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21 U.S.C. §§ 342(a)(1), 346, 371(a)

November 23, 2026

Commissioner of Food and Drugs
U.S. Food and Drug Administration
10903 New Hampshire Avenue
Silver Spring, MD 20993

FDA Dockets Management Staff
U.S. Food and Drug Administration
5630 Fishers Lane, Room 1061
Rockville, MD 20852

Re: Citizen Petition to Regulate Microplastics in the American Food Supply

Dear Commissioner of Food and Drugs and Dockets Management Staff,

The Center for Biological Diversity and Food & Water Watch ("Petitioners") submit this citizen petition under sections 402(a)(1), 406, and 701(a) of the Federal Food, Drug, and Cosmetic Act ("the Act"), 21 U.S.C. §§ 342(a)(1), 346, 371(a); 5 U.S.C. § 553(e); and 21 C.F.R. §§ 10.25(a) and 10.30, to request that the Commissioner of Food and Drugs validate and publish methods for measuring microplastics, including nanoplastics, in food; establish action levels for microplastics in food under 21 C.F.R. §§ 109.4(c) and 109.6(d); and establish tolerances under section 406 of the Act, as set out in the Statement of Actions Requested.

A. STATEMENT OF ACTIONS REQUESTED

Petitioners request that the Food and Drug Administration (FDA) take the following actions. For purposes of this petition, "microplastics" means particles smaller than five millimeters in at least one dimension that are composed substantially of polyethylene, polypropylene, polystyrene, polyvinyl chloride, polyethylene terephthalate, polyurethane, or another synthetic polymer. The term includes nanoplastics, the subset of microplastics smaller than one micrometer.[1] These requests concern microplastics in food other than those present in accordance with a food additive regulation or an effective food-contact notification under section 409 of the Act.[2]

[1] U.S. Food & Drug Admin., Human Foods Program, Microplastics and Nanoplastics in Foods (content current as of July 24, 2024), https://www.fda.gov/food/environmental-contaminants-food/microplastics-and-nanoplastics-foods (last visited Nov. 2, 2026) [*hereinafter* FDA Microplastics Web Page] (describing microplastics as "typically considered less than five millimeters in size in at least one dimension" and nanoplastics as "typically considered to be less than one μm").
[2] 21 U.S.C. § 348(a); 21 C.F.R. § 109.6(a). Petitioners do not concede that a particle that breaks away from packaging or processing equipment and enters food is present "in accordance with" a regulation or notification authorizing the polymer's use in that article. Petitioners' position is that such particles fall within the actions requested here.

1) Validate and publish analytical methods for measuring microplastics in food, beginning with the foods and beverages that contribute most to dietary exposure. For each method, state the foods or classes of foods to which it applies, the particle sizes and polymers it measures, the unit in which it reports concentration, and its limit of quantification, and revise the methods as they improve. Use the methods to measure microplastics across the food supply and publish the results, including the share that comes from packaging and processing materials.[3]
2) As methods become validated for a food or class of foods, establish an action level for microplastics in that food under 21 C.F.R. §§ 109.4(c) and 109.6(d), at a level at which FDA "may regard the food as adulterated within the meaning of section 402(a)(1) of the act," announced in the Federal Register with an invitation for public comment as 21 C.F.R. § 109.4(c)(2) requires. Each action level should be set at the lowest level that can be obtained in that food and lowered as methods and manufacturing practices improve.[4]
3) Once validated methods are in place for a food or class of foods, initiate a proceeding to establish a tolerance for microplastics in that food under section 406 of the Act, 21 U.S.C. § 346, and 21 C.F.R. § 109.4(a), limiting the quantity of microplastics "to such extent as [FDA] finds necessary for the protection of public health," at a level FDA sets by taking into account, as section 406 requires, "the extent to which the use of such substance is required or cannot be avoided," and that is no less protective of public health than the action level it replaces, if any.

B. STATEMENT OF GROUNDS

This petition is supported by extensive scientific evidence demonstrating that microplastics contaminate virtually all categories of food consumed in the United States, that Americans are exposed to substantial quantities of microplastics through dietary intake, and that such exposure poses serious risks to human health. Copies of the references cited are submitted with this petition. The discussion below offers scientific evidence to counter two of FDA's stated positions on microplastics: that the evidence does not demonstrate a health risk and that methodological limitations in existing studies justify continued inaction. With regard to FDA's position that the agency can only take regulatory action if it determines that microplastics "adversely affect human health,"[5] this position misstates the statutory standard and is contrary to law.

