Microplastics and the Human Body: Exposure, Translocation, and the Margin-of-Exposure Question

An evidence synthesis · Holistic Quality LLC

Author: Levi Robey · Holistic Quality LLC · **Contact:** levi@holisticquality.io

Version: 1.0 · **Published:** 2026-07-03 · **Last updated:** 2026-07-03

DOI: 10.5281/zenodo.21172814 (this version) · concept DOI 10.5281/zenodo.21172813 (always resolves to the latest version)

Revision note (2026-07-03): assigned the Zenodo version DOI 10.5281/zenodo.21172814 (concept DOI 10.5281/zenodo.21172813) and stated the CC BY 4.0 license in the document body. No change to the evidence, claims, or citations — the 2026-07-03 content review passed unchanged; this revision records the assigned DOIs and license only.

Document type: Working evidence synthesis (not peer-reviewed)

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> **How this was produced.** This synthesis was assembled with AI-assisted literature review and drafting, then **human-verified**: every primary study cited below was checked against its published record (journal, volume, issue, pages, and DOI confirmed; the characterized finding checked against the paper's abstract or full text), and figures that could not be traced to a verifiable source were removed or restated. Where the evidence is contested — and for microplastics and human health, most of it is — we say so. This is a **selective synthesis of representative, high-quality studies, not an exhaustive systematic review.** We regard transparency about method as part of the credibility of the result.

Executive summary

Microplastic and nanoplastic particles are now **detectable in human tissues and excreta**: independent studies have reported them in human stool, in human blood, and in human placental tissue. Estimates of human intake run to the **tens — and by some estimates hundreds — of thousands of particles per year** from food, drink, and inhaled air combined. That exposure is real, and it is measurable.

What is **not** established is harm. None of the human detection studies assessed health effects, and every major review of the field reaches the same calibrated conclusion: plausible hazard *mechanisms* exist — largely inferred from cell-culture and animal-model work, or by analogy to other particulates — but **whether current human exposure to microplastics causes disease is unknown and not yet demonstrated.** The World Health

Organization's assessment of microplastics in drinking-water judged the risk to be *low at current exposure levels*, while explicitly cautioning that this rests on a **limited evidence base with major knowledge gaps** — not a clean bill of health.

The honest state of the science is therefore a **detection-and-exposure certainty paired with a health-effects uncertainty**. The load-bearing open question is the **margin of exposure**: how the doses at which effects appear in laboratory systems compare to the doses humans actually receive. That question is not yet answered, principally because the sub-10-micron and nanoplastic fraction — the fraction most likely to cross biological barriers — is at or beyond the limit of current measurement, so real human exposure is probably *underestimated*, and because most toxicology uses pristine spherical plastic beads rather than the weathered, irregular fragments people are actually exposed to. This synthesis lays out what has been shown, what has not, and what would resolve the gap.

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1. Human biomonitoring: what has actually been detected

Three independent studies establish that microplastics reach the interior of the human body. Each is a **detection/quantification study in a small sample**; none was designed to, or did, measure a health outcome.

- **Human stool (Schwabl et al., 2019).** A prospective case series of **eight healthy volunteers** (aged 33–65) found microplastics in **all eight** stool samples, at a **median of 20 microplastic particles (50–500 µm) per 10 g** of stool, spanning **nine different polymer types** (polypropylene and polyethylene terephthalate the most abundant). The authors are explicit about the limits: few participants, a single sample each, and the origin and fate of the particles in the gut not investigated — "further research on … the potential effect on human health is needed." [1]
- **Human blood (Leslie et al., 2022).** In whole blood from **22 healthy donors**, four high-production-volume polymers were identified and quantified for the first time, with a **mean summed concentration of 1.6 µg/mL** of plastic particles ≥700 nm (polyethylene terephthalate, polyethylene, and polymers of styrene most common). An important precision point: this is a **mass-concentration** measurement (pyrolysis–gas-chromatography/mass-spectrometry), **not a particle count** — it says how much polymer mass was present, not how many discrete particles. The donor set is small, and the study explicitly did **not** assess whether this exposure constitutes a health risk. [2]
- **Human placenta (Ragusa et al., 2021).** Analysis of **six placentas** from physiological pregnancies found **12 microplastic fragments (5–10 µm) in four of the six**. This is genuine first-of-its-kind evidence of particles at the maternal–fetal interface, but it warrants careful reading: only **three of the twelve fragments were confirmed as an actual polymer (polypropylene)**; the other nine were characterized by pigment alone. Very small N, a low absolute particle count, partial polymer confirmation, and — again — no health-effects assessment. "First evidence" is the authors' own framing, and signals

preliminary status. [3]

What these establish, and what they do not. Together they show, from three different biological compartments and by three different methods, that microplastic material crosses into the human body and is not merely an external or gut-lumen contaminant. They do **not** establish a concentration–response relationship, a biological effect, or a disease association — none was measured. They are existence proofs of exposure, not evidence of harm.

