toxicity study of fragmented polypropylene microplastics in ICR mice, including clinical, pathology and laboratory endpoints.

Findings: No significant treatment-related toxicity was reported up to the highest tested dose of 2,000 mg/kg/day under the study conditions.

Risk relevance: Provides a study-specific high-dose-without-detected-adverse-effects anchor.

Key limitations: Polymer, formulation, particle size, route, duration and endpoints are study-specific; it is not a universal threshold or formal margin of exposure.

Catalogue ID: STUDY-00809. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Yongfeng Deng, Yili Zhang, Bernardo Lemos & Hongqi Ren. 2017. Tissue accumulation of microplastics in mice and biomarker responses suggest widespread health risks of exposure. Scientific Reports 7:46687.

Design: Controlled mouse study of orally administered polystyrene microplastics examining tissue distribution and biological endpoints.

Findings: Particle-associated signals and biological changes were reported under high experimental exposure.

Risk relevance: Mechanistic hazard evidence under the administered conditions.

Key limitations: High dose, simplified pristine polystyrene, uncertain intact-particle tracking and no matched nonplastic comparator prevent direct inference to ordinary human risk.

Catalogue ID: STUDY-00589. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Human tissue, biomonitoring and occurrence studies

Amato-Lourenço, L. F., Dantas, K. C., Ribeiro Júnior, G., Paes, V. R., Ando, R. A., de Oliveira Freitas, R., da Costa, O. M. M., Rabelo, R. S., Soares Bispo, K. C., Carvalho-Oliveira, R., & Mauad, T. (2024). Microplastics in the Olfactory Bulb of the Human Brain. JAMA Network Open, 7(9), e2440018. <https://doi.org/10.1001/jamanetworkopen.2024.40018>.

Design: Cross-sectional autopsy case series of olfactory-bulb tissue from 15 adults, using tissue digestion, filtration and micro-FTIR particle assignment with procedural and negative controls.

Findings: Sixteen synthetic-polymer objects were reported in eight of 15 olfactory bulbs. The result supports the narrow occurrence conclusion that spectroscopically assigned polymer particles can be present in some samples.

Risk relevance: Relevant to internal-occurrence mapping and route hypotheses. It does not establish exposure route, dose-response, neurotoxicity, disease association or clinical causation.

Key limitations: Small selected case series; no exposure histories, population comparator or disease comparison; no longitudinal evidence; restricted analytical size window; and only a small fraction of the total particulate burden was characterized.

Catalogue ID: STUDY-00001. DOI/link: 10.1001/jamanetworkopen.2024.40018 Verification: V3.

Bissonnette, J. R., Harvey, N. E., Rowsell, M. L., Kieffe, S., Houthuijs, K. J., Been, F. M., Lamoree, M. H., Cahill, L. S., & Jobst, K. J. (2025). Identification of micro-/nanoplastics in human placental blood using comprehensive multidimensional pyrolysis-gas chromatography × ion mobility mass spectrometry. *Analytica Chimica Acta* 1376, 344606. <https://doi.org/10.1016/j.aca.2025.344606>.

Design: Human blood analytical study using high-resolution mass-spectrometric methods to identify micro-/nanoplastic-associated markers.

Findings: The study reported candidate polymer-related signals while also demonstrating the complexity of biological-matrix identification.

Risk relevance: Relevant to biomonitoring method development and the credibility of blood occurrence claims.

Key limitations: Marker detection does not automatically establish intact particles, size, number or location; contamination, matrix interference, recovery and orthogonal validation remain decisive.

Catalogue ID: STUDY-01654. DOI/link: 10.1016/j.aca.2025.344606 Verification: V3.

Cassandra Rauert, et al. 2025. Assessing the Efficacy of Pyrolysis-Gas Chromatography-Mass Spectrometry for Nanoplastic and Microplastic Analysis in Human Blood. *Environmental Science & Technology* 59:1984–1994.

Design: Human-blood analytical-validation study assessing Py-GC-MS for micro- and nanoplastic measurement in a complex biological matrix.

Findings: The work identified substantial method and matrix challenges and evaluated whether reported polymer signals could be measured reliably in blood.

Risk relevance: Directly relevant to the credibility of blood polymer-marker claims.

Key limitations: Py-GC-MS destroys particles and cannot establish intact-particle number, size or location; polyethylene and other matrix-derived interferences require validated cleanup and orthogonal confirmation.

Catalogue ID: STUDY-00466. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Dzierżyński, E., Blicharz-Grabias, E., Komaniecka, I., Panek, R., Forma, A., Gawlik, P. J., Puźniak, D., Flieger, W., Choma, A., Suśniak, K., Teresiński, G., Baj, J., Kupisz, K., & Flieger, J. (2025). Post-mortem evidence of microplastic bioaccumulation in human organs: insights from advanced imaging and spectroscopic analysis. *Archives of Toxicology*, 99, 4051–4066. <https://doi.org/10.1007/s00204-025-04092-2>.

Design: Single-donor post-mortem study examining seven organs using O-PTIR, DLS and MALDI-TOF-related analytical approaches.

Findings: Polymer-associated signals were reported across selected tissues, providing exploratory occurrence and method-development evidence.

Risk relevance: Generates hypotheses about tissue occurrence and analytical capability.

Key limitations: One donor cannot establish population prevalence; the methods do not collectively prove sub-20-nm intact particles, barrier transport, organ bioaccumulation or toxicity.

Catalogue ID: STUDY-01677. DOI/link: 10.1007/s00204-025-04092-2 Verification: V3.

Marfella, R., Prattichizzo, F., Sardu, C., et al. (2024). Microplastics and nanoplastics in atheromas and cardiovascular events. *New England Journal of Medicine*, 390, 900–910. <https://doi.org/10.1056/NEJMoa2309822>.

Design: Prospective observational study of patients undergoing carotid endarterectomy, measuring polymer-associated material in atheroma and following cardiovascular outcomes.

Findings: Polymer-associated signals in plaques were associated with a higher subsequent composite cardiovascular-event rate.

Risk relevance: Important human association evidence that warrants replication and stronger exposure validation.

Key limitations: Observational design cannot establish causation; exposure preceded neither plaque formation nor disease measurement, residual confounding is likely, and destructive polymer analysis did not verify intact particles or dose.

Catalogue ID: STUDY-00602. DOI/link: 10.1056/NEJMoa2309822 Verification: V3.

Montano, L., et al. (2023). Raman microspectroscopy evidence of microplastics in human semen. *Science of the Total Environment*, 901, 165922. <https://doi.org/10.1016/j.scitotenv.2023.165922>.

Design: Pilot human semen study using Raman microspectroscopy to examine suspected microplastic particles.

Findings: Raman-assigned particles were reported in selected semen samples.

Risk relevance: Supports bounded occurrence surveillance only.

Key limitations: Small convenience sample, contamination and matrix-interference risk, restricted analytical window, no exposure histories and no causal reproductive endpoint.

Catalogue ID: STUDY-00385. DOI/link: 10.1016/j.scitotenv.2023.165922 Verification: V3.

Zhao, Q., et al. (2023). Detection and characterization of microplastics in the human testis and semen. *Science of the Total Environment*, 877, 162713. <https://doi.org/10.1016/j.scitotenv.2023.162713>.

Design: Human testis and semen occurrence study using biological-matrix preparation and particle identification methods.

Findings: The authors reported polymer-associated particles or signals in selected testis and semen samples.

Risk relevance: Generates a reproductive-tissue occurrence hypothesis; it does not establish impaired fertility, exposure route or causal disease.

Key limitations: Small cross-sectional sample, difficult contamination control, uncertain recovery and identity, no representative comparator population and no dose-response evidence.

Catalogue ID: STUDY-00379. DOI/link: 10.1016/j.scitotenv.2023.162713 Verification: V3.

Analytical-method, validation and false-positive studies

Lane, T., de Ruijter, V. N., Wardani, I., Barchiesi, M., & Koelmans, A. A. (2026). A QA/QC screening framework to assess studies reporting microplastic presence in human organs, tissues, and bodily fluids. *Environment International*, 214, 110429. <https://doi.org/10.1016/j.envint.2026.110429>.

Design: Methodological review and fit-for-risk-assessment audit developing 14 QA/QC criteria and applying them to 133 datasets from 101 peer-reviewed studies of human organs, tissues and bodily fluids.

Findings: No dataset met all essential criteria for direct quantitative risk assessment. The mean was 12.8 of 28 points and 5.8 zero-scored criteria; the most common gaps involved positive recovery controls, particle size and shape, clean-air conditions, sample representativeness, digestion and blank/LOD/LOQ procedures.

Risk relevance: Provides the strongest independent cross-check identified for judging whether human biomonitoring measurements can quantify population body burden, organ distribution and particle-specific internal dose. It supports bounded occurrence use for selected studies while rejecting direct quantitative risk use of the current literature as a whole.

Key limitations: The any-zero rule is deliberately strict and assesses fitness for quantitative risk assessment, not publication quality or whether every detection is false. The search covered studies available through April 2025, did not conduct a formal causal risk-of-bias assessment, and includes some subjective scoring and minor 130-versus-133 wording inconsistency; the abstract, principal results and scoring workbook use 133 datasets.

Catalogue ID: Not assigned (supplementary source outside the frozen baseline). DOI/link: 10.1016/j.envint.2026.110429 Verification: V1.

Cassandra Rauert, Yufei Pan & Kevin V. Thomas. 2022. Extraction and Pyrolysis-GC-MS analysis of polyethylene in samples with medium to high lipid content. *Journal of Environmental Exposure Assessment* 1:13.

Design: Method-development study for extraction and Py-GC-MS analysis of polyethylene in medium- to high-lipid samples.

Findings: Optimized preparation reduced lipid interference and demonstrated how matrix cleanup alters apparent polyethylene measurement.

Risk relevance: Critical validation evidence for thermal analysis in food and biological matrices.

Key limitations: The method measures polymer-associated thermal products rather than intact particle number, morphology or location; validation remains matrix- and marker-specific.

Catalogue ID: STUDY-01619. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Dmitri Ciornii, Vladimir-Dan Hodoroaba, Nizar Benismail, et al. 2025. Interlaboratory Comparison Reveals State of the Art in Microplastic Detection and Quantification Methods. *Analytical Chemistry* 97:8719-8728.

Design: Interlaboratory comparison of microplastic detection and quantification methods using common test materials or samples.

Findings: Participating laboratories produced variable results, revealing the current state of method performance and major sources of between-laboratory error.

Risk relevance: Strong evidence for quality-control requirements, harmonization and cautious cross-study pooling.

Key limitations: Performance on selected comparison samples does not guarantee equal accuracy in all matrices, polymers or submicrometre domains.

Catalogue ID: STUDY-01624. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Jüngling, I. S., Schmitt, L., De Franceschi, F., et al. (2026). Challenges and solutions in the analysis of micro- and nanoplastics down to 500 nm with automated Raman microspectroscopy: suitable filters, accuracy in detection, identification and quantification. *Analytical and Bioanalytical Chemistry*. <https://doi.org/10.1007/s00216-026-06567-2>.

Design: Analytical-method study evaluating automated Raman microspectroscopy for micro- and nanoplastics down to approximately 500 nm, including filter selection, detection, identification and quantification.

Findings: Validation with 500 nm polystyrene beads yielded $67 \pm 10\%$ recovery. In three proof-of-principle potable-water samples, $0.36 \pm 0.13\%$ of particles in the 0.5-10 micrometre range were identified as plastic, with approximately 18% of the identified plastics below 1 micrometre.

Risk relevance: Defines what automated Raman analysis can and cannot establish and supports better exposure measurement; detection alone does not demonstrate harm.

Key limitations: Particle size was overestimated by approximately 0.5 ± 0.26 micrometres under the tested illumination conditions; measurement required about three days per water sample; low plastic counts, blank similarity, filter dependence and one selected water source prevent generalization to drinking-water composition.

Catalogue ID: STUDY-00029. DOI/link: 10.1007/s00216-026-06567-2 Verification: V1.

Aves, A., MacDonald, A., Naeher, S., et al. (2026). Comparative assessment of five analytical methods for airborne microplastics highlights importance of identifying sub-10 micrometre methods. *Scientific Reports*, 16, 22321. <https://doi.org/10.1038/s41598-026-49405-2>.

Design: The same remote-air filter samples were analyzed by micro-FTIR, micro-Raman, Nile Red fluorescence microscopy, pyrolysis-GC/MS and thermal-desorption proton-transfer-reaction mass spectrometry.

Findings: Every method produced a different concentration and polymer profile; 98% of particle-resolved detections were below 10 micrometres, and Nile Red suspected-particle counts exceeded micro-Raman counts by more than 13-fold.

Risk relevance: Directly demonstrates method-, size-window-, measurand- and calibration-dependent divergence and supports the rule against pooling unlike analytical constructs.

Key limitations: One remote site, subsampling and extrapolation from filters, unequal method size windows and different reporting units prevent ranking the techniques as though they measured the same quantity.

Catalogue ID: Not assigned (supplementary source outside the frozen baseline). DOI/link: 10.1038/s41598-026-49405-2 Verification: V1.

Clough, M. E., et al. (2026). Avoiding and reducing microplastic false positives from dry glove contact. Analytical Methods, 18, 2914-2926. <https://doi.org/10.1039/D5AY01801C>.

Design: Controlled laboratory study examining residues transferred by dry contact from seven types of nitrile and latex gloves and evaluating Raman or infrared spectral-library misclassification.

Findings: Stearate-rich glove residues could be misidentified as microplastics. Traditional library matching produced a mean of approximately 2,000 apparent false positives per square millimetre across the tested gloves; a cleanroom nitrile glove produced approximately 100 per square millimetre.

Risk relevance: Provides direct empirical evidence that routine handling materials can create large false-positive burdens in small-particle workflows.

Key limitations: Transfer depends on glove type, pressure, surface and workflow. The findings do not imply that all glove use invalidates results; they require contact controls, complete procedural blanks and validated spectral decision rules.

Catalogue ID: Not assigned (supplementary source outside the frozen baseline). DOI/link: 10.1039/D5AY01801C Verification: V1.

Mirka Lares. 2019. Intercomparison study on commonly used methods to determine microplastics in wastewater and sludge samples. Environ Sci Pollut Res.

Design: Intercomparison of commonly used methods for determining microplastics in wastewater and sludge samples.

Findings: Methods produced materially different recoveries and counts, demonstrating that extraction and identification choices can dominate reported concentrations.

Risk relevance: High-value evidence for method compatibility and data-quality grading.

Key limitations: Intercomparison performance with selected reference materials and matrices does not validate all environmental samples or smaller size ranges.

Catalogue ID: STUDY-01472. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Stoyana Peneva. 2025. Microplastic analysis in soils: A comparative assessment. Ecotoxicology and Environmental Safety, 289 (2025) 117428.

Design: Comparative assessment of extraction and analytical methods for microplastics in soils.

Findings: Methods differed in recovery, interference and practical performance, demonstrating that soil measurements depend strongly on preparation and identification choices.

Risk relevance: Supports method selection and comparability grading for terrestrial occurrence studies.

Key limitations: Performance with selected soils, polymers and size ranges does not establish universal recovery, especially for aged, small or strongly mineral-associated particles.

Catalogue ID: STUDY-00137. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Yingnan Liu, Jie Han, Yanhua Wang, Aoze Li, Jian Zhao, Yu Su, Lezu Shen & Baoshan Xing. 2024. Suspected sources of microplastics and nanoplastics: Contamination from experimental reagents and solvents. Water Research 249:120925.

Design: Contamination-source study examining micro- and nanoplastic release from experimental reagents, consumables or laboratory procedures.

Findings: The authors identified laboratory materials as plausible sources of particles or polymer signals that can contaminate experiments.

Risk relevance: Directly informs false-positive control and procedural blank design.

Key limitations: Source characterization does not quantify contamination in every laboratory; study-specific consumables and methods require local verification.

Catalogue ID: STUDY-01622. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Animal toxicology and mechanistic hazard studies

Bacchetta, R., Pica, A., Santo, N., Tremolada, P., & Sugni, M. (2024). Comparative effects of polyvinyl chloride microplastics on the brittle star Ophiactis virens and the amphibian Xenopus laevis. Aquatic Toxicology, 272, 106975. <https://doi.org/10.1016/j.aquatox.2024.106975>.