On health risk, FDA states that "[c]urrent scientific evidence does not demonstrate that levels of microplastics or nanoplastics detected in foods pose a risk to human health."[6] But the scientific literature

[3] 21 C.F.R. § 10.25(a) (a petition may ask the Commissioner "to take or refrain from taking any other form of administrative action"); FDA Microplastics Web Page, *supra* note 1 ("there are no standardized methods for how to detect, quantify, or characterize microplastics and nanoplastics").
[4] 21 C.F.R. §§ 109.4(c), 109.6(d); U.S. Food & Drug Admin., Ctr. for Food Safety & Applied Nutrition, Supporting Document for Recommended Maximum Level for Lead in Candy Likely To Be Consumed Frequently by Small Children (Nov. 2006), Docket No. 2005D-0481, § III, https://www.fda.gov/food/environmental-contaminants-food/supporting-document-recommended-maximum-level-lead-candy-likely-be-consumed-frequently-small (last visited Nov. 9, 2026) [*hereinafter* Lead in Candy Supporting Document] (describing FDA's policy of reducing lead exposure "to the lowest level that can practically be obtained"); U.S. Food & Drug Admin., Closer to Zero: Reducing Childhood Exposure to Heavy Metals from Foods (content current as of Oct. 27, 2026), https://www.fda.gov/food/environmental-contaminants-food/closer-zero-reducing-childhood-exposure-heavy-metals-foods (last visited Nov. 9, 2026) [*hereinafter* Closer to Zero] (action levels are proposed "based on toxicological data and what is achievable by manufacturers").
[5] FDA Microplastics Web Page, *supra* note 1.
[6] *Id.*

now includes peer-reviewed studies of *human* tissue and patients documenting that microplastics are present in human arterial tissue, cardiac tissue, liver, colon, placenta, and blood, and that patients whose carotid artery plaque contained microplastics had a higher rate of heart attack, stroke, or death from any cause. The Minderoo-Monaco Commission, a comprehensive review of the global plastics and human health literature prepared by 48 authors, concluded that microplastic exposure is suspected to be a digestive hazard to humans, and that most evaluation of health effects currently rests on animal and laboratory studies because methods for quantifying human exposure remain at an early stage of development.[7]

A majority of the members of the Children's Health Protection Advisory Committee of the U.S. Environmental Protection Agency (EPA), in a November 2025 letter to the agency's administrator, identified specific plastic-associated chemicals as associated with harm to children's neurodevelopment and reproductive development, and with cardiovascular disease.[8] A 2024 systematic review applied the Navigation Guide method, a structured procedure for rating the quality and strength of a body of evidence that the World Health Organization has used in its own burden-of-disease work, to three human observational studies and twenty-eight animal studies. It concluded that microplastic exposure is "suspected" to harm human digestive, reproductive, and respiratory health, rating the evidence on sperm quality and on suppression of digestive immune function as high quality and the remaining outcomes as moderate.[9] The majority of EPA's Children's Health Protection Advisory Committee adopted that conclusion in its November 2025 letter, writing that current evidence indicates that microplastics are "'suspected' to harm human reproductive, digestive, and respiratory health, with a suggested link to colon and lung cancer."[10] FDA's parent department, the Department of Health and Human Services, reached a parallel practical judgment in April 2026, when its Advanced Research Projects Agency for Health committed $144 million to measuring microplastics in the human body and identifying which polymers cause harm, on the stated premise that plastic from food, air, and water is accumulating in people and that this is a growing threat to health.[11] The claim that the evidence "does not demonstrate" a risk shows that the agency has failed to engage with the current evidence.

FDA also rests its refusal to act on its statement that "many of the scientific studies have used methods of variable, questionable, and/or limited accuracy and specificity."[12] But the methodological limitations described in FDA's own 2024 paper, which it cites in support, are a reason for more regulatory

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[7] Philip J. Landrigan, Hervé Raps, Maureen Cropper et al., The Minderoo-Monaco Commission on Plastics and Human Health, 89 Annals of Global Health art. 23 (2023), https://doi.org/10.5334/aogh.4056, at 89 [*hereinafter* Commission Report].

[8] Children's Health Protection Advisory Committee, Letter A to Administrator Lee Zeldin, Plastics and Children's Health Charge Response (Nov. 4, 2025), U.S. EPA Docket No. EPA-HQ-OA-2025-0261-0029, at 1, 4–5 [*hereinafter* CHPAC Letter A]. Letter A is the response of 23 of the Committee's 25 members; one member submitted a separate letter, Letter B, in the same document. Letter A states that the bisphenol A outcomes it lists are drawn from systematic reviews and meta-analyses of moderate quality, except for polycystic ovary syndrome, which rests on a high-quality review, and that the phthalate outcomes are likewise drawn from reviews of moderate quality except for asthma. *Id.* at 4–5.