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2. How much are humans exposed to?

Estimated intake (Cox et al., 2019). The most-cited estimate of human microplastic consumption evaluated ~26 studies against recommended dietary intakes and put **annual intake from food and beverages at roughly 39,000–52,000 particles**, rising to **74,000–121,000 particles per year once inhalation is added.** Three caveats are load-bearing and must travel with the number:

1. The **74,000 and 121,000 figures are the low and high endpoints across age and sex groups** — not a single universal per-person value.
2. The estimate covers only about **15% of caloric intake** (the fraction for which data existed); the authors state the values are "subject to large amounts of variation" and are **likely underestimates**.
3. These are **particle counts, not mass or dose of a toxicant** — a count says nothing on its own about biological effect.

The same analysis found drinking water to be a major and modifiable contributor: people who meet their water intake from **bottled water ingest on the order of 90,000 additional particles per year, versus about 4,000 for those drinking tap water.** [4]

The regulatory read (WHO, 2019). The World Health Organization's assessment, *Microplastics in drinking-water*, concluded that microplastics in drinking water **do not appear to pose a health risk at current exposure levels** ("low" risk, in the report's own terms) — but the conclusion is provisional by WHO's own framing: it rests on a **limited evidence base**, WHO flagged **significant knowledge gaps** and called for more research and standardized monitoring, and it judged the microplastic risk **far smaller than the well-established risk from waterborne pathogens**, which should remain the priority for drinking-water safety. This is a *reassuring-but-limited* finding, not a declaration that microplastics are safe; a synthesis that quotes "low risk" without the "on limited evidence, gaps remain" qualifier misrepresents the source. [5]

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3. What the toxicology shows — and why it does not yet transfer to humans

The hazard case for microplastics is built primarily on **cell-culture and animal-model** evidence and on **analogy** to better-studied particulates (ambient PM, engineered nanomaterials, occupational dusts). It is a case for *plausibility*, not for demonstrated human harm.

- **Model-system toxicity is real but its human relevance is unresolved (Yong et al., 2020).** A review of mammalian systems concludes that micro- and nanoplastics can accumulate in tissues and exert toxicity — oxidative stress, gut-microbiota disturbance, metabolic and inflammatory effects — **as demonstrated in mouse models and human cell lines**, while stating plainly that "the pathophysiological consequences of acute and chronic … exposure in the mammalian system, particularly humans, are yet unclear," with "negative, yet unclear long-term consequences." The mechanisms are extrapolated from models, not established in people. [6]
- **The human-health case is inferential (Wright & Kelly, 2017).** An earlier review reasons that plausible **particle, chemical, and microbial hazards** exist by analogy to complementary fields, but is explicit that "the human health effects … are unknown" and that "a robust evidence-base of exposure levels is currently lacking" — i.e., quantifying real-world exposure is the unmet prerequisite before health effects can be assessed. [7]
- **The calibrated expert position (Vethaak & Legler, 2021).** A *Science* Perspective by Vethaak & Legler makes the field's honest center of gravity explicit: humans continually inhale and ingest microplastics, yet whether these contaminants pose a substantial risk to human health remains far from settled, and closing that question depends on filling key knowledge gaps — above all the inability of current analytical tools to measure the **sub-10-µm and nanoplastic fraction**, which means true exposure is likely underestimated. [8]

The particle-translocation analogy — and its limits. The one place where a mechanism for small-particle entry into deep tissue is comparatively well characterized is *ambient ultrafine particulate matter*, where combustion-derived particles have been reported to translocate from the respiratory tract toward the brain, and magnetite nanoparticles consistent with combustion sources have been identified in human brain tissue. [9][10] These findings make it *plausible* that sufficiently small plastic particles could similarly cross barriers — but they are about combustion particulates, not plastics, and importing them wholesale would over-claim. They are cited here as a mechanistic analogy and a research pointer, not as evidence that microplastics do the same in humans.

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4. The margin-of-exposure question (the crux)

The scientifically decisive question is not "are microplastics present in people?" (they are) but **"how do the exposures at which effects appear compare to the exposures people actually receive?"** — the *margin of exposure* (MOE), the ratio of an effect threshold (a no-observed-adverse-effect level or benchmark dose from toxicology) to estimated real human intake. A large margin indicates low concern; a small one indicates a need for risk reduction.

That ratio **cannot yet be computed with confidence**, for structural reasons:

- **The numerator is uncertain.** Most in-vivo toxicology uses **pristine, spherical polystyrene beads at high doses**, whereas real exposure is to **weathered, irregular, chemically heterogeneous fragments** across many polymers and sizes. Effect thresholds derived from the former may not represent the latter, and dose metrics (mass? particle number? surface area?) are not standardized.
- **The denominator is probably understated.** Because the sub-10-µm and nanoplastic fraction is at or beyond current measurement limits (§3), estimates like Cox's almost certainly **miss the most biologically relevant particles**, so true intake — especially of the fraction most able to cross membranes — is likely higher than measured.
- **The exposure metric and the effect metric do not yet align.** Human intake is reported as particle *counts* (Cox) or polymer *mass* (Leslie); toxicology reports effects against still other dose metrics. Until these are reconciled, an apples-to-apples MOE is not available.