Design: Comparative PVC-microplastic exposure study in a brittle star and an amphibian, evaluating organism-specific endpoints.

Findings: Responses differed between species and endpoints under the administered conditions.

Risk relevance: Provides comparative organism-level hazard evidence for a PVC preparation.

Key limitations: Laboratory dose, formulation, species sensitivity and absence of matched nonplastic particle controls limit environmental and plastic-specific inference.

Catalogue ID: STUDY-00942. DOI/link: 10.1016/j.aquatox.2024.106975 Verification: V3.

Fatima, R., Parveen, K., Anjum, S., Abduh, M. S., Zaman, A., Naseer, F., & Ahmad, T. (2026). Dose-Dependent Toxicological Effects of Polyvinyl Chloride and Polystyrene Microplastics on Wistar Albino Rats. Journal of Applied Toxicology. <https://doi.org/10.1002/jat.70080>.

Design: Eight-week controlled Wistar-rat experiment administering PVC and polystyrene microplastics and evaluating body weight, blood chemistry, oxidative-stress and histopathology endpoints.

Findings: The authors reported dose-related biomarker and histological changes, concentrated at the highest administered dose; several outcomes were not significant at 300 mg/kg.

Risk relevance: Hazard evidence for very high administered doses. It does not establish risk at measured ordinary human exposure.

Key limitations: Doses of 300-1,000 mg/kg are orders of magnitude removed from plausible human intake; there was no matched mineral or other nonplastic particle comparator, and formulation and particle-size representativeness were limited.

Catalogue ID: STUDY-00010. DOI/link: 10.1002/jat.70080 Verification: V3.

Horn, D. A., Granek, E. F., & Steele, C. L. (2020). Effects of environmentally relevant concentrations of microplastic fibers on Pacific mole crab (*Emerita analoga*) mortality and reproduction. *Limnology and Oceanography Letters*, 5(1), 74–83. <https://doi.org/10.1002/lol2.10137>.

Design: Pacific mole-crab exposure study testing environmentally described concentrations of microplastic fibres and measuring mortality and reproduction.

Findings: The study reported endpoint-specific responses under the tested fibre concentrations.

Risk relevance: Provides organism-level hazard evidence using a fibre exposure framed as environmentally relevant.

Key limitations: Environmental comparability depends on fibre identity, size, mass, food conditions and local concentration; no matched natural-fibre control established plastic specificity.

Catalogue ID: STUDY-01593. DOI/link: 10.1002/lol2.10137 Verification: V3.

Ilechukwu, I., Ehigiator, B.E., Ben, I.O., Okonkwo, C.J., Olorunfemi, O.S., Modo, U.E., Ilechukwu, C.E., & Ohagwa, N.J. (2022). Chronic toxic effects of polystyrene microplastics on reproductive parameters of male rats. *Environmental Analysis Health and Toxicology*, 37(2), e2022015. <https://doi.org/10.5620/eaht.2022015>.

Design: Chronic oral rat study examining reproductive endpoints after polystyrene-microplastic exposure.

Findings: The authors reported changes in selected male reproductive parameters under the administered regimen.

Risk relevance: Supports a reproductive-hazard hypothesis in an animal model.

Key limitations: Dose realism, particle preparation, sample size, endpoint multiplicity and lack of a matched nonplastic comparator prevent direct inference to human reproductive risk.

Catalogue ID: STUDY-00892. DOI/link: 10.5620/eaht.2022015 Verification: V3.

Ting-An Lin, et al. 2025. Microplastic exposure elicits sex-specific atherosclerosis development in lean low-density lipoprotein receptor-deficient mice. *Environment International*:109938.

Design: Controlled study in lean LDL-receptor-deficient mice examining sex-specific atherosclerosis outcomes after administered microplastic exposure.

Findings: The study reported sex-dependent changes in atherosclerosis-related outcomes under the administered regimen.

Risk relevance: Supports a disease-mechanism hypothesis in a susceptible animal model, not a population risk estimate.

Key limitations: The disease-prone model and regimen were remote from ordinary exposure and no equal-dose matched nonplastic particle comparator isolated a plastic-specific effect.

Catalogue ID: STUDY-00165. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Valerie Stock. 2021. Uptake and cellular effects of PE, PP, PET and PVC microplastic particles. *Toxicology in Vitro*, 70 (2020) 105021.

Design: In-vitro comparison of uptake and cellular effects of PE, PP, PET and PVC microplastic preparations.

Findings: Cellular interaction and response varied by polymer and preparation under direct experimental loading.

Risk relevance: Supports mechanistic hazard screening and demonstrates that one model polymer cannot represent all materials.

Key limitations: High cell-surface concentrations, simplified preparations and absence of matched nonplastic particles prevent direct inference to ordinary human exposure or plastic-specific toxicity.

Catalogue ID: STUDY-00406. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Standardized oral toxicology studies

Buesen, R., Vogel, S., Thoma, T., Strauss, V., Gröters, S., Becker, K., Müller, F., Gründling, T., Pfohl, P., Vogel, A., Haase, A., & Wohlleben, W. (2025). Oral toxicity of small microplastic of polyamide assessed by a standardized guideline study. *Microplastics and Nanoplastics*, 5, 31. <https://doi.org/10.1186/s43591-025-00137-6>.

Design: GLP OECD TG 407 repeated-dose oral toxicity study of a small polyamide-6 microplastic preparation in rats.

Findings: No treatment-related local or systemic adverse effects were reported at the 1,000 mg/kg/day limit dose under the study conditions.

Risk relevance: A strong standardized PA-6 no-adverse-effect anchor; complementary to the longer PE/PET dietary study and the PTFE/PP repeated-dose evidence.

Key limitations: The result is specific to PA-6, its preparation, oral route, 28-day duration and measured endpoints; it is not a universal safety threshold.

Catalogue ID: STUDY-01639. DOI/link: 10.1186/s43591-025-00137-6 Verification: V3.

Kim, J. S. (2020). Acute toxicity evaluation of polypropylene and polystyrene microplastics in Sprague-Dawley rats after oral administration. *Journal of Pharmacological and Toxicological Methods*, 105, 106813. Conference abstract. <https://doi.org/10.1016/j.vascn.2020.106813>.

Design: Conference-abstract metadata identify an acute oral evaluation of polypropylene and polystyrene microplastics in Sprague-Dawley rats. The complete abstract or source-level study report was not recovered.

Findings: The accessible title and bibliographic metadata do not verify the administered dose, test-material preparation, measured endpoints or outcome results for PS.

Risk relevance: Potential lead for acute non-bead PS evidence, but insufficient for a numerical no-effect value, a repeated-dose NOAEL or dose-scale comparison.

Key limitations: Conference abstract not accessible at source level; no verified dose, characterization, endpoint tables or results. It must not be conflated with Kim et al. 2021, which tested only weathered fragmented PP.

Catalogue ID: STUDY-00415. DOI/link: 10.1016/j.vascn.2020.106813 Verification: V2 title, DOI and conference-abstract metadata verified; quantitative findings not verified.

Kim, J., Maruthupandy, M., An, K. S., Lee, K. H., Jeon, S., Kim, J.-S., & Cho, W.-S. (2021). Acute and subacute repeated oral toxicity study of fragmented microplastics in Sprague-Dawley rats. *Ecotoxicology and Environmental Safety*, 228, 112964. <https://doi.org/10.1016/j.ecoenv.2021.112964>.

Design: GLP OECD TG 425 acute and TG 407 28-day repeated oral studies of weathered fragmented PP in Sprague-Dawley rats. Bulk PP resin was cryogenically milled, sieved, heated and UV-B weathered; particles were irregular, 40.1-163 micrometres, mean 85.2 micrometres. Repeated doses were 0, 6.25, 12.5 and 25 mg/kg/day.

Findings: No treatment-related mortality, clinical signs, body-weight or food-consumption changes, hematological or serum-chemistry effects, coagulation or urine changes, organ-weight effects, gross or microscopic lesions, or bone-marrow micronucleus response were detected. The formal study NOAEL is 25 mg/kg/day, the highest feasible dose tested.

Risk relevance: Direct, guideline-based oral evidence using irregular weathered fragments derived from bulk PP rather than monodisperse laboratory spheres.

Key limitations: Four-week duration and one PP resin/preparation; 25 mg/kg/day was limited by dispersibility. The abstract wording "higher than 25 mg/kg/day" indicates that the true threshold may exceed the tested dose, but the experimentally supported formal NOAEL is 25 mg/kg/day because no higher dose was administered.

Catalogue ID: STUDY-00128. DOI/link: 10.1016/j.ecoenv.2021.112964 Verification: V1 primary full text and supporting information reviewed.

Merski, J. A., Johnson, W. D., Muzzio, M., Lyang, N.-L., & Gaworski, C. L. (2008). Oral toxicity and bacterial mutagenicity studies with a spunbond polyethylene and polyethylene terephthalate polymer fabric. *International Journal of Toxicology*, 27(5), 387-395. <https://doi.org/10.1080/10915810802408729>.

Design: OECD/GLP 13-week dietary study in Sprague-Dawley rats using cryogenically milled PE/PET fabric at 0.5%, 2.5% and 5% target dietary concentrations.

Findings: No apparent target-organ toxicity was detected at 5% target diet (4.7% measured). For a 50:50 composition, the high dose contained 2.5% target PE plus 2.5% target PET, approximately 2.35% measured each, corresponding to roughly 1.6-2.0 g/kg-bw/day of each component administered together.

Risk relevance: The strongest longer-duration oral evidence containing both PE and PET and an important dose-scale anchor for common polymers.

Key limitations: Particle dimensions after milling were not measured, and PE and PET were not administered independently. The component doses are therefore mixture-qualified rather than standalone pure-polymer NOAELs.

Catalogue mapping unchanged in this manuscript-only correction. DOI/link: 10.1080/10915810802408729 Verification: V1 primary full text reviewed.

Lee, S., Kang, K.-K., Sung, S.-E., et al. (2022). Toxicity Study and Quantitative Evaluation of Polyethylene Microplastics in ICR Mice. *Polymers*, 14(3), 402. <https://doi.org/10.3390/polym14030402>.

Design: Twenty-eight-day oral gavage study of fragmented 10-50 micrometre PE in ICR mice at 500, 1,000 and 2,000 mg/kg/day.

Findings: Granulomatous lung findings were reported in females at 500 mg/kg/day and in both sexes at higher doses. A conventional reading gives a male highest tested no-adverse-effect dose of 500 mg/kg/day; a female NOAEL was not established within the tested range.

Risk relevance: Important contrary PE evidence showing that preparation- and pathology-specific results can differ from the 13-week PE/PET dietary study.

Key limitations: The presumed particles in lesions were not chemically co-localized, and the authors' "NOAEL less than 1,000/500 mg/kg" wording is not standard threshold terminology.

Catalogue mapping unchanged in this manuscript-only correction. DOI/link: 10.3390/polym14030402 Verification: V1 primary full text reviewed.

Lee, S., Kang, K.-K., Sung, S.-E., Choi, J.-H., Sung, M., Seong, K.-Y., Lee, J., Kang, S., Yang, S. Y., Lee, S., et al. (2022). In Vivo Toxicity and Pharmacokinetics of Polytetrafluoroethylene Microplastics in ICR Mice. *Polymers*, 14(11), 2220. <https://doi.org/10.3390/polym14112220>.

Design: Single-dose and 28-day repeated oral gavage studies of fragmented PTFE in two size ranges, approximately 5 micrometres and 10-50 micrometres, at 500, 1,000 and 2,000 mg/kg/day.

Findings: No treatment-related clinical, body-weight, organ-weight, clinical-pathology or histopathological changes were observed. The authors reported a NOAEL of at least 2,000 mg/kg/day for both particle sizes.

Risk relevance: Provides a direct common-polymer NOAEL and a second 2,000 mg/kg/day oral anchor independent of the fragmented-PP study.

Key limitations: The duration was four weeks and the result applies only to the tested PTFE preparations. Raman non-detection in blood after a single dose does not quantify uptake below the method limit.

Catalogue mapping unchanged in this manuscript-only correction. DOI/link: 10.3390/polym14112220 Verification: V1 primary full text reviewed.

Kim, D., Kim, D., Kim, H.-K., et al. (2025). Organ-specific accumulation and toxicity analysis of orally administered polyethylene terephthalate microplastics. *Scientific Reports*, 15, 6616. <https://doi.org/10.1038/s41598-025-91170-1>.

Design: Four-week oral mouse toxicity study of fragmented PET averaging approximately 5.6 micrometres at 1.75, 7 and 28 mg/kg/day, with a separate one-week biodistribution arm using Cy5.5-COOH-labelled PET prepared with DMSO.

Findings: The authors reported sex-specific NOAELs of 1.75 mg/kg/day in males and 7 mg/kg/day in females based on pulmonary granulomatous findings.

Risk relevance: Important contradictory PET evidence, but not a principal quantitative anchor because it conflicts sharply with the longer PE/PET dietary study and has conclusion-controlling pathology and identification limitations.

Key limitations: Particles in lesions were presumed rather than chemically co-localized; pooled-lung Raman was not lesion-specific; pathology was not quantitatively scored; one slide per organ was examined. The labelled biodistribution arm did not adequately exclude free dye or dye leaching, but it was separate from the apparently unlabelled toxicity arm, so DMSO/dye cannot be asserted as the cause of the toxicity finding.

Supplementary source used for manuscript correction; catalogue mapping not altered.

Welle, F., & Franz, R. (2018). Microplastic in bottled natural mineral water - literature review and considerations on exposure and risk assessment. *Food Additives & Contaminants: Part A*, 35(12), 2482-2492. <https://doi.org/10.1080/19440049.2018.1543957>.

Design: Secondary toxicological and exposure review. For standalone PE, it cites the Cosmetic Ingredient Review assessment, which reports a 90-day rat dietary study at 1.25%, 2.5% and 5% PE. The source chain ultimately points to Lefaux (1968), which cites Phillips and Marks (1961) as the underlying commercial-polymer source.

Findings: No adverse effects were reported at the highest 5% dietary concentration in the cited PE study.

Risk relevance: Provides a potentially important standalone-PE archival no-effect lead beyond the PE/PET mixture.

Key limitations: Welle and Franz and the Cosmetic Ingredient Review are secondary sources. The original Phillips and Marks (1961) article was not independently reviewed in this update. Formulation, particle dimensions, exact food intake, pathology scope and dose conversion therefore remain unverified; 5% diet is not elevated to a formal NOAEL.

Catalogue ID: Not assigned (supplementary archival source outside the frozen baseline). DOI/link: 10.1080/19440049.2018.1543957

Verification: V1 review full text reviewed; underlying PE experiment pending.

Lefaux, R. (1968). Practical Toxicology of Plastics. Historical secondary source citing Phillips, I., & Marks, G. C. (1961), Plastics in foodstuffs applications, *British Plastics*, July 1961, 385-390.

Design: Historical secondary review of polymer toxicology summarizing commercial-resin feeding studies and citing Phillips and Marks (1961) as the underlying source for the PE and PS findings.

Findings: Reports no adverse effects in rats fed Marlex 50 PE at 1.25%, 2.5% or 5% of the diet for 90 days. It also reports no abnormalities in growth, blood examinations or anatomopathology in rats fed 4% Styron 475 and Styron 666 polystyrene grades.

Risk relevance: Provides archival oral-tolerance context for real commercial PE and PS resins rather than monodisperse laboratory spheres.

Key limitations: The evidence is secondary and the original Phillips and Marks article was not independently reviewed. The PS duration, exact grade formulations, particle form, group design, food consumption and pathology scope remain unverified. The findings do not establish formal NOAELs and do not represent fragmented or weathered microplastic preparations.

Catalogue ID: Not assigned (supplementary historical source outside the frozen baseline). Verification: V1 photographed book pages reviewed; underlying Phillips and Marks article pending.

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

Design: Lifespan oral VCM carcinogenicity study in Wistar rats using Carina S 65-02 PVC powder as the dietary carrier. The main control diet contained 10% VCM-free PVC powder with residual VCM below 0.3 ppm; males and females were followed to weeks 135 and 144, respectively.

Findings: VCM-related adverse effects increased with VCM dose. The VCM-free PVC control provided long-duration exposure to a high proportion of PVC powder without a PVC-specific treatment comparison.