[9] Nicholas Chartres, Courtney B. Cooper, Garret Bland et al., Effects of Microplastic Exposure on Human Digestive, Reproductive, and Respiratory Health: A Rapid Systematic Review, 58 Environmental Science & Technology 22843–22864 (2024), https://doi.org/10.1021/acs.est.3c09524, at 22843, 22848, 22858.

[10] CHPAC Letter A, *supra* note 8, at 6.

[11] U.S. Department of Health and Human Services, ARPA-H launches groundbreaking, $144 million program to combat toxic microplastics in the human body (Apr. 2, 2026), https://www.hhs.gov/press-room/arpa-h-launches-groundbreaking-144-million-program-combat-toxic-microplastics-human-body.html (last visited Nov. 4, 2026).

[12] FDA Microplastics Web Page, *supra* note 1.

intervention, not less.[13] Many of the analytical shortcomings the paper identifies cause analysts to miss particles that are actually present. For example, visual methods cannot detect particles below one millimeter, certain spectroscopic methods cannot resolve particles below ten micrometers, the chemical digestion used to clear away food destroys certain polymers, and density separation, the step that floats plastic off from heavier material, recovers fewer small particles.[14] Because these limitations fall hardest on the smallest particles, reported levels likely understate exposure to them, even though some methods can also overcount by misidentifying particles.[15]

Finally, FDA's legal framing presents the most fundamental problem. FDA states that it "can take regulatory action" if it "determines, based on scientific evidence, that microplastics or nanoplastics in food, including packaged food and beverages, adversely affect human health." That is not the statutory standard. Section 402(a)(1) of the Act deems food adulterated when it bears or contains a substance that "may render" it "injurious to health."[16] The Supreme Court, construing the "may render" language, held that it reaches food containing an added poisonous or deleterious ingredient that "may possibly injure the health" of a consumer, and that the government need not show that the food has affected the public health.[17] FDA's stated "adversely affect" standard is an impermissibly high bar and is not supported by the statute. The evidence in this petition, including studies of human tissue and patients, satisfies Congress's standard that a substance "may render" food "injurious to health," and that showing authorizes the action Petitioners request. FDA need not wait for conclusive proof of harm before acting.

C. ENVIRONMENTAL IMPACT

The requested action levels and tolerances are categorically excluded from the requirement to prepare an Environmental Assessment or an Environmental Impact Statement under the National Environmental Policy Act pursuant to 21 C.F.R. § 25.32(m), which covers an "[a]ction to prohibit or otherwise restrict or reduce the use of a substance in food, food packaging, or cosmetics." They meet that criterion because action levels and tolerances for microplastics would restrict or reduce the presence of microplastics in

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[13] Timothy V. Duncan, Sadia Afrin Khan, Anil K. Patri & Stacey Wiggins, Regulatory Science Perspective on the Analysis of Microplastics and Nanoplastics in Human Food, 96 Analytical Chemistry 4343–4358 (2024), https://doi.org/10.1021/acs.analchem.3c05408, at 4349.

[14] Timothy V. Duncan, Sadia Afrin Khan, Anil K. Patri & Stacey Wiggins, Supporting Information to: Regulatory Science Perspective on the Analysis of Microplastics and Nanoplastics in Human Food, 96 Analytical Chemistry 4343–4358 (2024), https://doi.org/10.1021/acs.analchem.3c05408, at S-2, S-4 to S-5, S-7, S-9.

[15] Agency assertions regarding a lack of a validated methodology should not preclude all regulatory action. EPA, which claims it lacks a methodology to monitor for microplastics in drinking water (even though methodologies are available and approved at the state level, see, e.g., California State Water Resources Control Board, Resolution No. 2022-0032, Adopting a Policy Handbook Establishing a Standard Method of Testing and Reporting of Microplastics in Drinking Water (Sept. 7, 2022), https://waterboards.ca.gov/drinking_water/certlic/drinkingwater/documents/microplastics/rs2022-0032.pdf, at 2–3), acknowledged concern about microplastics and proposed to add them to its Sixth Contaminant Candidate List. EPA characterized the proposed listing as "a first step toward defining and better understanding potential public health risk from exposure via drinking water." Drinking Water Contaminant Candidate List 6—Draft, 91 Fed. Reg. 17,186, 17,193 (Apr. 6, 2026) (to be codified at 40 C.F.R. pt. 141). In that instance, a sister agency is at least proposing to take some regulatory action despite its purported limitations, although it must do more to meet its legal obligations and address public concern. See, e.g., FWW et al. Comments on CCL 6, Docket No. EPA-HQ-OW-2022-0946; FWW et al. Comments on UCMR 6, Docket No. EPA-HQ-OW-2023-0469.