This is why the responsible summary is **"detected, plausibly hazardous by analogy, effect-versus-exposure margin unresolved"** — not "safe" and not "proven harmful."

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5. Detection versus demonstrated harm

The distinction that governs this whole literature deserves to be stated precisely, because it is the one most often collapsed in public discussion.

- **Detection** — that microplastics are present in human blood, stool, and placenta — is **established** (§1), within the limits of small samples and evolving methods.
- **Exposure** — that humans ingest and inhale large numbers of particles annually — is **established as an estimate** (§2), with the caveats that it is likely an underestimate and is a count, not a dose.
- **Harm** — that this exposure causes disease at current levels — is **not established.** No human study has demonstrated it; the mechanistic case is from models and analogy; and the field's own reviews and the WHO assessment converge on "unknown / low-at-current-levels-on-limited-evidence."

Presence in the body is a necessary condition for a health effect, but not a sufficient one. The prudent posture that this evidence supports is **exposure-reduction as reasonable precaution where it is cheap and easy** (e.g., the bottled-vs-tap-water differential), coupled with **honesty that a causal human-health claim is not yet available.**

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6. Key gaps

- **The nano fraction is unmeasured.** Analytical tools cannot yet reliably quantify sub-10-µm and nanoplastic particles — the fraction most able to cross membranes — so both exposure and internal dose are likely underestimated.
- **Toxicology does not match real exposure.** Pristine polystyrene beads at high doses are the workhorse of the literature; weathered, irregular, multi-polymer fragments at realistic doses are understudied.
- **No standardized dose metric.** Mass, particle number, and surface area are used inconsistently, preventing margin-of-exposure calculation.
- **No human effect data.** The detection studies do not follow health outcomes; there are no cohort studies linking measured internal microplastic burden to disease.
- **Additives and adsorbed chemicals** (plasticizers, flame retardants, sorbed pollutants) may carry risk distinct from the particles themselves and are often not separated out.

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7. Priority research directions

1. **Analytical methods for the nanoplastic fraction** (below ~1 µm), with reference materials and inter-laboratory standardization, so exposure and internal dose can be measured, not modeled.
2. **Toxicology on environmentally realistic particles** — weathered, irregular, mixed-polymer fragments at realistic doses — reporting a standardized dose metric.
3. **A margin-of-exposure analysis** once (1) and (2) exist: benchmark-dose modeling of effect thresholds against measured (not modeled) human intake, size- and polymer-stratified.
4. **A human cohort** linking a measured internal microplastic burden (blood/tissue) to prospectively collected health outcomes — the study none of the current detection papers is.
5. **Separation of particle effects from additive/adsorbate effects**, to attribute any hazard correctly.

How to cite

This page is the **full evidence review** — the citable version of record. Cite it as:

> Robey, L. (2026). *Microplastics and the Human Body: Exposure, Translocation, and the Margin-of-Exposure Question* (full evidence review, Version 1.0). Holistic Quality LLC. doi:10.5281/zenodo.21172814 · https://holisticquality.io/research/microplastics-and-the-human-body-full *This full report is the citable version of this work. The version DOI (doi:10.5281/zenodo.21172814) is frozen to this version (v1.0); the version-independent concept DOI (doi:10.5281/zenodo.21172813) always resolves to the latest version.*

A summary of this work is available as the summary brief.

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All citations were independently verified against their published sources (journal, volume, issue, pages, and DOI confirmed; the characterized finding checked against the paper). Toxicological statements in §3 reflect animal/in-vitro/cell-line evidence unless a human study is named; references 9–10 concern combustion-derived ultrafine particulates and are cited as a mechanistic analogy, not as evidence about plastics.

Disclosures

Competing interests. The author is the founder and principal of Holistic Quality LLC, the commercial publisher of this report, which develops regulator-facing safety-data and compliance products in subject areas that include environmental and chemical exposure. A sibling property under the same parent entity, the Institute for Cognitive Sovereignty, may cite this work in public advocacy. These constitute a competing interest. To mitigate it, every quantitative claim and citation was independently source-verified, the limits of the evidence are stated throughout, and the author retained full and sole editorial control — no commercial customer or third party reviewed or influenced the content.

Funding: none (self-funded). **Data availability:** synthesis of previously published literature; no new data were generated. **AI use:** produced with AI-assisted review and drafting, human-verified; the named author is responsible for all content. **ORCID:** 0009-0005-6946-3569. **Peer-review status:** self-published working paper; not peer-reviewed.