Risk relevance: Unusually long-duration direct oral PVC-resin exposure and valuable chronic tolerance context.

Key limitations: The study was designed to determine VCM carcinogenicity, not PVC-polymer toxicity. The additional ad-libitum 10% PVC control was autopsied but not examined histologically, feeding schedules differed, and the result is resin/formulation-specific. No formal PVC or uPVC NOAEL can be derived.

Catalogue ID: Not assigned (supplementary primary source outside the frozen baseline). Verification: V1 primary full text reviewed.

Lu, T., Liu, H., Yuan, X., et al. (2025). Chronic exposure to polyethylene terephthalate microplastics induces gut microbiota dysbiosis and disordered hepatic lipid metabolism in mice. *Ecotoxicology and Environmental Safety*, 298, 118330. <https://doi.org/10.1016/j.ecoenv.2025.118330>.

Design: Seventeen-week oral-gavage study in male ICR mice using irregular approximately 6-12 micrometre PET at 0, 5, 50 or 500 micrograms/day, with liver pathology, clinical chemistry, microbiome and metabolomic endpoints.

Findings: The authors reported no significant pathological or liver-function changes at 5 micrograms/day; 50 and 500 micrograms/day produced increasing hepatic lipid accumulation, oxidative stress, apoptosis, structural injury and functional abnormalities.

Risk relevance: Important longer-duration PET dose-response evidence and the most informative new contradiction to a simple high PET no-effect narrative.

Key limitations: The low dose increased liver coefficient; only males were tested; gavage and one particle preparation were used; no matched nonplastic-particle control isolated PET specificity; mechanistic multiplicity and the study's disputed high human-intake premise constrain direct risk inference.

Catalogue ID: Not assigned (supplementary source outside the frozen baseline). DOI/link: 10.1016/j.ecoenv.2025.118330 Verification: V1 official open full text reviewed.

Lewis, A. A., Biales, A. D., Miller, C., et al. (2026). A single oral exposure to polyethylene terephthalate microplastics causes mild metabolic and gastrointestinal disruption: Dose and sex as effect modifiers. *Drug and Chemical Toxicology*, 1-18. <https://doi.org/10.1080/01480545.2026.2643619>.

Design: Three-month-old male and female Sprague-Dawley rats received one oral gavage of cryomilled PET nurdle particles at 0, 5 or 50 mg/kg and were monitored for approximately 18 hours, followed by serum, liver and gastrointestinal analyses.

Findings: Low-dose males, but not high-dose males, showed decreases in metabolic rate, respiratory exchange ratio and insulin, together with duodenal gene-expression changes. Platelets increased dose-dependently in males; females showed reduced metabolic rate at 50 mg/kg and dose-dependent LDL increases.

Risk relevance: Acute PET hazard evidence demonstrating sex dependence and non-monotonic responses.

Key limitations: Single exposure, very short follow-up, mixed biomarker and transcriptomic endpoints, uncertain adversity, no repeated-dose design and no matched nonplastic-particle comparator. It cannot establish a repeated-dose NOAEL or ordinary-exposure risk.

Catalogue ID: Not assigned (supplementary source outside the frozen baseline). DOI/link: 10.1080/01480545.2026.2643619 Verification: V2 official EPA abstract and dataset metadata verified.

Mierzejewski, K., Kurzyńska, A., Golubska, M., et al. (2025). Oral exposure to PET microplastics induces the pancreatic immune response and oxidative stress in immature pigs. BMC Genomics, 26, 578. <https://doi.org/10.1186/s12864-025-11760-1>.

Design: Four-week study in fifteen approximately 20 kg eight-week-old piglets, with five animals per group receiving gelatin capsules containing 0, 0.1 or 1 g/day PET. Particles ranged from 7.6 to 416.9 micrometres, with a predominant diameter of 158.5 micrometres.

Findings: The low dose changed one pancreatic gene; the high dose changed 86 genes, including immune and inflammatory pathways, and was associated with oxidative-stress indicators.

Risk relevance: Large-animal oral PET mechanistic evidence across an approximate 5-50 mg/kg/day dose range.

Key limitations: Small groups, transcriptomic and oxidative-stress endpoints rather than apical toxicity, no matched nonplastic particles and potential reuse of the same animal cohort across related porcine publications. No formal NOAEL is established.

Catalogue ID: Not assigned (supplementary source outside the frozen baseline). DOI/link: 10.1186/s12864-025-11760-1 Verification: V1 official open full text reviewed.

Thomas, W. C., McGrath, L. F., Baarson, K. A., Auletta, C. S., Daly, I. W., & McConnell, R. F. (1991). Subchronic oral toxicity of cellulose acetate in rats. Food and Chemical Toxicology, 29(7), 453-458. [https://doi.org/10.1016/0278-6915\(91\)90090-T](https://doi.org/10.1016/0278-6915(91)90090-T).

Design: CD Sprague-Dawley rats received cellulose acetate by dietary admixture at 0, 500, 2,500 or 5,000 mg/kg/day for 94-96 consecutive days, with clinical observations, ophthalmology, body weight, food consumption, haematology, clinical chemistry, urinalysis, organ weights, gross pathology and histopathology.

Findings: No mortality or compound-related adverse effect was detected. The authors identified 5,000 mg/kg/day, the highest dose tested, as the subchronic NOAEL. The high dose represented approximately 7.2-10% of the diet; reduced nutritional efficiency was attributed to displacement of nutritive feed by non-nutritive bulk.

Risk relevance: Provides a well-documented direct-polymer oral NOAEL and extends the selected study-specific dose scale to 5,000 mg/kg/day.

Key limitations: Cellulose acetate is a semi-synthetic thermoplastic, the test material was not an environmentally weathered microplastic preparation, and the high dietary concentration can alter nutritional density. The value is study- and material-specific.

Catalogue ID: Not assigned (supplementary primary source outside the frozen baseline). DOI/link: 10.1016/0278-6915(91)90090-T Verification: V1 primary source package reviewed.

Haskell Laboratory. (1961). Medical Research Project No. 1080, Report No. 56-61. E. I. du Pont de Nemours and Company. Unpublished archival PTFE feeding report.

Design: Six male rats per group received diets containing 25% of several PTFE preparations for three weeks, followed by a two-week recovery period. The preparations differed in manufacture and residual dispersing-agent content.

Findings: PTFE prepared without dispersing agent did not adversely affect body-weight gain or liver weight. Preparations containing residual AHT, C8-APFC or C9-AFC dispersants produced slightly heavier or enlarged livers.

Risk relevance: Direct historical evidence that very-high-diet PTFE effects depended on formulation and residual processing aids rather than polymer name alone.

Key limitations: Very small groups, male rats only, three-week duration, limited endpoint set and no modern particle characterization. The study does not establish a comprehensive or chronic PTFE NOAEL.

Catalogue ID: Not assigned (historical supplementary primary source outside the frozen baseline). Verification: V1 archival report reviewed.

Haskell Laboratory. (1968). Three-Week Feeding Study in Rats with Polytetrafluoroethylene Resins (Teflon). Medical Research Project No. 1080, Report No. 224-68. E. I. du Pont de Nemours and Company.

Design: Eighty male ChR-CD rats, ten per group, received seven PTFE preparations at 10% of the diet for 21 days followed by a 22-day recovery period. Residual dispersing-agent content and preparation method differed across materials.

Findings: Three heated plant-prepared powders with very low residual dispersant produced no adverse body-weight, liver-weight or liver-histopathology findings. Dispersion-derived powders with higher surfactant residues produced reduced weight gain, enlarged livers or hepatocellular changes. A later EPA conversion represented 10% diet as approximately 6,000 mg/kg/day.

Risk relevance: Strong historical corroboration that relatively clean PTFE powder has very low intrinsic oral toxicity and that residual surfactants can dominate formulation toxicity.

Key limitations: Twenty-one-day duration, male rats only and liver-focused endpoints. The approximate mg/kg/day conversion was not a direct administered-dose measurement, and the referenced 90-day Report 49-60 remains unrecovered.

Catalogue ID: Not assigned (historical supplementary primary source outside the frozen baseline). Verification: V1 archival report and pathology material reviewed.

Otaka, T., Miyoshi, K., Komai, Y., & Nakayoshi, H. (1978). Safety evaluation of polyethylene terephthalate resins (I): Studies on the acute and subacute (3 months) toxicities by oral administration in rats. Journal of the Food Hygienic Society of Japan, 19(5), 431-442. <https://doi.org/10.3358/shokueishi.19.431>.

Design: Rats received chloroform extracts of titanium-catalyzed PET at 5, 30, 180 or 400 mg/kg/day and germanium-catalyzed PET at 5, 30 or 180 mg/kg/day by daily gavage for 90 days.

Findings: No treatment-related toxicity was identified under the study conditions despite some statistically significant differences judged incidental or within normal ranges.

Risk relevance: Relevant to PET extractables and deliberately severe solvent-extraction conditions, but not a direct PET-particle or high-molecular-mass PET-polymer NOAEL.

Key limitations: The administered material was the chloroform-extractable fraction rather than PET resin particles. The study cannot be used in the direct-polymer dose figure. It identifies Haskell Report 17-73 / Medical Research Project 1609 as a separate direct PET-resin feeding study that remains unrecovered.

Catalogue ID: Not assigned (supplementary primary source outside the frozen baseline). DOI/link: 10.3358/shokueishi.19.431 Verification: V1 primary full text reviewed.

Human epidemiology and clinical-association studies

Kong, F., Fang, L., Gui, Y., Lan, H., Zhao, P., et al. (2025). Polyethylene and Polyvinyl Chloride Nanoplastics in Human Follicular Fluid and Seminal Plasma: Impact on Fertilization and Sperm Quality. *ACS Nano*. <https://doi.org/10.1021/acsnano.5c00918>.

Design: Cross-sectional human fertility-clinic study measuring polyethylene and PVC nanoplastic-associated signals in follicular fluid and seminal plasma and comparing reproductive endpoints.

Findings: Polymer-associated measurements were correlated with selected fertilization or sperm-quality measures.

Risk relevance: Important hypothesis-generating human association evidence.

Key limitations: Cross-sectional design, selected clinical population, contamination and matrix-validation challenges, multiple comparisons and no temporality prevent causal inference.

Catalogue ID: STUDY-01671. DOI/link: 10.1021/acsnano.5c00918 Verification: V3.

Lee, D.-W., Jung, J., Park, S.-A., et al. (2024). Microplastic Particles in Human Blood and Their Association with Coagulation Markers. *Scientific Reports*. <https://doi.org/10.1038/s41598-024-81931-9>.

Design: Cross-sectional human blood study measuring microplastic-associated particles or signals and coagulation markers.

Findings: The study reported associations between measured particle burden and selected coagulation indicators.

Risk relevance: Generates a testable human cardiovascular or coagulation hypothesis.

Key limitations: No temporality, limited sample size, potential confounding, contamination and analytical identity uncertainty prevent attribution of coagulation changes to plastic exposure.

Catalogue ID: STUDY-00561. DOI/link: 10.1038/s41598-024-81931-9 Verification: V3.

Shen, Y., Ning, Y., Chen, F., Wang, X., Zheng, D., Sun, Y., Zhang, X., & Zhang, X. (2026). Impact of placental microplastics on birth anthropometrics: A cross-sectional study. *Ecotoxicology and Environmental Safety* 309, 119572. <https://doi.org/10.1016/j.ecoenv.2025.119572>.

Design: Cross-sectional placental study relating measured microplastics to infant birth anthropometrics.

Findings: The authors reported associations between placental measurements and selected birth-size outcomes.

Risk relevance: Provides hypothesis-generating human developmental evidence.

Key limitations: Cross-sectional sampling at delivery, uncertain exposure timing, maternal and clinical confounding, multiple comparisons and difficult placental matrix validation prevent causation.

Catalogue ID: STUDY-01652. DOI/link: 10.1016/j.ecoenv.2025.119572 Verification: V3.

Zhang, X., Li, L., Zhang, Y., Liu, B., Wang, X., & Sun, L. (2025). Placental microplastics contamination and its impact on thyroid function in newborns. *Ecotoxicology and Environmental Safety* 304, 119056. <https://doi.org/10.1016/j.ecoenv.2025.119056>.

Design: Cross-sectional placental study examining microplastic contamination and neonatal thyroid-function measures.

Findings: The study reported associations between placental polymer measurements and selected newborn thyroid indicators.

Risk relevance: Generates a human endocrine-development hypothesis requiring replication.

Key limitations: Exposure and outcome were measured cross-sectionally; confounding, multiple testing, contamination and analytical validation prevent causal or risk-threshold conclusions.

Catalogue ID: STUDY-01653. DOI/link: 10.1016/j.ecoenv.2025.119056 Verification: V3.

Ecotoxicology and organism-effect studies

Clukey, K. E., Lepczyk, C. A., Balazs, G. H., Work, T. M., & Lynch, J. M. (2017). Investigation of plastic debris ingestion by four species of sea turtles collected as bycatch in pelagic Pacific longline fisheries. *Marine Pollution Bulletin*, 120(1–2), 117–125. <https://doi.org/10.1016/j.marpolbul.2017.04.064>.

Design: Field necropsy study of plastic-debris ingestion in four sea-turtle species caught as fisheries bycatch.

Findings: Plastic debris was found in gastrointestinal tracts, with burden differing among species and individuals.

Risk relevance: Provides real-world ingestion and species-comparison evidence, primarily for larger debris.

Key limitations: Bycatch sample, gut-content endpoint, no unexposed comparison and mixed debris sizes prevent attribution of disease or microplastic-specific effects.

Catalogue ID: STUDY-01602. DOI/link: 10.1016/j.marpolbul.2017.04.064 Verification: V3.

Duncan, E. M., Broderick, A. C., Fuller, W. J., Galloway, T. S., Godfrey, M. H., Hamann, M., Limpus, C. J., Lindeque, P. K., Mayes, A. G., Omeyer, L. C. M., Santillo, D., Snape, R. T. E., & Godley, B. J. (2019). Microplastic ingestion ubiquitous in marine turtles. *Global Change Biology*, 25(2), 744–752. <https://doi.org/10.1111/gcb.14519>.

Design: Multi-region field study of microplastic ingestion in marine turtles.

Findings: Microplastic ingestion was widespread among sampled turtles and regions.

Risk relevance: Strong occurrence evidence for ingestion by marine turtles.

Key limitations: Gut contents do not establish tissue accumulation or adverse effects; sampling, contamination, size cutoff and lack of causal comparison limit risk inference.

Catalogue ID: STUDY-01346. DOI/link: 10.1111/gcb.14519 Verification: V3.

Ogonowski, M., Wenman, V., Danielsson, S., & Gorokhova, E. (2018). Hydrophobic organic contaminants are not linked to microplastic uptake in Baltic Sea herring. bioRxiv. <https://doi.org/10.1101/363127>.

Design: Baltic herring field study testing whether hydrophobic organic contaminant burdens were related to microplastic uptake.

Findings: The study found no clear link between microplastic uptake and contaminant burden in the sampled fish.

Risk relevance: Important field counterevidence to a dominant microplastic-vector pathway.

Key limitations: One species, region, chemical set and analytical sensitivity cannot exclude every vector scenario; low plastic exposure can reduce statistical power.

Catalogue ID: STUDY-00646. DOI/link: 10.1101/363127 Verification: V3.

Zandra Gerdes. 2019. A novel method for assessing microplastic effect in suspension through mixing test and reference materials. Scientific Reports.

Design: Daphnia depuration experiment testing whether microplastics materially transport PCBs relative to other exposure pathways.

Findings: The study found limited support for microplastic-mediated PCB transfer and emphasized depuration and background pathways.

Risk relevance: Strong counterevidence to a universal “Trojan horse” assumption and a useful matched-pathway comparison.

Key limitations: One organism, polymer, chemical mixture and concentration range cannot define all vector scenarios; field relevance depends on plastic abundance relative to food and natural particles.

Catalogue ID: STUDY-00545. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Soil, crop and terrestrial-system studies

Collin J. Weber. 2022. Investigating the dispersal of macro- and microplastics on agricultural fields 30 years after sewage sludge application. Scientific Reports, <https://doi.org/10.1038/s41598-022-10294-w>.

Design: Field investigation of macro- and microplastic distribution on agricultural land approximately 30 years after sewage-sludge application.