[16] FDA Microplastics Web Page, *supra* note 1; 21 U.S.C. § 342(a)(1).

[17] *United States v. Lexington Mill & Elevator Co.*, 232 U.S. 399, 411 (1914).

food. The requests to validate and publish methods and to survey the food supply are studies that would not introduce any substance into the environment. To Petitioners' knowledge, no extraordinary circumstances exist that would require an environmental assessment.[18] To the extent environmental review is required, the requested actions aim to reduce microplastic contamination of food, and Petitioners are not aware of any adverse environmental effect they would have.

D. ECONOMIC IMPACT

While implementation of microplastic regulations may impose costs on food manufacturers and the packaging industry, these costs must be weighed against the public health benefits of reduced ingestion of microplastics, as well as against the other environmental and health costs currently imposed by plastic production and pollution. FDA's regulatory impact analysis should account for:

1. Healthcare costs associated with plastic production and with microplastic-related health effects, including effects of chemicals used in plastics;[19]
2. Lost productivity from illness and disability attributable to plastic production and chemicals used in plastics;[20] and
3. Environmental impacts of plastic production and pollution and cleanup costs for plastic contamination.[21]

E. CERTIFICATION

The undersigned certifies, that, to the best knowledge and belief of the undersigned, this petition includes all information and views on which the petition relies, and that it includes representative data and information known to the Petitioners which are unfavorable to the petition.

[18] 21 C.F.R. §§ 25.15(a), (d), 25.20(j), 25.21, 25.32(m).

[19] One study found $249 billion in disease burden in the United States in 2018 alone, representing 1.22% of GDP, solely from chemicals used in plastics. The study authors note that this is an underestimate, because of the limited subset of chemicals and diseases covered. Leonardo Trasande, Roopa Krithivasan, Kevin Park, Vladislav Obsekov & Michael Belliveau, Chemicals Used in Plastic Materials: An Estimate of the Attributable Disease Burden and Costs in the United States, 8 Journal of the Endocrine Society art. bvad163 (2024), https://doi.org/10.1210/jendso/bvad163, at 1, 7.

[20] *Id.*

[21] The present value of global economic damage from marine plastic litter by 2050 is estimated to be 434 billion dollars. Alistair McIlgorm, Karen Raubenheimer, Daniel E. McIlgorm & Rachel Nichols, The cost of marine litter damage to the global marine economy: Insights from the Asia-Pacific into prevention and the cost of inaction, 174 Marine Pollution Bulletin art. 113167 (2022), https://doi.org/10.1016/j.marpolbul.2021.113167, at 1.

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Executive Summary

Microplastics are plastic particles smaller than five millimeters in at least one dimension, and they now contaminate virtually every type of food and beverage Americans consume. Scientific investigations have detected them in meat, seafood, table salt, bottled water, honey, beer, fruits, vegetables, and processed foods. More significantly, researchers have found these particles inside the human body itself, accumulating in blood, the colon, the liver, the placenta, breast milk, and more. They have also been detected in the arterial plaques of patients who went on to have heart attacks and strokes, and to die at substantially higher rates than patients whose plaque contained no detectable plastic. Research on the extent of this contamination and its potential consequences for human health has grown substantially in recent years. FDA has so far treated scientific uncertainty as a reason not to act, but the evidence now supports regulatory action under existing federal law, and FDA must act now to protect the American public.

This petition asks FDA to begin limiting microplastics in food. The Act treats food as "adulterated" when it contains an added poisonous or deleterious substance that may render it injurious to health. Any limit depends on being able to measure microplastics in food, and FDA has said there are no standardized methods for doing so. Petitioners therefore ask FDA first to validate and publish testing methods and to measure how much microplastic is in the food supply. An action level is a non-binding level at which FDA may regard food as adulterated. As methods become available for each food, Petitioners ask FDA to set action levels at the lowest level that can be obtained and to lower them over time, consistent with FDA's stated policy for lead and its Closer to Zero approach of lowering levels over time. FDA should then adopt a formal tolerance, no less protective of public health than the action level it replaces, under section 406 of the Act, the provision Congress wrote for contaminants that food producers cannot avoid.