Findings: Plastic residues remained detectable and spatially redistributed decades after application.

Risk relevance: Demonstrates long-term persistence and a durable sludge-to-soil pathway.

Key limitations: One field history and analytical window do not establish a general soil concentration, fraction of all particles or adverse ecological effect.

Catalogue ID: STUDY-00554. DOI/link: 10.1038/s41598-022-10294-w Verification: V3.

Li, M., Wu, D., Wu, D., Guo, H., & Han, S. (2021). Influence of polyethylene-microplastic on environmental behaviors of metals in soil. Environmental Science and Pollution Research, 28, 28329–28336. <https://doi.org/10.1007/s11356-021-12718-y>.

Design: Controlled soil-spiking experiment testing several polyethylene-microplastic loadings and measuring zinc and lead adsorption/desorption behavior.

Findings: Effects on metal behavior were concentrated at the highest plastic loading; no significant difference was reported at the lowest tested loading, which the authors considered closer to typical environmental levels.

Risk relevance: Relevant to chemical-partitioning mechanisms and provides useful lower-concentration null evidence. It is not direct evidence of polymer-particle toxicity.

Key limitations: The highest treatment was extremely large; the system used simplified polyethylene and selected soil/metals, lacked a matched natural-particle comparator and measured partitioning rather than organism dose or effect.

Catalogue ID: STUDY-00062. DOI/link: 10.1007/s11356-021-12718-y Verification: V3.

Lin, D., Yang, G., Dou, P., Qian, S., Zhao, L., Yang, Y., & Fanin, N. (2020). Microplastics negatively affect soil fauna but stimulate microbial activity: Insights from a field-based microplastic addition experiment. Proceedings of the Royal Society B, 287, 20201268. <https://doi.org/10.1098/rspb.2020.1268>.

Design: Field-based soil experiment examining soil fauna and microbial activity after microplastic addition.

Findings: The study reported reduced selected soil-fauna measures but stimulated microbial activity, demonstrating endpoint-dependent rather than uniformly adverse responses.

Risk relevance: Direct field-based ecological evidence under tested conditions.

Key limitations: Polymer, concentration, soil, duration and endpoints are specific; a particle-free control does not establish that effects are unique to plastic.

Catalogue ID: STUDY-01419. DOI/link: 10.1098/rspb.2020.1268 Verification: V3.

Schöpfer, L., et al. (2022). Microplastics persist in an arable soil but do not affect soil microbial biomass, enzyme activities, or crop yield. Journal of Plant Nutrition and Soil Science, 185, 836–849. <https://doi.org/10.1002/jpln.202200062>.

Design: Long-term arable-soil field experiment measuring persistence of introduced microplastics and responses of soil microbial biomass, enzyme activity and crop yield.

Findings: The particles persisted, but the tested treatments did not produce detectable adverse effects on the measured microbial endpoints or crop yield under the field conditions and duration studied.

Risk relevance: Directly relevant field-scale agricultural evidence that persistence does not automatically imply impaired soil function or crop production.

Key limitations: Power and generalizability are bounded by the tested polymer mixture, concentration, soil, crop rotation, duration and endpoint set.

Catalogue ID: STUDY-00012. DOI/link: 10.1002/jpln.202200062 Verification: V3.

Stuart J. F. F. Ramage, et al. 2025. Microplastics in agricultural soils following sewage sludge applications: Evidence from a 25-year study. *Chemosphere* 376:144277.

Design: Long-term agricultural-field study examining microplastics in soils following repeated sewage-sludge applications over approximately 25 years.

Findings: Repeated sludge application was associated with persistent and higher soil microplastic burdens, demonstrating a durable terrestrial source pathway.

Risk relevance: Establishes long-term source, persistence and redistribution evidence. It does not show that the detected plastic fraction caused adverse soil or crop effects.

Key limitations: Site history, sludge composition, soil depth, particle recovery and analytical cutoff limit transfer to other systems; occurrence is not a measure of ecological harm.

Catalogue ID: STUDY-00102. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Zhu, R., Zhang, Z., Zhang, N., Zhong, H., Zhou, F., Zhang, X., Liu, C., Huang, Y., Yuan, Y., Wang, Y., Li, C., Shi, H., Rillig, M. C., Dang, F., Ren, H., Zhang, Y., & Xing, B. (2025). A global estimate of multiecosystem photosynthesis losses under microplastic pollution. *Proceedings of the National Academy of Sciences*, 122(11), e2423957122. <https://doi.org/10.1073/pnas.2423957122>.

Design: Global modeling synthesis estimating crop-production losses attributed to microplastic pollution through multiple indirect assumptions.

Findings: The paper projected large global crop losses, but did not directly demonstrate microplastic-caused yield effects at global scale.

Risk relevance: Functions as strong contrary modeling evidence that must be stress-tested rather than accepted as measured crop harm.

Key limitations: The causal chain combines heterogeneous laboratory studies and modeled climate/yield relationships, does not adequately account for dose realism, background particles, carbon-dioxide fertilization or documented benefits of plastic mulch, and cannot support a direct global attribution.

Catalogue ID: STUDY-01024. DOI/link: 10.1073/pnas.2423957122 Verification: V3.

Wastewater, treatment and sludge studies

Akarsu, C., et al. (2021). Removal of microplastics from wastewater through electrocoagulation–electroflotation and membrane filtration processes. *Water Science and Technology*, 84(7), 1648–1662. <https://doi.org/10.2166/wst.2021.356>.

Design: Controlled wastewater-treatment comparison of electrocoagulation-electroflotation and membrane filtration for microplastic removal.

Findings: Both processes removed model particles under tested conditions, with performance depending on operating conditions and particle properties.

Risk relevance: Supports engineering treatment options.

Key limitations: Model water and particles may overstate full-scale performance; energy, fouling, recovery, residual management and transfer rather than destruction remain relevant.

Catalogue ID: STUDY-00737. DOI/link: 10.2166/wst.2021.356 Verification: V3.

Edo, C., González-Pleiter, M., Leganés, F., Fernández-Piñas, F. & Rosal, R. 2020. Fate of Microplastics in Wastewater Treatment Plants and Their Environmental Dispersion with Effluent and Sludge. *Environmental Pollution* 259:113837. <https://doi.org/10.1016/j.envpol.2019.113837>.

Design: Wastewater-treatment field study tracing microplastics through treatment and environmental dispersion with effluent and sludge.

Findings: Most counted material was retained in solid residuals while a smaller fraction left with effluent, demonstrating pathway transfer rather than complete elimination.

Risk relevance: Supports treatment mass-flow and residual-stream management.

Key limitations: Removal estimates depend on influent/effluent matching, recovery, size window and polymer confirmation; transfer to sludge is not destruction.

Catalogue ID: STUDY-01418. DOI/link: 10.1016/j.envpol.2019.113837 Verification: V3.

Michielssen, M. R., Michielssen, E. R., Ni, J., & Duhaime, M. B. (2016). Fate of microplastics and other small anthropogenic litter (SAL) in wastewater treatment plants depends on unit processes employed. *Environmental Science: Water Research & Technology*, 2, 1064–1073. <https://doi.org/10.1039/C6EW00207B>.

Design: Wastewater-treatment study examining fate of microplastics and other small anthropogenic litter across treatment stages.

Findings: Retention depended on particle properties and treatment process, with substantial transfer to sludge.

Risk relevance: Provides process-specific removal and residual-stream evidence.

Key limitations: Counts depend on analytical cutoff, recovery and matching of samples; apparent water removal does not equal destruction or total mass elimination.

Catalogue ID: STUDY-01453. DOI/link: 10.1039/C6EW00207B Verification: V3.

Niina Kärkkäinen. 2025. Microplastic Pollution via Wastewater Effluent and Sewage Sludge: Special Focus on Microplastic Fibres in Compost. *Water, Air, & Soil Pollution*.

Design: Field/source-characterization study tracing microplastic fibres through wastewater effluent, sewage sludge and compost-related pathways.

Findings: Fibres were reported in wastewater-derived streams and compost, supporting sewage sludge and organic amendments as routes by which particles can reach soil.

Risk relevance: Provides occurrence and transfer evidence for residual streams. Capture or transfer to sludge is not particle destruction and does not by itself establish toxicity.

Key limitations: Results are site-, matrix-, size-cutoff- and method-dependent; fibre identity, recovery, treatment representativeness and the much larger organic and mineral solids background constrain interpretation.

Catalogue ID: STUDY-00054. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Talvitie, J., Mikola, A., Koistinen, A., & Setälä, O. (2017). Solutions to microplastic pollution – Removal of microplastics from wastewater effluent with advanced wastewater treatment technologies. *Water Research*, 123, 401–407. <https://doi.org/10.1016/j.watres.2017.07.005>.

Design: Full-scale or pilot wastewater-treatment study comparing advanced polishing technologies for microplastic removal from effluent.

Findings: Advanced treatment substantially reduced counted particles in the treated water, with performance differing among processes.

Risk relevance: Supports treatment and source-control options.

Key limitations: Count removal depends on matched size windows and recovery; captured particles are transferred to residual streams rather than necessarily destroyed.

Catalogue ID: STUDY-00416. DOI/link: 10.1016/j.watres.2017.07.005 Verification: V3.

Tanaka, S., & Shimatani, S. (2023). Microplastics Derived from Chemical Fibers. *Material Cycles and Waste Management Research*, 34(3), 178–182. <https://doi.org/10.3985/mcwmr.34.178>.

Design: Technical/source review of microplastics derived from chemical fibres, including generation and waste-management pathways.

Findings: The source describes fibre formation and release pathways and identifies textiles and fibre products as controllable sources.

Risk relevance: Useful for source characterization and mitigation planning.

Key limitations: A technical synthesis is not an independent exposure or toxicity dataset; release estimates and terminology require source-level verification and separation of natural, regenerated and synthetic fibres.

Catalogue ID: STUDY-01176. DOI/link: 10.3985/mcwmr.34.178 Verification: V3.

Fragmentation, weathering and environmental-fate studies

Bagheri, A. R., Laforsch, C., Greiner, A., & Agarwal, S. (2017). Fate of so-called biodegradable polymers in seawater and freshwater. *Global Challenges*, 1, 1700048. <https://doi.org/10.1002/gch2.201700048>.

Design: Comparative laboratory study of polymers marketed as biodegradable in seawater and freshwater.

Findings: Environmental fate differed strongly by polymer and medium, and “biodegradable” did not mean rapid disappearance in both waters.

Risk relevance: Important substitution and persistence context.

Key limitations: Laboratory conditions, formulation and duration do not map directly to all natural environments; surface change or mass loss is not necessarily complete mineralization.

Catalogue ID: STUDY-01446. DOI/link: 10.1002/gch2.201700048 Verification: V3.

Chowreddy, R. R., Fredriksen, S. B., Anwar, H., Mylvaganam, B., Iveland, A., & Kamfjord, T. (2026). Degradation behaviour of different polyethylene and polypropylene materials under long-term accelerated weathering conditions. *Journal of Polymer Research*, 33, 45. <https://doi.org/10.1007/s10965-026-04777-x>.

Design: Controlled long-term accelerated-weathering study of commercial virgin and recycled polyethylene and polypropylene materials with differing formulations and stabilizer packages.

Findings: Weathering behavior differed by polymer grade, recycled content and stabilization package, showing that degradation cannot be generalized from the polymer name alone.

Risk relevance: Relevant to product durability and secondary-particle source formation, not direct evidence of biological harm.

Key limitations: Accelerated chamber exposure does not map uniquely to outdoor service time; spectrum, temperature, humidity, specimen geometry and incomplete fragmentation mass balance constrain field extrapolation.

Catalogue ID: STUDY-00048. DOI/link: 10.1007/s10965-026-04777-x Verification: V3.

Napper, I. E., & Thompson, R. C. (2019). Environmental deterioration of biodegradable, oxo-biodegradable, compostable and conventional plastic carrier bags in the sea, soil and open-air over a 3-year period. *Environmental Science & Technology*, 53(9), 4775–4783. <https://doi.org/10.1021/acs.est.8b06984>.

Design: Long-duration field exposure of biodegradable, oxo-biodegradable, compostable and conventional carrier bags in sea, soil and open-air environments.

Findings: Several bags remained functional or persisted after years, and degradation depended strongly on material and environment.

Risk relevance: Shows that biodegradability labels do not guarantee rapid disappearance across receiving environments.

Key limitations: Specific commercial products, item geometry and sites cannot define all formulations; visual deterioration is not complete mineralization.

Catalogue ID: STUDY-01377. DOI/link: 10.1021/acs.est.8b06984 Verification: V3.

Song, Y. K., Hong, S. H., Jang, M., Han, G. M., Jung, S. W., & Shim, W. J. (2020). Rapid Production of Micro- and Nanoplastics by Fragmentation of Expanded Polystyrene Exposed to Sunlight. *Environmental Science & Technology*, 54(18), 11191–11200. <https://doi.org/10.1021/acs.est.0c02288>.

Design: Controlled sunlight-weathering experiment examining fragmentation of expanded polystyrene into micro- and nanoscale material.

Findings: Sunlight exposure rapidly increased the number of smaller fragments under the experimental conditions.

Risk relevance: Demonstrates a fragmentation mechanism and secondary-particle source.

Key limitations: Fragment number increase does not create additional polymer mass or demonstrate toxicity; accelerated exposure, product formulation and geometry constrain environmental rate extrapolation.

Catalogue ID: STUDY-01249. DOI/link: 10.1021/acs.est.0c02288 Verification: V3.

Weinstein, J. E., Crocker, B. K., & Gray, A. D. (2016). From macroplastic to microplastic: Degradation of high-density polyethylene, polypropylene, and polystyrene in a salt marsh habitat. Environmental Toxicology and Chemistry, 35(7), 1632–1640. <https://doi.org/10.1002/etc.3432>.

Design: Field-weathering study of HDPE, polypropylene and polystyrene items deployed in a salt-marsh habitat and examined for degradation and fragmentation.

Findings: The tested materials exhibited polymer- and condition-dependent surface change, embrittlement and fragmentation, supporting environmental generation of secondary microplastics from larger items.

Risk relevance: Relevant to environmental persistence and source formation. Fragmentation changes particle number and surface area but does not create additional polymer mass or demonstrate biological harm.

Key limitations: Weathering rates depend on formulation, geometry, ultraviolet exposure, temperature, oxygen, abrasion and biofouling. Results from one habitat and item geometry should not be generalized to all products or environments.

Catalogue ID: STUDY-00005. DOI/link: 10.1002/etc.3432 Verification: V3.

Natural-particle and matched-comparator studies

Martin Ogonowski (2018). What we know and what we think we know about microplastic effects - A critical perspective. Current Opinion in Environmental Science & Health, 1 (2018) 41-46. doi:10.1016/j.coesh.2017.09.001.

Design: Critical review of microplastic-effect evidence with emphasis on experimental design, dose realism and the need for appropriate particle comparators.

Findings: The review concluded that many reported effects cannot be assigned specifically to plastic because experiments often compare plastic particles only with particle-free controls.

Risk relevance: Provides the conceptual basis for separating generic particle responses from plastic-specific effects.

Key limitations: A critical review does not generate an independent dose-response dataset; conclusions depend on the quality and comparability of the underlying studies.

Catalogue ID: STUDY-00106. DOI/link: 10.1016/j.coesh.2017.09.001 Verification: V3.

Ogonowski, M., Schür, C., Jarsén, Å., & Gorokhova, E. (2016). The Effects of Natural and Anthropogenic Microparticles on Individual Fitness in *Daphnia magna*. PLOS ONE, 11(5), e0155063. <https://doi.org/10.1371/journal.pone.0155063>.

Design: *Daphnia* experiment comparing natural and anthropogenic microparticles at comparable concentrations and evaluating fitness endpoints.

Findings: Natural and plastic particles both affected selected endpoints, with responses depending on particle properties and food conditions.

Risk relevance: Core comparative evidence that many responses are generic particle effects rather than uniquely plastic.

Key limitations: Comparator matching was not perfect and one species cannot represent all ecosystems, but the design is more informative than plastic-versus-clean-water tests.

Catalogue ID: STUDY-00717. DOI/link: 10.1371/journal.pone.0155063 Verification: V3.

Ogonowski, M., Wagner, M., Rogell, B., Haave, M., & Lusher, A. (2023). Microplastics could be marginally more hazardous than natural suspended solids: A meta-analysis. Ecotoxicology and Environmental Safety, 264, 115406. <https://doi.org/10.1016/j.ecoenv.2023.115406>.

Design: Meta-analysis comparing experimental hazards of microplastics with natural suspended solids.

Findings: The pooled analysis suggested microplastics may be marginally more hazardous on average, but uncertainty and heterogeneity were substantial and direct comparator evidence remained limited.

Risk relevance: One of the strongest contrary syntheses on particle specificity; it addresses experimental hazard, not ordinary-exposure risk.

Key limitations: Studies differed in organisms, endpoints, size, shape, concentration and comparator matching; publication and selection biases and high experimental doses constrain generalization.

Catalogue ID: STUDY-00131. DOI/link: 10.1016/j.ecoenv.2023.115406 Verification: V3.

Suaria, G., Achtypi, A., Perold, V., Lee, J.R., Pierucci, A., Bornman, T.G., Aliani, S., & Ryan, P.G. (2020). Microfibers in oceanic surface waters: a global characterization. Science Advances, 6, eaay8493. <https://doi.org/10.1126/sciadv.aay8493>.

Design: Global survey characterizing microfibres in oceanic surface waters using microscopy and spectroscopic identification.

Findings: Fibres were widespread, but the great majority of chemically characterized fibres were natural or cellulosic rather than synthetic plastic.

Risk relevance: Critical context for interpreting “microfibre” counts and plastic-specific ocean exposure.

Key limitations: Surface sampling, geographic coverage, size cutoff and fibre recovery constrain global mass estimates; natural fibres and synthetic polymers have different persistence and hazard profiles.

Catalogue ID: STUDY-00995. DOI/link: 10.1126/sciadv.aay8493 Verification: V3.

W. Courtene-Jones, F. De Falco, F. Burgevin, et al. 2024. Are Biobased Microfibers Less Harmful than Conventional Plastic Microfibers: Evidence from Earthworms. Environmental Science & Technology.

Design: Earthworm comparison of biobased and conventional plastic microfibres using survival, growth or biological endpoints.

Findings: The study tested whether biobased fibres were less harmful and found material-dependent rather than automatically lower effects.

Risk relevance: Direct substitution and matched-fibre evidence.

Key limitations: One soil organism, fibre set, dose and laboratory matrix do not define field ecological superiority; chemistry and physical fibre effects require separation.

Catalogue ID: STUDY-00463. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Yardy, L., & Callaghan, A. (2021). Microplastic and Organic Fibres in Feeding, Growth and Mortality of Gammarus pulex. *Environments*, 8(8), 74. <https://doi.org/10.3390/environments8080074>.

Design: Gammarus pulex feeding experiment comparing microplastic and organic fibres and evaluating ingestion, growth and mortality.

Findings: Responses depended on fibre type and exposure conditions, providing a direct plastic-versus-organic comparison.

Risk relevance: Useful matched-comparator evidence for fibre effects.

Key limitations: Fibre dimensions, chemistry, concentration, food dilution and one-species laboratory conditions limit generalization.

Catalogue ID: STUDY-01376. DOI/link: 10.3390/environments8080074 Verification: V3.

Additives, leachates and sorbed-chemical studies

Bejgarn, S., MacLeod, M., Bogdal, C., & Breitholtz, M. (2015). Toxicity of leachate from weathering plastics: An exploratory screening study with Nitocra spinipes. *Chemosphere*, 132, 114–119.

<https://doi.org/10.1016/j.chemosphere.2015.03.010>.

Design: Exploratory aquatic screening study testing leachates from weathered plastic products in Nitocra spinipes.

Findings: Leachate toxicity varied substantially among products and weathering conditions, demonstrating formulation-specific chemical hazard.

Risk relevance: Supports product-leachate and constituent assessment, not a generic intact-particle hazard.

Key limitations: Extraction severity, product composition and laboratory concentration prevent generalization to ordinary environmental exposure.

Catalogue ID: STUDY-01370. DOI/link: 10.1016/j.chemosphere.2015.03.010 Verification: V3.

Bethanie Carney Almroth. 2023. Single-use take-away cups of paper are as toxic to aquatic midge larvae as plastic cups. *Environmental Pollution*, 330 (2023) 121836.

Design: Comparative aquatic toxicity study of leachates from single-use paper and plastic takeaway cups using midge larvae.

Findings: Leachates from both paper and plastic cups produced adverse responses under the test conditions, showing that nonplastic alternatives can also release biologically active constituents.

Risk relevance: Relevant to product and substitution assessment; it cautions against attributing every effect to intact plastic particles.

Key limitations: Leachate testing reflects specific product formulations and extraction conditions, not ordinary intact-particle exposure or comparative lifecycle risk.

Catalogue ID: STUDY-00192. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Hirai, H., Takada, H., Ogata, Y., Yamashita, R., Mizukawa, K., Saha, M., Kwan, C., Moore, C., Gray, H., Laursen, D., Zettler, E. R., Farrington, J. W., Reddy, C. M., Peacock, E. E., & Ward, M. W. (2011). Organic micropollutants in marine plastics debris from the open ocean and remote and urban beaches. *Marine Pollution Bulletin*, 62(8), 1683–1692.

<https://doi.org/10.1016/j.marpolbul.2011.06.004>.

Design: Global field survey of persistent organic pollutants associated with plastic pellets collected from beaches.

Findings: Pellets accumulated diverse organic contaminants, with concentrations varying geographically and by compound.

Risk relevance: Establishes sorption and spatial chemical-monitoring utility.

Key limitations: Concentrations on pellets do not establish that plastics dominate organism exposure; bioavailability, desorption and competing sediment and food pathways must be quantified.

Catalogue ID: STUDY-01584. DOI/link: 10.1016/j.marpolbul.2011.06.004 Verification: V3.

Lee, H., Byun, D.-E., Kim, J. M., & Kwon, J.-H. (2018). Desorption of hydrophobic organic chemicals from fragment-type microplastics. *Ocean Science Journal*, 53, 631–639. <https://doi.org/10.1007/s12601-018-0051-x>.

Design: Laboratory desorption study of hydrophobic organic chemicals from fragment-type microplastics.

Findings: Desorption varied by chemical, polymer, aging and medium under the test conditions.

Risk relevance: Informs chemical release and vector mechanisms.

Key limitations: Batch desorption is not organism dose or demonstrated risk; real exposure must include water, food, sediment and natural-particle pathways.

Catalogue ID: STUDY-01474. DOI/link: 10.1007/s12601-018-0051-x Verification: V3.

Rochman, C. M., Hoh, E., Kurobe, T., & Teh, S. J. (2014). Long-Term Sorption of Metals Is Similar among Plastic Types: Implications for Plastic Debris in Aquatic Environments. *PLOS ONE*, 9(1), e85433.

<https://doi.org/10.1371/journal.pone.0085433>.

Design: Long-term field/laboratory sorption study comparing metals associated with multiple plastic types.

Findings: Metal sorption was broadly similar among tested plastic types and depended on environmental exposure and surface condition.

Risk relevance: Supports chemical-partitioning context and argues against treating polymer identity alone as a unique vector mechanism.

Key limitations: Sorption concentration is not bioavailable organism dose; natural sediment, organic matter, food and desorption pathways determine real exposure.

Catalogue ID: STUDY-00715. DOI/link: 10.1371/journal.pone.0085433 Verification: V3.

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

Design: Field/organism study measuring chemical pollutants sorbed to ingested microbeads from personal-care products.

Findings: Ingested microbeads carried sorbed chemicals under the sampled or experimental conditions.

Risk relevance: Supports the plausibility of a particle-associated chemical pathway.

Key limitations: Chemical presence on beads does not show increased total organism dose or harm relative to dissolved, dietary, sediment and natural-particle sources.

Catalogue ID: STUDY-01616. DOI/link: 10.1021/acs.est.5b06280 Verification: V3.

Modeling, exposure-estimation and measured-versus-modeled studies

Borrelle, S. B., Ringma, J., Law, K. L., et al. (2020). Predicted growth in plastic waste exceeds efforts to mitigate plastic pollution. Science, 369, 1515–1518. <https://doi.org/10.1126/science.aba3656>.

Design: Global scenario model projecting plastic-waste generation, leakage and the effect of mitigation strategies.

Findings: Projected growth in waste and leakage exceeded the reductions achieved by modeled current commitments, while combined interventions reduced but did not eliminate leakage.

Risk relevance: Provides systems-level source and policy scenario context.

Key limitations: Results depend on production, waste-management, behavior, geography and policy assumptions and are not measurements of environmental concentration or health risk.

Catalogue ID: STUDY-00656. DOI/link: 10.1126/science.aba3656 Verification: V3.

Eriksen, M., Lebreton, L. C. M., Carson, H. S., et al. (2014). 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. <https://doi.org/10.1371/journal.pone.0111913>.

Design: Comparative laboratory study of contaminant sorption or biological transfer involving microplastics and other environmental particles.

Findings: The study showed that contaminant interactions depend on particle material and environmental context rather than being unique to plastic.

Risk relevance: Provides particle-comparator evidence for evaluating chemical-vector claims.

Key limitations: Simplified batch or organism conditions, selected chemicals and imperfect matching constrain field risk inference.

Catalogue ID: STUDY-00716. DOI/link: 10.1371/journal.pone.0111913 Verification: V3.

Lebreton, L. C. M., van der Zwet, J., Damsteeg, J.-W., Slat, B., Andrady, A., & Reisser, J. (2019). A Global Mass Budget for Positively Buoyant Macroplastic Debris in the Ocean. Scientific Reports. <https://doi.org/10.1038/s41598-019-49413-5>.

Design: Global mass-budget model for positively buoyant macroplastic debris in the ocean.

Findings: The model reconciled input, fragmentation, beaching, transport and observed floating inventories under explicit assumptions.

Risk relevance: Provides quantitative fate and inventory context for macroplastic sources of secondary particles.

Key limitations: Model structure, input estimates, size classes, degradation and removal assumptions determine the result; it is not a direct total-ocean measurement.

Catalogue ID: STUDY-00546. DOI/link: 10.1038/s41598-019-49413-5 Verification: V3.

Meijer, L.J.J., van Emmerik, T., van der Ent, R., Schmidt, C., & Lebreton, L. (2021). More than 1000 rivers account for 80% of global riverine plastic emissions into the ocean. Science Advances, 7, eaaz5803. <https://doi.org/10.1126/sciadv.aaz5803>.

Design: Global river-emission model ranking rivers by estimated plastic transport to the ocean.

Findings: The model estimated that more than 1,000 rivers collectively account for most modeled riverine plastic emissions, rather than a small handful alone.

Risk relevance: Useful for source prioritization and testing intervention scenarios.

Key limitations: Predictions depend on waste, hydrology, population and calibration data and concern modeled macroplastic emissions rather than measured microplastic exposure or harm.

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Catalogue ID: STUDY-00948. DOI/link: 10.1126/sciadv.aaz5803 Verification: V3.

Weiss, L., Ludwig, W., Heussner, S., et al. (2021). The missing ocean plastic sink: Gone with the rivers. Science, 373, 107–111. <https://doi.org/10.1126/science.abe0290>.

Design: River-to-ocean mass-balance analysis examining the apparent mismatch between modeled river inputs and observed ocean plastic stocks.

Findings: The authors argued that substantial material may be retained, transformed or removed in river, coastal and nearshore systems before reaching open ocean inventories.

Risk relevance: Provides an alternative fate interpretation and challenges simple input-to-stock assumptions.

Key limitations: Mass balance and residence-time assumptions are uncertain, and modeled missing sinks require independent field validation.

Catalogue ID: STUDY-00657. DOI/link: 10.1126/science.abe0290 Verification: V3.

Established nonplastic dust hazards, generic particle overload and risk-communication context

Borm, P. J. A., & Driscoll, K. E. (2019). The hazards and risks of inhaled poorly soluble particles - where do we stand after 30 years of research? Particle and Fibre Toxicology, 16, 11. <https://doi.org/10.1186/s12989-019-0294-4>.

Design: Peer-reviewed expert review/commentary synthesizing decades of inhalation research on poorly soluble low-toxicity particles, lung particle overload, species differences and human relevance.

Findings: Very high retained lung burdens can impair macrophage-mediated clearance and drive persistent inflammation. Responses under overload may reflect generic particle loading rather than material-specific toxicity, and extrapolation of rat overload tumors to ordinary human exposure is disputed.

Risk relevance: Supports the report's conclusion that biological effects at extreme particle loading are not unique to plastic and cannot be projected directly to ordinary exposure.

Key limitations: Focused on inhalation and a defined class of poorly soluble particles; it is not a direct microplastic exposure study and does not imply that all particles have equal hazard.

Catalogue ID: Not assigned (supplementary comparative-particle context outside frozen baseline). DOI/link: 10.1186/s12989-019-0294-4
Verification: V1.

Steenland, K., 't Mannetje, A., Boffetta, P., et al. (2001). Pooled exposure-response analyses and risk assessment for lung cancer in 10 cohorts of silica-exposed workers: an IARC multicentre study. Cancer Causes & Control, 12(9), 773-784. <https://doi.org/10.1023/A:1012214102061>.

Design: Pooled exposure-response analysis of ten occupational cohorts comprising silica-exposed workers.

Findings: The pooled analysis identified an exposure-response relationship between cumulative respirable crystalline silica exposure and lung-cancer risk.

Risk relevance: Provides direct human evidence that respirable quartz is a specific high-hazard dust constituent and therefore a materially different comparator from ordinary low-toxicity plastic dust.

Key limitations: Occupational exposure levels, co-exposures and silica-specific pathology limit generalization to household dust or other materials; the study does not quantify ordinary environmental silica risk.

Catalogue ID: Not assigned (supplementary established-hazard context outside frozen baseline). DOI/link: 10.1023/A:1012214102061
Verification: V2.

Demers, P. A., Kogevinas, M., Boffetta, P., et al. (1995). Wood dust and sino-nasal cancer: pooled reanalysis of twelve case-control studies. American Journal of Industrial Medicine, 28(2), 151-166. <https://doi.org/10.1002/ajim.4700280202>.

Design: Pooled reanalysis of twelve case-control studies evaluating occupational wood-dust exposure and sino-nasal cancer.

Findings: The analysis provided strong support for an association between occupational wood-dust exposure and sino-nasal cancer, particularly adenocarcinoma.

Risk relevance: Demonstrates that a common nonplastic dust can have a well-established human carcinogenic hazard, providing a clear contrast with the absence of comparable evidence for ordinary plastic dust.

Key limitations: The evidence concerns occupational exposure and a specific cancer site; it should not be interpreted as evidence that all household wood particles are materially hazardous at ordinary concentrations.

Catalogue ID: Not assigned (supplementary established-hazard context outside frozen baseline). DOI/link: 10.1002/ajim.4700280202
Verification: V2.

Tchounwou, P. B., Yedjou, C. G., Patlolla, A. K., & Sutton, D. J. (2012). Heavy metal toxicity and the environment. EXS, 101, 133-164. https://doi.org/10.1007/978-3-7643-8340-4_6.

Design: Peer-reviewed review of environmental occurrence, exposure pathways and mechanisms of toxicity for arsenic, cadmium, chromium, lead, mercury and related toxic elements.

Findings: The review identifies these elements as major public-health toxicants capable of multi-organ injury, with toxicity dependent on chemical species, route and dose; several are established or probable human carcinogens.

Risk relevance: Supports substance-specific assessment of toxic metal- and metalloid-containing dust rather than treating every particle as equivalent. It provides an established-hazard contrast to ordinary common-polymer particles.

Key limitations: The term heavy metal is broad and chemically imprecise; hazard depends on the individual element, oxidation state, compound, solubility, route and dose. The review is not a direct comparison with microplastics.

Catalogue ID: Not assigned (supplementary established-hazard context outside frozen baseline). DOI/link: 10.1007/978-3-7643-8340-4_6
Verification: V2.

Colloca, L., & Barsky, A. J. (2020). Placebo and nocebo effects. *New England Journal of Medicine*, 382, 554-561. <https://doi.org/10.1056/NEJMr1907805>.

Design: Peer-reviewed clinical review of placebo and nocebo effects, including the role of negative expectations, learning, communication and symptom attribution.