The scientific record supporting this petition documents contamination that is systemic rather than episodic, arising through multiple pathways that no food producer can eliminate through good manufacturing practice. Agricultural soils receive microplastics from plastic-coated seeds, controlled-release fertilizer, sewage sludge spread as fertilizer, pesticides, and road runoff, introducing particles at the base of the food chain. Seafood and farmed fish accumulate plastic particles through ingestion across the marine food chain. Plastic packaging can release particles into food and beverages during ordinary use and can release chemicals into food, especially when heated. Industrial processing equipment made of plastic can add further contamination during manufacturing. Because these pathways operate at massive scale and are embedded in the infrastructure of American food production, no individual farmer or facility can stop the contamination, and controlling it requires regulatory action at the national level.

Peer-reviewed studies in human cells and laboratory animals have found harmful effects of plastic particles on multiple organ systems, and observational studies of patients have found associations between plastic in the body and disease. Studies in human cells and laboratory animals show that microplastics can cross biological barriers, accumulate in organs and tissues, trigger inflammation, impair liver function, alter the composition of the gut microbiome, and interfere with cardiovascular function, and that chemicals in plastics can disrupt hormonal signaling. A 2024 observational study published in the New England Journal of Medicine followed 257 patients after surgery to remove carotid artery plaque. Those whose plaque contained plastic particles had a heart attack or stroke, or died from any cause, at nearly three times the rate of those whose plaque did not (6.1 versus 2.2 events per 100 patient-years) over an average of 34 months.

Children face outsized danger from this plastic contamination. Their bodies are still developing, and their endocrine systems are particularly sensitive to chemical disruption. Microplastics have been detected in

placental tissue and in the first feces of newborns, establishing that exposure begins before birth and continues through breastfeeding. In November 2025, a majority of the members of EPA's Children's Health Protection Advisory Committee reported that plastic-associated chemicals are associated with harm to children's neurological and reproductive development, and with cardiovascular disease. The protection of children from contamination that begins in utero and persists throughout development is among the most compelling public health justifications for the regulatory action requested in this petition.

FDA has clear statutory authority to act. The adulteration standard under section 402(a)(1) of the Act requires only that a substance may render food injurious to health, a threshold the evidence now satisfies. FDA's regulations provide action levels for unavoidable contaminants, and section 406 provides the tolerance that should follow. Petitioners respectfully urge the agency to begin that work now, while research on the severity of the risk continues.

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STATEMENT OF GROUNDS (CONT'D)[22]

I. Plastic in the American Food System

Microplastics are plastic particles smaller than five millimeters in at least one dimension, and microplastic contamination is now documented in virtually every category of food and beverage consumed by Americans. Researchers have detected plastic particles in seafood, meat, dairy products, fruits, vegetables, grains, honey, salt, bottled water, and beer, among other foods and beverages. The contamination is not confined to processed foods or specific brands. It appears in crops, in wild-caught and farmed seafood, and in bottled and tap water. No diet, whether plant-based, pescatarian, or omnivorous, avoids exposure to plastic particles through food.

The contamination occurs as a systemic consequence of choices made across decades about how to grow, process, package, and distribute food at an industrial scale. The following sections examine the major routes through which microplastics contaminate American food products, document the breadth of contamination across food categories, and establish that exposure through dietary pathways has become both unavoidable and substantial under current production and regulatory systems.

A. Exponential Growth of Plastic Production

Global production of plastic resins and fibers increased from 2 million metric tons in 1950 to 380 million metric tons in 2015, an average growth rate of 8.4 percent a year.[23] This growth rate is roughly 2.5 times the compound annual growth rate of global gross domestic product during the same period.[24] By 2019, global plastics production had reached 460 million metric tons, and current projections indicate plastic use could reach 1,231 million metric tons by 2060 under baseline scenarios.[25] The cumulative total of all plastics ever manufactured from 1950 through 2020 now exceeds 10 billion metric tons.[26] Half of all plastics ever produced were manufactured in just the fourteen years preceding 2020.[27] Growth in global virgin plastic production has consistently exceeded global population growth: between 1950 and 2020, global consumption rose steadily, following a linear trend, from 1 to 60 kilograms per person.[28] Six polymer types account for 83 percent of global resin production: polyethylene, polypropylene, polystyrene, polyvinyl chloride (PVC), polyethylene terephthalate (PET), and polyurethane.[29]

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[22] This petition documents the contamination of the American food supply with microplastic and the health harms that contamination causes. It is not, and does not purport to be, a comprehensive survey of the scientific literature. The peer-reviewed evidence on microplastic contamination is expanding rapidly across dozens of disciplines, and any single document prepared will necessarily capture only a portion of what is known. Petitioners have endeavored instead to present a representative and legally sufficient record that demonstrates the breadth of contamination pathways, the range of food categories affected, and the seriousness of the documented health consequences.