Findings: Negative expectations can produce or amplify adverse symptoms and influence health behavior even without a corresponding toxic or pharmacological mechanism.

Risk relevance: Supports proportionate risk communication: repeatedly framing a low-evidence exposure as a hidden threat can create real symptoms, distress and health-service burden.

Key limitations: The review concerns clinical and experimental nocebo phenomena rather than microplastics specifically. It does not imply that all reported symptoms are psychogenic or that genuine hazards should be minimized.

Catalogue ID: Not assigned (supplementary risk-communication context outside frozen baseline). DOI/link: 10.1056/NEJMr1907805
Verification: V2.

Technical, review and context records

American Chemistry Council and invited experts. 2022. Summary Report: May 25 & 26, 2022 Microplastic Reference Materials Invited Expert Workshop. Unresolved issue: The exact public authoritative copy, version date, named report authorship and stable provenance for the locally retained ACC microplastic-reference-materials workshop summary remain unresolved.

Design: Invited-expert workshop summary on development and use of microplastic reference materials.

Findings: The workshop documented priorities for representative particles, characterization, homogeneity, stability and interlaboratory use.

Risk relevance: Useful method-development and quality-assurance context.

Key limitations: Workshop consensus is not an independently validated reference-material standard; exact authorship, version and public provenance remain unresolved.

Catalogue ID: STUDY-01199. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Author not established from catalogue. 2023. Jambeck Was Wrong: Reanalysis of Updated Data on Plastic and Ocean Inputs. Unpublished local analytical report. Unresolved issue: The local analytical report remains unpublished and lacks a verified author, version date, underlying data/equations, uncertainty analysis, and peer-review or institutional provenance.

Design: Unpublished local reanalysis of modeled plastic inputs to the ocean.

Findings: The document challenges earlier global input estimates using updated data and alternative assumptions.

Risk relevance: Potential sensitivity analysis for source estimates.

Key limitations: Author, version, equations, data package, uncertainty analysis, peer review and institutional provenance are unverified, so it cannot control a conclusion.

Catalogue ID: STUDY-01172. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Ball Corporation. [year not established]. Global Plastics and Recycling: Industry One-Pager. Local PDF. Unresolved issue: The exact authoritative Ball Corporation one-page source corresponding to the local record, its publication date, named authorship and stable version provenance remain unresolved.

Design: Industry one-page summary of global plastics and recycling statistics.

Findings: The document presents high-level production, waste or recycling claims for communication purposes.

Risk relevance: Provides industry and system context only.

Key limitations: Exact source, date, authorship, definitions and underlying datasets are unresolved; a one-pager is not primary scientific evidence.

Catalogue ID: STUDY-01427. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Chesapeake Bay Program Scientific and Technical Advisory Committee. [date not established]. Microplastics in the Chesapeake Bay: Scientific and Technical Assessment. Formal regional report.

Design: Formal Chesapeake Bay scientific and technical assessment of microplastics occurrence, monitoring and research needs.

Findings: The assessment synthesized regional sources, occurrence data, analytical limitations and management-relevant knowledge gaps.

Risk relevance: Provides authoritative regional context and a map to primary evidence.

Key limitations: It is a technical synthesis rather than an independent experiment; conclusions reflect its evidence cutoff and heterogeneous underlying methods.

Catalogue ID: STUDY-01082. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Chesapeake Bay Program. [year not established]. Microplastics in the Chesapeake Bay. Explanatory and monitoring one-pager; local PDF. Unresolved issue: The Chesapeake Bay one-page explanatory record lacks a verified author/date/stable source, and its exact derivative relationship to the formal STAC report represented by STUDY-01082 remains unresolved.

Design: Regional explanatory and monitoring one-pager on microplastics in Chesapeake Bay.

Findings: The document summarizes regional occurrence, sources and monitoring concerns.

Risk relevance: Useful public-facing regional context.

Key limitations: Author, date, stable source and derivative relationship to the formal STAC assessment remain unresolved; numerical claims require primary-source verification.

Catalogue ID: STUDY-01373. DOI/link: No verified DOI or stable link recorded. Verification: V3.

DeArmitt, C. / Phantom Plastics. 2026. Microplastics and Tap Water: Plastic Pipes and Alternative Pipe Materials. Technical evidence review/local PDFs. Unresolved issue: Two local 2026 Phantom Plastics technical-review PDFs remain without a verified public/final version, stable source URL, or source-level version lineage.

Design: Local technical evidence review of microplastics in tap water and plastic versus alternative pipe materials.

Findings: The review compares occurrence, source attribution and material alternatives across drinking-water infrastructure studies.

Risk relevance: Provides source-mapping and comparative-material context.

Key limitations: No verified public final version or version lineage is established; as a local synthesis it cannot replace the cited primary studies.

Catalogue ID: STUDY-01134. DOI/link: No verified DOI or stable link recorded. Verification: V3.

DeArmitt, C. 2024. Microplastic Ingestion & Accumulation. Independent mass-balance analysis; local PDF. Unresolved issue: The local mass-balance analysis remains without a verified public version, stable version date, source-level data package, or exact equivalence to any related public material.

Design: Local independent mass-balance analysis of human microplastic ingestion and modeled accumulation.

Findings: The analysis places modeled intake, elimination and retained burden into a quantitative mass framework.

Risk relevance: Potential context for exposure-scale and retention claims.

Key limitations: No verified public version, stable date, reproducible source-level data package or equivalence to published material is established; it must not independently support conclusions.

Catalogue ID: STUDY-01193. DOI/link: No verified DOI or stable link recorded. Verification: V3.

DeArmitt, C. 2024. Ridgeway Rockbags — Scientific Assessment. Product-specific technical assessment; local final and draft PDFs. Unresolved issue: Multiple local draft/final Ridgeway Rockbags assessment PDFs remain; the exact commissioned final version, release date and stable public provenance have not been independently established.

Design: Product-specific technical assessment of Ridgeway Rockbags and related environmental claims.

Findings: The assessment synthesizes material, degradation, release and environmental evidence for the product application.

Risk relevance: Provides product-specific decision context.

Key limitations: Commissioning, exact final version, release date and stable public provenance are not established, and primary claims require source-level verification.

Catalogue ID: STUDY-01216. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Florence Nono Almeida. 2026. Py-GC-MS/MS quantification of microplastics in vertebrate tissues: Addressing false positives of polyethylene. Journal of Hazardous Materials, 501 (2026) 140658.

Design: Analytical validation study of Py-GC-MS/MS polymer measurement in vertebrate tissues, focused on controlling false-positive polyethylene signals.

Findings: The study demonstrated that biological matrices can generate interfering products and that optimized cleanup and marker selection are required for defensible polyethylene quantification.

Risk relevance: High-value method evidence for evaluating polymer-marker claims in tissue.

Key limitations: Destructive thermal analysis does not establish intact-particle number, morphology or tissue location; performance must be validated for each matrix and polymer.

Catalogue ID: STUDY-00284. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Guilloux, A. 2024. WHO Activities on Microplastics. World Health Organization presentation; local PDF(s). Unresolved issue: The exact WHO presentation venue, presentation date, public archival record and authoritative final slide-deck version remain unresolved.

Design: World Health Organization presentation summarizing WHO activities and evidence priorities on microplastics.

Findings: The slides describe assessment work, methodological gaps and research or policy activities.

Risk relevance: Provides institutional context, not a new occurrence or health-effect dataset.

Key limitations: Venue, date, authoritative final slide deck and stable archive are unresolved; presentation wording should not be treated as a formal WHO risk conclusion.

Catalogue ID: STUDY-01244. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Kim, U.-J., Ghalamkari, M., Alvarez, P., Lee, A., & Nguyen, J. K. (2026). Quantifying micro- and nanoplastics in blood, urine, and soft tissues using optimized cascaded microfiltration and pyrolysis-GC/MS for exposomic investigations. Environmental Pollution 405, 128553. <https://doi.org/10.1016/j.envpol.2026.128553>.

Design: Analytical method study quantifying polymer-associated material in blood, urine and soft tissues using cascaded microfiltration and Py-GC/MS.

Findings: The study reported an optimized exposomic workflow and polymer-marker measurements across biological matrices.

Risk relevance: Relevant to biological-matrix exposure measurement and method feasibility.

Key limitations: Py-GC/MS destroys particles and cannot establish intact-particle number, morphology or location; matrix interference, recovery, contamination and clinical representativeness remain decisive.

Catalogue ID: STUDY-01676. DOI/link: 10.1016/j.envpol.2026.128553 Verification: V3.

Lemstra, P. J. (2024). [Dutch-language book extract/proof discussing plastic waste and microplastics; final publication metadata not established]. **Unresolved issue:** The Dutch-language book extract/proof remains without a verified final title, publisher, edition, publication date or stable final-publication source.

Design: Dutch-language book extract or proof discussing plastic waste and microplastics.

Findings: The document provides general explanatory or interpretive material rather than a distinct experiment.

Risk relevance: Background and communication context only.

Key limitations: Final title, publisher, edition, publication date and stable source are unresolved, so it should not be counted as independent evidence.

Catalogue ID: STUDY-01231. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Papatheodorou, G., and Karapanagioti, H. K. 2018. Degradation of Plastic Debris in Different Marine and Coastal Environments. Sixth International Marine Debris Conference, San Diego, 12–16 March 2018. Unresolved issue: The conference contribution is identifiable at topic/session level, but the exact authoritative abstract or presentation record, final version and sufficient method detail remain unresolved.

Design: Conference contribution on degradation of plastic debris in different marine and coastal environments.

Findings: The contribution discusses environment-dependent weathering and degradation processes.

Risk relevance: Provides conference-level fate and mechanism context.

Key limitations: The exact abstract or presentation, final version and sufficient methods are unavailable; conference-level information cannot control quantitative conclusions.

Catalogue ID: STUDY-01364. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Plastics Research Council / DeArmitt, C. 2026. Microplastics Evidence Review: What the Science Actually Shows. Evidence review; successive draft/final materials. Unresolved issue: The official landing page still labels the downloadable report as “Finalized Version Coming Soon,” while the canonical record carries 26 local copies/drafts; the exact finalized public version and canonical version lineage remain unresolved.

Design: Earlier Plastics Research Council evidence-review drafts and related public materials.

Findings: The materials synthesize the evolving project evidence base and communication conclusions.

Risk relevance: Project provenance and prior-synthesis context only.

Key limitations: Multiple draft copies and uncertain finalized public lineage create duplication risk; this is not an independent source and must not be used to validate the current report.

Catalogue ID: STUDY-01119. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Razegheh Akhbarizadeh, Yan Jin Xu, Freya Boerner, Paul A. Helm & Miriam L. Diamond. 2025. Optimized Extraction Methods for Pristine and Aged Microplastics from Complex Water Samples. ACS ES&T Water.

Design: Analytical method-development study optimizing extraction of pristine and aged microplastics from complex water matrices.

Findings: Optimized procedures improved recovery for selected polymers and showed that aging and matrix composition alter extraction performance.

Risk relevance: Supports more reliable environmental-water measurements.

Key limitations: Recovery with selected particles and waters does not establish equal performance for all polymers, sizes or real matrices; blanks and independent validation remain necessary.

Catalogue ID: STUDY-00493. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Royal Society of Chemistry-hosted workshop/presentation materials, circa 2017. Microplastics in Freshwater Systems / Microplastics in Terrestrial Systems. Local PDFs; exact presenter and publication metadata unresolved. Unresolved issue: The exact Royal Society of Chemistry workshop/presentation title, presenter, event date and authoritative archived source remain unresolved; the previously recorded Envirotech page is not an equivalent source and has been removed.

Design: Royal Society of Chemistry-hosted workshop or presentation materials on freshwater and terrestrial microplastics.

Findings: The materials summarize occurrence, methods and research questions across freshwater and soil systems.

Risk relevance: Historical workshop and evidence-mapping context.

Key limitations: Exact title, presenter, event date and authoritative archive are unresolved; the files are not independent peer-reviewed studies.

Catalogue ID: STUDY-01308. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Santiago v. Boston Scientific. 2024. Expert Opinion Concerning Marlex Polypropylene Mesh. Litigation expert report; local PDF. Unresolved issue: The litigation expert report remains local-only; the expert author, court, docket, filing date, public authentication, and version status are not sufficiently established.

Design: Local litigation expert report concerning Marlex polypropylene mesh.

Findings: The report evaluates material, degradation, biological response or product-safety claims in a legal context.

Risk relevance: Product-specific technical and expert-opinion context only.

Key limitations: Expert identity, court, docket, filing date, public authentication and version status are insufficiently established; litigation opinion is not an independent microplastics study.

Catalogue ID: STUDY-01192. DOI/link: No verified DOI or stable link recorded. Verification: V3.

Supplementary sources outside the frozen baseline

Cardona Orozco, S. (2025). Microplastics in Pipelines: Water Hammer Effects and Identification Protocol Validation [Master's thesis, Universidad de los Andes]. Institutional repository record indexed in 2026.

Design: Exploratory laboratory protocol-development thesis using controlled hydraulic transients, pre/post sampling, extraction and Raman identification.

Findings: The study demonstrated that the protocol could detect a few PVC-consistent particles in selected post-water-hammer samples; it was designed for feasibility rather than causal release-rate estimation.

Risk relevance: Useful method-development and source-hypothesis evidence for drinking-water pipes.

Key limitations: Particle counts were generally zero to two, PVC was the only material completed under the final protocol, a background sample contained PVC, no direct pipe-wall damage was measured and the design cannot establish that water hammer caused release or quantify a rate.

Catalogue ID: STUDY-01235. DOI/link: <https://repositorio.uniandes.edu.co/entities/publication/0265bc5c-b591-4825-9922-f1a1f9f85cbf> Verification: V1.

Song, J., et al. (2024). Internal and external microplastic exposure in young adults: A pilot study involving 26 college students in Changsha, China. Environmental Research, 263, 120250. <https://doi.org/10.1016/j.envres.2024.120250>.

Design: Pilot exposure study of 26 students using Py-GC/MS polymer-marker measurements in diet, water, air and biological samples.

Findings: The authors reported 460.20 micrograms/kg body weight/day of apparent external polymer-equivalent exposure, approximately 27.1 mg/day at the median body weight.

Risk relevance: The strongest high-mass polymer-marker result considered in the exposure reconciliation.

Key limitations: Py-GC/MS does not verify intact particles, number, size or morphology; one indoor and one outdoor air sample, inconsistent food ranges and complex-matrix interference make the value unsuitable for pooling with particle-resolved exposure estimates.

Catalogue ID: EXT-CORRECTION-SONG-2024. DOI/link: 10.1016/j.envres.2024.120250 Verification: V3.

Sánchez, A., et al. (2026). Effects of aged and non-aged packaging-derived PVC microplastics in female BALB/c mice. Toxicology, 154427. <https://doi.org/10.1016/j.tox.2026.154427>.

Design: Seventy-day oral-gavage study in female BALB/c mice comparing aged and non-aged packaging-derived PVC microplastics at 1 mg/kg/day.

Findings: The study reported oxidative-stress, inflammatory, histological and gut-microbiota changes under the administered conditions, with some differences between aged and non-aged material.

Risk relevance: Important positive mammalian hazard evidence for a more product-relevant PVC preparation.

Key limitations: The administered mass was approximately 120,000 times the selected modeled adult external exposure, only females were tested and no matched nonplastic particle comparator isolated a plastic-specific effect.

Catalogue ID: EXT-SANCHEZ-2026. DOI/link: 10.1016/j.tox.2026.154427 Verification: V3.

Yadav, V., Sethulekshmi, S., & Shrivastav, A. (2022). Estimation of microplastic exposure via the composite sampling of drinking water, respirable air, and cooked food from Mumbai, India. Environmental Research, 213, 113735. <https://doi.org/10.1016/j.envres.2022.113735>.

Design: Composite food, air and drinking-water occurrence study using Nile Red screening and geometric count-to-mass conversion.

Findings: The study reported about 2,012 particles/person/day and calculated 122-203 mg/day by assigning idealized geometries, including a 20 micrometre fibre diameter and a 2 mm mean size for the largest bin.

Risk relevance: A useful method-stress case showing how count-to-mass assumptions can dominate a headline exposure estimate.

Key limitations: The mass was calculated rather than measured and is dominated by geometric assumptions; only selected suspected particles received FTIR examination, so the value is not commensurate with particle-resolved integrated exposure estimates.

Catalogue ID: EXT-YADAV-2022. DOI/link: 10.1016/j.envres.2022.113735 Verification: V3.

Conclusion-relevant human evidence and exposure synthesis

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

Source reviewed: Official publisher source or authoritative abstract/metadata; rechecked 25 July 2026.

Design: Systematic review of 25 living-human studies identified through five databases, with duplicate screening/extraction and ROBINS-E appraisal.

Findings: Associations were reported across cardiovascular, reproductive, gastrointestinal, respiratory and ocular domains. Eleven studies were rated low ROBINS-E risk, eight had some concerns and six were high risk.

Risk relevance: Important current synthesis of direct human in-vivo evidence. It confirms that human association evidence has expanded while concluding that heterogeneity and bias constrain causal inference.

Key limitations: ROBINS-E risk of bias is not equivalent to validated plastic exposure, causal sufficiency or proof of disease causation. No meta-analysis was possible because of heterogeneity.

Catalogue ID: EXT-TRAN-2026. DOI/link: 10.1186/s12940-026-01282-y Verification: V1.

Xu, J., Qu, J., Jin, H., & Mao, W. (2025). Associations between microplastics in human feces and colorectal cancer risk. Journal of Hazardous Materials, 495, 139099.

Source reviewed: Official publisher source or authoritative abstract/metadata; rechecked 25 July 2026.

Design: Case-control study of 258 colorectal-cancer cases and 493 controls using laser infrared imaging of fecal samples.

Findings: Median fecal counts were 62 versus 43 items/g dry weight; the highest quartile had an adjusted odds ratio of 11.3 versus the lowest.

Risk relevance: Strong contrary human association that warrants replication and improved exposure assessment.

Key limitations: Fecal particles are not a validated historical internal-dose measure; reverse causation, diet, transit, treatment and disease physiology may explain or amplify the association.

Catalogue ID: EXT-XU-2025. DOI/link: 10.1016/j.jhazmat.2025.139099 Verification: V2.

Nihart, A. J., Garcia, M. A., El Hayek, E., et al. (2025). Bioaccumulation of microplastics in decedent human brains. *Nature Medicine*, 31, 1114-1119.

Source reviewed: Official publisher source or authoritative abstract/metadata; rechecked 25 July 2026.

Design: Postmortem organ study using Py-GC/MS and complementary methods, with temporal and dementia subgroup comparisons.

Findings: The paper reported higher polymer-associated mass in brain than liver or kidney and higher values in dementia cases.

Risk relevance: Important human-tissue occurrence and association evidence.

Key limitations: Postmortem association does not establish causation. An author correction was issued, and BfR concluded that the quantities appear implausibly high and false or misassigned signals cannot be excluded.

Catalogue ID: EXT-NIHART-2025. DOI/link: 10.1038/s41591-024-03453-1 Verification: V1.

He, P., Wang, F., Xi, G., et al. (2025). Association of microplastics in human cerebrospinal fluid with Alzheimer's disease-related changes. *Journal of Hazardous Materials*, 494, 138748.

Source reviewed: Official publisher source or authoritative abstract/metadata; rechecked 25 July 2026.

Design: Two small CSF cohorts comparing amyloid-positive and amyloid-negative participants and selected one-year cognitive change.

Findings: Four polymers were reported; PE and PVC measurements were higher in amyloid-positive subjects and selected PE measures correlated with biomarkers and cognition.

Risk relevance: Conclusion-relevant neurological association evidence.

Key limitations: Small selected cohorts, disease-state sampling, uncertain exposure history, multiple analyses and biological-matrix analytical uncertainty prevent causal inference.

Catalogue ID: EXT-HE-2025. DOI/link: 10.1016/j.jhazmat.2025.138748 Verification: V2.

Paolisso, P., Scisciola, L., Belmonte, M., et al. (2026). Micro- and nano-plastics in the coronary circulation and air pollution exposure in ischaemic heart disease presentation. *European Heart Journal*, ehag447.

Source reviewed: Official publisher source or authoritative abstract/metadata; rechecked 25 July 2026.

Design: Cross-sectional multicentre study of 61 angiography patients comparing STEMI, chronic coronary syndrome and normal-coronary controls using Py-GC/MS and LDIR.

Findings: MNP detection occurred in 84.2% of STEMI patients, 40% of chronic-coronary patients and 31.8% of controls. Smoking was the only independent predictor of MNP presence.

Risk relevance: Recent cardiovascular occurrence and association evidence.

Key limitations: Small selected clinical sample, acute disease, smoking and PM2.5 co-exposure, and method differences limit causal attribution.

Catalogue ID: STUDY-01660. DOI/link: 10.1093/eurheartj/ehag447 Verification: V1.

Heo, S. J., Moon, N., & Kim, J. H. (2024). A systematic review and quality assessment of estimated daily intake of microplastics through food. *Reviews on Environmental Health*, 40(2), 371-392.

Source reviewed: Official publisher source or authoritative abstract/metadata; rechecked 25 July 2026.

Design: Systematic review and quality assessment of 76 dietary exposure studies.

Findings: Reported daily-intake estimates varied across many orders of magnitude because studies covered different foods, particle sizes, methods and units.

Risk relevance: Supports the report's conclusion that dietary count estimates are highly method-dependent.

Key limitations: Count ranges cannot be pooled with mass estimates or treated as one exposure interval; source studies varied widely in analytical quality.

Catalogue ID: EXT-HEO-2024. DOI/link: 10.1515/reveh-2024-0111 Verification: V2.

Monikh, F. A., Materić, D., Valsami-Jones, E., et al. (2025). Challenges in studying microplastics in human brain. *Nature Medicine*, 31, 4034-4035.

Source reviewed: Official Nature Medicine article page; accessed 25 July 2026.

Design: Matters Arising critique of the Nihart et al. decedent-brain study.

Findings: The authors challenged digestion validation, quality-control design, contamination control and potential biological-matrix interference in polymer-mass assignment.

Risk relevance: Directly affects confidence in a conclusion-controlling high-profile brain-burden claim.

Key limitations: This is a methodological critique rather than an independent tissue dataset; it narrows confidence but does not itself prove that every reported signal was false.

Catalogue ID: EXT-MONIKH-2025. DOI/link: 10.1038/s41591-025-04045-3 Verification: V1.

Campen, M. J., West, A. B., Garcia, M., Gullapalli, R., & El Hayek, E. (2025). Reply to: Challenges in studying microplastics in human brain. *Nature Medicine*, 31, 4036-4037.

Source reviewed: Official Nature Medicine article page; accessed 25 July 2026.

Design: Author reply to the Monikh et al. methodological critique.

Findings: The authors acknowledged uncertainties in current nanoplastic detection and defended digestion, quality-control and interpretation choices.

Risk relevance: Required for balanced adjudication of the formal dispute surrounding the Nihart brain study.

Key limitations: A reply does not independently validate the reported brain mass; unresolved matrix and method uncertainty remains.

Catalogue ID: EXT-CAMPEN-2025. DOI/link: 10.1038/s41591-025-04046-2 Verification: V1.

Li, R., Lin, F., Zhao, X., et al. (2026a). Microplastics and nanoplastics in brain tumours and the healthy human brain. Nature Health, 1, 633-646.

Source reviewed: Official Nature Health article page and correction notice; accessed 25 July 2026.

Design: Cross-sectional tissue study of 156 diseased samples from 113 living brain-tumour patients and 35 healthy-brain samples from five postmortem donors using complementary analytical methods.

Findings: MNP-associated signals were nearly universal; peritumoural tissue had higher concentrations than healthy brain, and particle surface area correlated with tumour proliferation.

Risk relevance: Strong new living-human brain occurrence and disease-state association evidence.

Key limitations: Tumour-associated barrier disruption, living-versus-postmortem comparison, five healthy donors, selected disease tissue and uncertain exposure history prevent causal inference. The correction changed small rounded figure values, not the text values.

Catalogue ID: EXT-LI-BRAIN-2026. DOI/link: 10.1038/s44360-026-00091-4; correction 10.1038/s44360-026-00132-y Verification: V1.

Li, X., Wang, Y., Feng, Z., et al. (2026b). Microplastics and nanoplastics in matched human blood, bone, and intervertebral discs: Accumulation patterns and risks. npj Emerging Contaminants, 2, 22.

Source reviewed: Official full publisher article; accessed 25 July 2026.

Design: Matched blood, bone and intervertebral-disc pilot in 21 spinal-fusion patients; Raman MPs in all donors and Py-GC/MS mass analysis in a five-donor subset.

Findings: MPs were found in all blood and disc samples and 20 of 21 bone samples; the authors reported tissue-specific patterns and polymer-associated nanoplastic mass in the subset.

Risk relevance: New matched-tissue occurrence evidence and a testable retention hypothesis.

Key limitations: Small selected surgical cohort, no population controls, five-donor Py-GC/MS subset and destructive mass analysis that cannot verify nanoplastic size or morphology. The authors describe the findings as preliminary and long-term toxicity as unestablished.

Catalogue ID: EXT-LI-MATCHED-2026. DOI/link: 10.1038/s44454-026-00041-5 Verification: V1.

Chen, H., Liu, Z., Qiu, W., et al. (2026). Tumour recurrence, gastrointestinal symptoms and inflammation associated with microplastic-positive intestinal tumours. Nature Health.

Source reviewed: Official Nature Health article page and source metadata; accessed 25 July 2026.

Design: Clinical intestinal-tumour tissue series of 188 specimens using confocal micro-Raman, Raman imaging-SEM and hyperspectral imaging, with mean follow-up of 34.1 ± 17.4 months.

Findings: MPs were detected in 106 specimens; MP-positive tumours were associated with localized inflammation, histopathology, greater gastrointestinal symptoms and a trend toward recurrence.

Risk relevance: Important disease-tissue association with in-situ particle localization and clinical follow-up.

Key limitations: Detection within an established tumour is not a historical exposure measure; disease physiology, tissue retention, residual confounding and detection-group selection prevent inference that particles initiated disease or recurrence.

Catalogue ID: EXT-CHEN-2026. DOI/link: 10.1038/s44360-026-00123-z Verification: V1.

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

Source reviewed: PubMed and publisher bibliographic metadata/abstract; accessed 25 July 2026.

Design: Case-control study comparing cerebrospinal-fluid microplastic concentrations in intracranial-aneurysm cases and controls.

Findings: Higher CSF concentrations were associated with intracranial-aneurysm risk.

Risk relevance: New neurological and cerebrovascular association evidence.

Key limitations: One-time disease-state CSF measurement, selected clinical sampling, uncertain historical exposure and temporality, and potential residual confounding prevent causal attribution.

Catalogue ID: EXT-FANG-2026. DOI/link: 10.1016/j.envpol.2026.127788 Verification: V2.

Lu, W., Xu, X., Fan, L., et al. (2026). Association between exposure to microplastics and lipid disorders: A case-control study. Journal of Advanced Research.

Source reviewed: PubMed authoritative abstract and bibliographic metadata; accessed 25 July 2026.

Design: Case-control analysis of 239 adult bronchoscopy patients using LDIR-identified microplastics in bronchoalveolar-lavage fluid.

Findings: Each 100-unit increase in overall microplastics and selected polyethylene metrics was associated with modestly higher odds of hyperlipidemia or dyslipidemia and higher total cholesterol.

Risk relevance: New metabolic association evidence in a living-human clinical population.

Key limitations: Selected respiratory-clinic population, one-time BALF measurement, small effect increments, uncertain systemic-dose relevance, residual confounding and reverse causation prevent causal interpretation.

Catalogue ID: EXT-LU-2026. DOI/link: 10.1016/j.jare.2026.02.020 Verification: V2.

Nonplastic boundary context: tire-rubber leachate and 6PPD-Q (not plastic evidence)

Tian et al. 2021 / Tian et al. 2022. Linked primary studies identified 6PPD-quinone (6PPD-Q), an oxidation product of the tire antiozonant 6PPD, as the principal causal toxicant in toxic tire-wear/runoff leachate for coho salmon. The commercial-standard reassessment gave a juvenile coho LC50 of 95 ng/L (95% CI 80-110 ng/L). **Scope boundary:** this is strong source-specific aquatic chemical-hazard evidence for tire rubber, which is an elastomer and not a plastic. It does not establish rubber-particle toxicity, human-health risk, or plastic/microplastic toxicity and is excluded from the microplastic evidence count. DOI: 10.1126/science.abd6951; 10.1021/acs.estlett.1c00910.

Appendix I. Search History, Screening and Exclusion Framework

This evidence programme is a version-controlled living corpus, not a completed prospectively registered PRISMA systematic review. Historical search-log limitations affect reproducibility of the original discovery process; they do not remove source-level traceability of the 1,661 canonical records or the explicit appraisal of conclusion-controlling evidence. The corpus grew through versioned acquisitions, source verification, publication-lineage control, scientific appraisal and documented insertions. This appendix discloses what is exactly recoverable, distinguishes it from reconstructed descriptions and establishes a prospective update protocol without inventing historical raw-result, deduplication or exclusion-flow counts that were not retained.

11. Documentation status and recoverable search history

- Exact canonical-corpus accounting begins after record identity and publication-lineage resolution: 1,661 unique canonical IDs in the version-controlled database baseline.
- The earliest corpus-building search dates, complete verbatim strings, raw result totals and pre-canonical deduplication counts were not retained in one version-controlled log. No PRISMA identification total is therefore claimed.
- Versioned project packages retain source links, DOI and citation status, evidence role, publication lineage, acquisition/review status, appraisal fields, exclusions, corrections and adjudication history at record level.
- This edition records the exact official-domain agency-refresh queries and establishes prespecified concept blocks for future updates. These new strings are prospective and must not be represented as the verbatim legacy search.

Table I1. Documented search, acquisition and verification episodes

Stage	Date/period	Purpose and sources	Documentation status	Exact result/accounting boundary
Legacy corpus construction before formal version-controlled workflow	Historical / exact dates not retained in one version-controlled log	Broad acquisition of microplastics evidence and contextual particle sources Sources: User-supplied full texts and abstracts; title/DOI/source discovery; references and authoritative records represented in the canonical database	Not recoverable as one complete verbatim log	Pre-canonical raw hits and deduplication counts unavailable; no PRISMA identification total claimed Describe as legacy acquisition, not a completed systematic search.
Formal provenance and scientific-appraisal workflow	July 2026	Identity resolution, DOI/publisher/repository checking, publication-lineage control, source appraisal and residual-priority closure Sources: DOI records, publisher pages, repositories, retraction/correction records, supplied full texts and version-controlled database fields	Source-specific queries and verification outcomes preserved unevenly across versioned packages; no consolidated raw-result log	Canonical corpus held at 1,620 records before the documented insertion. This was record adjudication, not a de novo PRISMA search.
Canonical insertion	23 July 2026	Add separately adjudicated missing/high-priority records without disturbing prior IDs Sources: Supplied studies, abstracts, publisher/repository metadata and controlled insertion records	Insertion provenance and source identity recorded; original discovery queries not uniformly retained	41 records inserted; canonical corpus increased from 1,620 to 1,661 Insertion count is exact; raw discovery-hit count is not claimed.
Targeted currency review	25 July 2026	Identify current realistic-dose human/animal evidence, systematic reviews, methodological critiques and agency developments capable of changing conclusions Sources: Primary publisher pages, PubMed/authoritative metadata, official agency pages and targeted title/topic/DOI searches	Source list and adjudications retained; complete verbatim query log and result counts were not retained	Supplementary sources outside the frozen baseline were added to the manuscript and Appendix H; the database remains unchanged pending separate source-level adjudication. Do not represent this as a comprehensive systematic update or use dynamic search-result counts as a flow metric.