[23] Roland Geyer, Jenna R. Jambeck & Kara Lavender Law, Production, use, and fate of all plastics ever made, 3 Science Advances art. e1700782 (2017), https://doi.org/10.1126/sciadv.1700782, at 1.

[24] *Id.* at 1.

[25] OECD, Global Plastics Outlook: Policy Scenarios to 2060 (OECD Publishing 2022), https://doi.org/10.1787/aa1edf33-en, at 22, 26, 62.

[26] Chao Liu, Roland Geyer & Shanying Hu, 100 years of plastic -- using the past to guide the future (arXiv preprint 2411.13618, 2024), https://doi.org/10.48550/arXiv.2411.13618, at 2, 5.

[27] *Id.* at 2.

[28] *Id.* at 3.

[29] *Id.*

In 2020, the mass of all plastic ever produced was about twice the combined mass of all land and marine animals.[30] Packaging is the largest single use of plastics, accounting for 29 percent of global virgin plastic consumption in 2020, and its growth was accelerated by a global shift from reusable to single-use containers.[31] In the United States, plastics rose from less than 1 percent of municipal solid waste by mass in 1960 to 18.5 percent of the waste that went to landfill in 2018.[32]

The permanence of plastic materials compounds the environmental and health consequences of this production trajectory. The vast majority of monomers used to manufacture plastics, including ethylene and propylene, are derived from fossil hydrocarbons.[33] None of the plastics commonly used today are biodegradable, meaning they accumulate in landfills and natural environments instead of decomposing.[34] The only methods to permanently eliminate plastic waste are destructive treatments such as combustion or pyrolysis, which produce their own harms.[35] By 2020, global plastic waste had cumulatively reached 8 gigatons, more than half of which came from the last 13 years.[36] Over the last 70 years, the world has recycled approximately 13 percent and incinerated 16 percent of its cumulative plastic waste, with the remaining 71 percent either landfilled or mismanaged.[37] One study estimated that the United States recycled only 6.2 percent of its end-of-life plastics in 2015.[38] If current production and waste management trends continue without intervention, roughly 12 billion metric tons of plastic waste will accumulate in landfills or natural environments by 2050.[39]

These production trends establish the context for understanding microplastic contamination of the American food supply. The materials that fragment into microplastics now permeating food and beverages represent the physical manifestation of seven decades of unconstrained plastic production growth. Polyethylene and polypropylene, the two most-produced polymers, are also among those most often detected in seafood, in the marine environment, and in human tissue, as discussed in Sections I.B.2 and II. The first 70 years of plastic mass production have shown remarkably stable and persistent growth in virgin plastic production and consumption, with waste management systems across the world unable to

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[30] Emily Elhacham, Liad Ben-Uri, Jonathan Grozovski, Yinon M. Bar-On & Ron Milo, Global human-made mass exceeds all living biomass, 588 Nature 442–444 (2020), https://doi.org/10.1038/s41586-020-3010-5, at 442–444.

[31] Liu et al., *supra* note 26, at 3; Geyer et al., *supra* note 23, at 1.

[32] Jenna R. Jambeck, Roland Geyer, Chris Wilcox et al., Plastic waste inputs from land into the ocean, 347 Science 768–771 (2015), https://doi.org/10.1126/science.1260352, at 768; U.S. Environmental Protection Agency, Plastics: Material-Specific Data (last updated Mar. 13, 2026), https://www.epa.gov/facts-and-figures-about-materials-waste-and-recycling/plastics-material-specific-data (last visited Nov. 4, 2026).

[33] Geyer et al., *supra* note 23, at 1.

[34] *Id.* at 1; David K. A. Barnes, Francois Galgani, Richard C. Thompson & Morton Barlaz, Accumulation and fragmentation of plastic debris in global environments, 364 Philosophical Transactions of the Royal Society B: Biological Sciences 1985–1998 (2009), https://doi.org/10.1098/rstb.2008.0205, at 1985.

[35] Geyer et al., *supra* note 23, at 1.

[36] Liu et al., *supra* note 26, at 4.

[37] *Id.*

[38] Jinghan Di, Barbara K. Reck, Alessio Miatto & Thomas E. Graedel, United States plastics: Large flows, short lifetimes, and negligible recycling, 167 Resources, Conservation and Recycling art. 105440 (2021), https://doi.org/10.1016/j.resconrec.2021.105440.

[39] Geyer et al., *supra* note 23, at 1, 3–4. The ocean is the largest natural sink for plastics. One study estimated that about 30 million metric tons of plastic had accumulated in the ocean by 2019. OECD, *supra* note 25, at 22, 125.

keep pace with the associated increase in plastic waste generation.[40] Given the scale and acceleration of plastic production, contamination of food systems is likely to intensify.

B. Routes of Contamination

Microplastics reach the American diet through several distinct pathways at different stages of food production and distribution. The full extent of microplastic contamination throughout the food value chain is still largely unknown, despite some analysis of widely used plastic polymers in food systems. The following subsections examine four major contamination routes: agricultural sources including soil contamination and plastic mulching practices; aquaculture and fisheries operations; industrial food processing facilities where machinery and worker contact introduce synthetic particles into products; and food packaging materials that shed particles and leach chemicals.

1. Agricultural Sources

Microplastic contamination of agricultural soils represents a direct pathway through which plastic particles enter the American food supply, affecting crops consumed by millions of people and livestock daily. Microplastics have been documented in soils, which may be an underestimated sink for them that could significantly affect human health and the economy through impacts on agriculture and land use.[41] Experiments show that crop plants can take up the smallest particles through their roots and move them into stems, leaves, flowers, and fruits.[42] Particles carried in the air also settle on soil and the surfaces of leaves.[43] Plastic particles move through soil via both biological and mechanical means. Soil-dwelling organisms including earthworms affect and facilitate the movement of plastic particles down through the soil profile, and this process depends on the size of the particles.[44] Farming operations including ploughing and harvesting also play an important role in moving these polymers through the soil and incorporating them into the soil matrix.[45]

Organic fertilizers constitute a significant route for microplastics to gain access to agricultural ecosystems. One such fertilizer is sewage sludge, the solid material left after wastewater treatment. Because sludge is cheap, farmers in many developed countries spread it on their fields, and in Europe and North America about half of all sewage sludge goes to agricultural use.[46] Municipal wastewater receives

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[40] Liu et al., *supra* note 26, at 6.
[41] E. Garrido Gamarro & V. Costanzo, Microplastics in food commodities – A food safety review on human exposure through dietary sources, Food Safety and Quality Series No. 18 (Food and Agriculture Organization of the United Nations 2022), https://doi.org/10.4060/cc2392en, at 17.
[42] Bhakti Jadhav & Agnieszka Medyńska-Juraszek, Microplastic and Nanoplastic in Crops: Possible Adverse Effects to Crop Production and Contaminant Transfer in the Food Chain, 13 Plants art. 2526 (2024), https://doi.org/10.3390/plants13172526, at 3; Gilda Carrasco Silva, Felipe M. Galleguillos Madrid, Diógenes Hernández et al., Microplastics and Their Effect in Horticultural Crops: Food Safety and Plant Stress, 11 Agronomy art. 1528 (2021), https://doi.org/10.3390/agronomy11081528, at 5; Garrido Gamarro & Costanzo, *supra* note 41, at 18.
[43] Jadhav & Medyńska-Juraszek, *supra* note 42, at 3; Garrido Gamarro & Costanzo, *supra* note 41, at 18.
[44] Matthias C. Rillig, Rosolino Ingraffia & Anderson A. de Souza Machado, Microplastic Incorporation into Soil in Agroecosystems, 8 Frontiers in Plant Science art. 1805 (2017), https://doi.org/10.3389/fpls.2017.01805, at 2; Garrido Gamarro & Costanzo, *supra* note 41, at 18.
[45] Rillig et al., *supra* note 44, at 2–3.
[46] Luca Nizzetto, Martyn Futter & Sindre Langaas, Are Agricultural Soils Dumps for Microplastics of Urban Origin?, 50 Environmental Science & Technology 10777–10779 (2016), https://doi.org/10.1021/acs.est.6b04140, at 10777.

microplastic particles from a range of everyday household sources, including laundry and the rinsing of personal care products, as well as stormwater runoff that carries plastic debris into combined sewer systems.[47] Research has found that more than 80 percent of plastic particles entering wastewater treatment facilities are concentrated in the resulting sludge.[48] One estimate puts the amount added to European farm soils through sludge and biosolids at 125 to 850 tons of microplastics a year for every million inhabitants.[49] This constitutes a major and continuing contamination source in agricultural lands where processed sewage sludge is utilized.