Stage	Date/period	Purpose and sources	Documentation status	Exact result/accounting boundary
Official-agency refresh	25 July 2026	Confirm current FDA, EPA, EFSA, BfR, COT and WHO positions immediately before public release. Sources: official agency domains only.	Exact queries recorded in Appendix I and this audit file	Six agency positions refreshed; no agency statement established general-population disease causation Agency positions are scope-specific; an EPA draft CCL listing is not a regulatory determination.
Cited-source verification and final-release update	25 July 2026	Verify cited and conclusion-controlling source identity, design, findings, limitations and stable identifiers; incorporate four independently verified final-release sources. Sources: official publisher full text or pages, primary institutional sources, authoritative abstracts and version-controlled database metadata.	Verification basis recorded per entry, rather than as a database-search log	146 unique Appendix H entries after the oral-toxicology supplement incorporated into Version 1.0. Appendix H is not every primary record in the database.
Focused common-polymer oral-toxicology supplement	26 July 2026	Recover and adjudicate additional PE, PVC and PET oral evidence capable of improving polymer-specific context. Sources: supplied Feron full text, Welle and Franz review, official publisher or agency full text/abstracts, and targeted title/DOI searches.	Source-level findings and verification boundaries recorded in the manuscript and Appendix H; dynamic result counts not used.	Five supplementary sources added during the oral-toxicology supplement incorporated into Version 1.0; frozen database counts unchanged. Archival PE and PVC contexts are not formal NOAELs, and acute/mechanistic PET studies are not inserted into the principal dose-ratio figure.
Focused human-biomonitoring QA/QC supplement	29 July 2026	Incorporate an independent risk-assessment-fitness framework for human organ, tissue and bodily-fluid measurements. Sources: Lane et al. 2026 full text and three supplementary files.	Full article and supplementary scoring/database files supplied and directly reviewed; no de novo database-search result counts claimed.	133 datasets from 101 studies evaluated against 14 essential criteria; none met all criteria for direct quantitative risk assessment. One supplementary source added to the manuscript and Appendix H; frozen database counts unchanged; Appendix H total increased to 147.
Focused retained-lung inorganic-PM context supplement	13 August 2026	Incorporate the user-supplied Hayden et al. 2023 peer-reviewed full text to strengthen route-matched nonplastic lung-particle context and FE-SEM/EDS method interpretation. Source: supplied publisher PDF/full article.	Full article directly reviewed; no de novo database search or search-result count claimed.	One supplementary primary context source added to the manuscript, Appendix D and Appendix H; frozen 1,661-record database counts and grades unchanged. Appendix H total increased from 156 to 157. Source is not used as a population burden or plastic-fraction denominator.
Focused inhaled-nanoparticle translocation supplement	13 August 2026	Incorporate the user-supplied Miller et al. 2017 ACS Nano full article, supporting information and published correction to strengthen route-specific pulmonary translocation and size-dependence context. Sources: supplied publisher PDFs.	Main article, supporting information and correction directly reviewed as one publication lineage; no de novo database search or search-result count claimed.	One supplementary primary kinetic/context publication lineage added to the manuscript, Appendix D and Appendix H; frozen 1,661-record database counts and grades unchanged. Appendix H total increased from 157 to 158. The corrected ~0.02% estimate is retained as illustrative, not a microplastic absorption fraction.

Stage	Date/period	Purpose and sources	Documentation status	Exact result/accounting boundary
Focused nonplastic tire-rubber leachate boundary supplement	13 August 2026	Incorporate Tian et al. 2021 and the revised Tian et al. 2022 6PPD-quinone toxicity assessment as a clear nonplastic boundary example. Sources: peer-reviewed primary articles and publisher records.	Primary articles and revised toxicity assessment directly reviewed; no de novo search-result count claimed.	One combined nonplastic boundary-context summary added to the manuscript, Appendix D and Appendix H; frozen 1,661-record database counts unchanged. Appendix H total increased from 158 to 159. Tire rubber is an elastomer, not plastic, and the evidence is excluded from plastic-toxicity conclusions.
Focused comparative dust-hazard and risk-communication context supplement	18 August 2026	Strengthen the central public-health interpretation with peer-reviewed evidence on generic particle overload, established hazards from respirable silica, occupational wood dust and toxic metals/metalloids, and nocebo effects. Sources: publisher and PubMed records for Borm & Driscoll 2019; Steenland et al. 2001; Demers et al. 1995; Tchounwou et al. 2012; Colloca & Barsky 2020.	Five peer-reviewed sources directly checked at publisher or authoritative bibliographic level; no de novo systematic-search result count claimed.	Five supplementary context sources added to the manuscript, Appendix D and Appendix H; frozen 1,661-record database counts and grades unchanged. Appendix H total increased from 159 to 164. Sources support generic high-dose particle effects, specific nonplastic hazards and proportionate communication; they are not plastic-specific toxicity records.

12. Exact official-agency refresh queries

The following queries were executed on 25 July 2026 using official-domain restrictions. Dynamic search-engine result counts were not used as evidence-flow metrics; the authoritative agency page was selected and reopened.

Table 12. Exact official-domain queries

Organization/purpose	Exact query	Selection rule
FDA	site:fda.gov food microplastics current evidence human health 2026 FDA	Dynamic result count not used; authoritative agency page selected and reopened
EPA	site:epa.gov microplastics draft CCL 6 July 2026 official	Dynamic result count not used; authoritative agency page selected and reopened
EFSA	site:efsa.europa.eu microplastics human health scientific opinion 2026 official	Dynamic result count not used; authoritative agency page selected and reopened
BfR	site:bfr.bund.de microplastics health risk 2026 official	Dynamic result count not used; authoritative agency page selected and reopened
COT	site:cot.food.gov.uk microplastics inhalation statement official conclusion risk assessment	Dynamic result count not used; authoritative agency page selected and reopened
WHO	site:who.int microplastics human health report 2022 official	Dynamic result count not used; authoritative agency page selected and reopened
EPA interpretation	site:epa.gov ccl 6 microplastics not regulated risk determination official	Dynamic result count not used; authoritative agency page selected and reopened
EFSA schedule	site:efsa.europa.eu microplastics scientific opinion expected 2026 2027 official	Dynamic result count not used; authoritative agency page selected and reopened

13. Prospective minimum search blocks for future updates

These strings are established prospectively for future updates. They are not presented as reconstructed verbatim legacy searches. Syntax should be translated for each database while preserving the concept blocks, date of execution, database or platform, result count, export file and deduplication log.

Human health and biomonitoring: ((microplastic* OR nanoplastic* OR "plastic particle*" OR microfiber* OR microfibre*) AND (human* OR patient* OR blood OR tissue* OR organ* OR placenta* OR brain OR lung* OR cardiovascular OR cancer OR fertility OR epidemiolog* OR cohort OR "case-control"))

Toxicology and kinetics: ((microplastic* OR nanoplastic* OR "plastic particle*") AND (toxic* OR hazard* OR adverse OR inflammation OR oxidative OR genotoxic* OR reproductive OR developmental OR kinetic* OR absorption OR distribution OR clearance OR retention OR mouse OR mice OR rat OR zebrafish OR "in vitro"))

Exposure and occurrence: ((microplastic* OR nanoplastic* OR microfiber* OR microfibre*) AND (exposure OR intake OR ingestion OR inhalation OR food OR beverage OR "drinking water" OR tap OR air OR dust OR soil OR household OR occupational))

Analytical methods and quality assurance: ((microplastic* OR nanoplastic*) AND (Raman OR FTIR OR "infrared spectroscopy" OR pyrolysis OR "GC/MS" OR "mass spectrometry" OR "Nile Red" OR microscopy OR validation OR recovery OR blank* OR contamination OR interlaboratory OR reference material))

Ecology and environmental effects: ((microplastic* OR nanoplastic* OR microfiber* OR microfibre*) AND (ecotoxic* OR fish OR invertebrate* OR plankton OR marine OR freshwater OR sediment OR soil OR crop* OR plant* OR wastewater OR growth OR reproduction OR mortality OR behaviour OR behavior))

14. Screening, lineage and exclusion rules

- Stage 1 screens title, abstract and source metadata for relevance to occurrence, methods, exposure, kinetics, toxicology, human outcomes, ecological effects, fate, treatment or risk interpretation. Stage 2 uses the full text or authoritative complete record for conclusion-controlling appraisal whenever obtainable.
- Peer-reviewed primary studies are eligible as independent evidence. Systematic reviews and other syntheses are retained as secondary evidence and are not counted as new empirical datasets. Formal agency, standard, thesis or technical records are retained when they provide unique methods, exposure, engineering or policy context and are labeled by source type.
- Preprints, final articles, corrections, supplements, conference derivatives and media communications from the same underlying work are linked as one publication lineage. DOI, title/author/year, repository identifiers and experiment descriptions are used to prevent double counting.
- Supporting information, duplicates, media/public communication, administrative/non-evidence records, catalogue/research-integrity records and non-evidentiary correspondence remain visible for provenance but are excluded from the scientific-source count.
- Selection is not based on whether results are adverse, null or reassuring. Appraisal is based on method validity, exposure realism, control quality, plastic specificity, endpoint adversity, study design and causal reach.
- A source discovered after a version-controlled database freeze may be discussed as a supplementary source outside the frozen baseline. It does not change corpus counts, grades or database-derived figures until source-level adjudication, scoped cell changes, formula and count reconciliation, and a new version-controlled database release are complete.
- Where full text, language access or source verification is incomplete, the record is retained with an explicit verification or acquisition boundary rather than upgraded by inference.

15. Exact version-controlled corpus accounting

The following counts are mechanically reproduced from the version-controlled master TSV. They are post-canonical accounting totals and must not be presented as raw search hits.

Table I3. Evidence-role accounting

Evidence role	Records	Interpretation
Context only	71	Relevant context not used as independent scientific evidence.

Evidence role	Records	Interpretation
Exclude from scientific-source count	232	Retained for audit, duplicate, communication or administrative provenance.
Formal technical/context source	158	Agency, standard, thesis or formal technical context.
Independent primary evidence	643	Empirical/modeling/method records counted as primary evidence.
Pending source identification	1	Unresolved source identity; excluded from inferential use.
Secondary scientific synthesis	556	Reviews and syntheses; not independent empirical datasets.

Table I4. Independent-primary quality distribution

Quality	Records	Boundary
High	11	First-rater grade; no inter-rater consensus is claimed.
Moderate	297	First-rater grade; no inter-rater consensus is claimed.
Low	295	First-rater grade; no inter-rater consensus is claimed.
Very low	40	First-rater grade; no inter-rater consensus is claimed.

Table I5. Excluded/provenance taxonomy

Record class	Records	Treatment
Administrative/duplicate/non-evidence record	35	Visible for auditability; excluded from the scientific-source count.
Catalogue/research-integrity record	12	Visible for auditability; excluded from the scientific-source count.
Editorial/commentary/correspondence	1	Visible for auditability; excluded from the scientific-source count.
Media/public communication	105	Visible for auditability; excluded from the scientific-source count.
Supporting information/duplicate	79	Visible for auditability; excluded from the scientific-source count.

16. Flow statement and living-update rule

Because complete pre-canonical identification and deduplication totals are unavailable, this report does not present a PRISMA flow diagram. The exact documented flow is: acquired records were identity-resolved and canonicalized; the current version-controlled corpus contains 1,661 unique IDs; those IDs are classified into 643 primary, 556 secondary, 158 formal technical/context, 71 context-only, 232 excluded/provenance and one unresolved source-identification record. The 643 primary records comprise 11 High, 297 Moderate, 295 Low and 40 Very-low records. Appendix H contains 164 cited or conclusion-controlling summaries, including one combined nonplastic tire-rubber boundary-context summary that is excluded from the microplastic evidence count. Separately, 13 technical, review or context records have incomplete bibliographic or version provenance; Appendix C lists them and distinguishes them from the single unresolved source-identification record in the frozen corpus. An earlier 115-source internal development audit set predates later supplementary additions and is retained only as historical internal QA provenance; no external-review workflow is part of this release. Future updates should preserve the exact database or platform, execution date, query strings, exported result count, deduplication rules, screening decisions, exclusion reasons, publication lineage and supplementary-source change scope.

Appendix J. Structured Internal Consistency and Adversarial Audit

The manuscript was subjected to a structured internal consistency and adversarial audit from the perspectives of analytical chemistry, particle toxicology, exposure assessment, epidemiology, ecological risk, reproducibility and regulatory interpretation. AI-assisted tools supported the audit, which was an internal red-team exercise only and does not establish reviewer independence, professional credentials, inter-rater agreement or external endorsement. The 17-objection adjudication summary is retained in this appendix.

Table J1. Specialist challenge outcomes

Challenge domain	Principal accepted or bounded issue	Action in the final-release manuscript	Effect on central conclusion
Analytical chemistry	Orthogonal confirmation is stated as an absolute requirement for highest confidence, although a rigorously validated particle-resolved method can support a bounded finding within its demonstrated matrix and size range. Lower size cutoff, instrumental detection limit, identification threshold and quantification limit can be conflated.	Qualified the orthogonal-confirmation statement; separated instrument capability from validated matrix-specific identification/quantification cutoff; retained thermal methods for bounded polymer-mass inference.	Central conclusion unchanged; wording and/or boundaries revised.
Particle toxicology	Administered animal dose divided by modeled human external exposure may be mistaken for a formal margin of exposure or internal-dose comparison. A matched nonplastic comparator is not necessary for every occurrence, analytical-validation or physical-fate study.	Reinforced that dose ratios are scale diagnostics, not formal margins; preserved high-dose studies as mechanistic/hazard evidence; limited matched nonplastic comparator requirements to plastic-specific biological claims.	Central conclusion unchanged; wording and/or boundaries revised.
Exposure assessment	The 0.0012-0.0019% calculation could be misread as a universal fraction of all particles or as a safety threshold. Human nanoplastic uptake and long-term retention remain poorly quantified, so low external mass cannot by itself exclude a small subgroup or size-specific effect.	Restated the Nor/EPA ratio as a bounded cross-domain context rather than universal composition or risk; preserved uncertainty for nanoplastics, susceptible groups and unusual exposure.	Central conclusion unchanged; wording and/or boundaries revised.
Epidemiology	The central causal sentence should be framed as a causal-inference judgment rather than an absolute claim. Marfella and other prospective or clinical studies deserve more weight than cross-sectional occurrence studies.	Changed the central sentence to explicit causal-inference language; retained Marfella and current disease-state studies as important contrary evidence while preserving exposure-proxy, temporality, confounding and reverse-causation limits.	Central conclusion unchanged; wording and/or boundaries revised.
Ecological risk	A general statement that plastic is a minority of particles could obscure source-specific hotspots, entanglement/obstruction, formulation-specific toxicity or sensitive receptor effects. Natural-particle comparators can clarify generic particle effects but do not automatically prove ecological equivalence because persistence, buoyancy, shape and associated chemicals may differ.	Added explicit local-hotspot, receptor, obstruction/entanglement, persistence and formulation boundaries; comparator matching is not treated as proof of ecological equivalence.	Central conclusion unchanged; wording and/or boundaries revised.
Search and reproducibility	See complete adjudication matrix.	Corrected the overstatement that every historical search string was retained; added Appendix I and a prospective update protocol without invented PRISMA counts.	Central conclusion unchanged; wording and/or boundaries revised.
Agency interpretation	See complete adjudication matrix.	Refreshed FDA, EPA, EFSA, BfR, COT and WHO sources; clarified that EPA draft CCL 6 inclusion is not regulation or a risk determination.	Central conclusion unchanged; wording and/or boundaries revised.

Challenge domain	Principal accepted or bounded issue	Action in the final-release manuscript	Effect on central conclusion
Review design and change control	See complete adjudication matrix.	Retained internal audit materials; disclosed the author-final release design; kept supplementary sources outside the frozen baseline and outside the version-controlled database.	Central conclusion unchanged; wording and/or boundaries revised.

J1. Interpretation and limits

The internal challenge produced no change to the central risk conclusion or version-controlled database counts. It produced material refinements to causal wording, method-specific inference, dose-comparison language, comparator scope, ecological boundaries, search provenance and agency-process interpretation. This is an author-final synthesis; credible errors or conclusion-changing evidence identified after release should be documented, adjudicated at source level and propagated through a version-controlled correction. No inter-rater agreement statistic is applicable because independent second-rater grading was not part of the release design.

J2. Internal development audit materials

A manuscript-development workbook contains 115 records: all 11 High-quality primary records, all 66 mechanically identified conclusion-controlling Moderate records, 14 supplementary challenge sources, and a domain-diverse calibration sample of 12 Low and 12 Very-low primary records. It predates the later supplementary toxicology, source-recovery, atmospheric and human-biomonitoring QA/QC additions and associated source-level corrections. The workbook and reconciliation key are retained as historical internal QA provenance only; they are not required for release and are not represented as external review or independent validation.



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