A farm's location relative to roads and towns can affect how much plastic builds up in its soil, in part because plastic particles from cars make their way from roadways into soils. A study of vegetable farms found that fields next to suburban roads held about 1.8 times as many microplastic particles as fields in residential areas.[50] The authors identified the likely sources as traffic, including particles worn from tires, household waste and sewage, organic fertilizer, and the plastic nets and bags used in farming.[51] This proximity effect indicates that urbanization patterns and agricultural planning choices have direct impacts on the plastic contamination load in food production areas.

Plastic materials used directly in farming are another major source of contamination. Plastic-coated seeds, controlled-release fertilizer, and pesticides are all sources of microplastics in soils.[52] Use of polymer-coated fertilizers has increased in large-scale commodity farming, especially in the United States, Canada, and China.[53] Plastic mulch, a plastic film laid over the soil, is widely used to shield seedlings and crops from environmental stress.[54] Its use results in plastic buildup in agricultural soils, particularly of high-density polyethylene (HDPE), linear low-density polyethylene (LLDPE), and low-density polyethylene (LDPE).[55] These materials degrade over time into smaller particles that remain in the soil environment.

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[47] Guadalupe Vianey Landeros Gonzalez, Gabriela Dominguez Cortinas, Malcolm Hudson, Peter Shaw & Ian D. Williams, A Review of the Origins of Microplastics Arriving at Wastewater Treatment Plants, 20 Detritus 41–55 (2022), https://doi.org/10.31025/2611-4135/2022.15224, at 41–43.

[48] Gaetano Di Bella, Santo Fabio Corsino, Federica De Marines et al., Occurrence of Microplastics in Waste Sludge of Wastewater Treatment Plants: Comparison between Membrane Bioreactor (MBR) and Conventional Activated Sludge (CAS) Technologies, 12 Membranes art. 371 (2022), https://doi.org/10.3390/membranes12040371, at 2.

[49] Nizzetto et al., *supra* note 46, at 10777.

[50] Yuling Chen, Yifei Leng, Xiaoning Liu & Jun Wang, Microplastic pollution in vegetable farmlands of suburb Wuhan, central China, 257 Environmental Pollution art. 113449 (2020), https://doi.org/10.1016/j.envpol.2019.113449, at 1, 4.

[51] *Id.* at 3, 5–6.

[52] Samuel J. Cusworth, William J. Davies, Martin R. McAinsh, Andrew S. Gregory, Jonathan Storkey & Carly J. Stevens, Agricultural fertilisers contribute substantially to microplastic concentrations in UK soils, 5 Communications Earth & Environment art. 7 (2024), https://doi.org/10.1038/s43247-023-01172-y, at 2; Center for International Environmental Law, Sowing a Plastic Planet: How Microplastics in Agrochemicals Are Affecting Our Soils, Our Food, and Our Future (Giulia Carlini & Dana Drugmand, 2022; reissued Dec. 2022), at 1, 5, https://resolutions.unep.org/resolutions/uploads/sowing_a_plastic_planet_-_how_microplastics_in_agrochemicals_are_affecting_our_soils_our_food_and_our_future_ciel.pdf_0.pdf (last visited Nov. 9, 2026).

[53] Center for International Environmental Law, *supra* note 52, at 6, 10.

[54] Zacharias Steinmetz, Claudia Wollmann, Miriam Schaefer et al., Plastic mulching in agriculture. Trading short-term agronomic benefits for long-term soil degradation?, 550 Science of the Total Environment 690–705 (2016), https://doi.org/10.1016/j.scitotenv.2016.01.153, at 691.

[55] *Id.* at 690, 692, 694.

These pathways run through practices common on American farms, including sludge application and polymer-coated fertilizer, and they put microplastics into the soils where food crops grow.

2. Aquaculture and Fisheries

Aquaculture and commercial fisheries supply a substantial portion of seafood consumed in the United States, and plastic contamination in these products establishes a direct exposure pathway for American consumers. Plastic materials are widely used in the aquaculture industry and in fisheries.[56] Buoys, nets, ropes, and other types of gear are made of synthetic materials.[57] The makeup of plastic polymers detected in marine organisms generally mirrors the types of plastic employed in aquaculture and fishing gear, whether to cultivate or harvest these organisms.[58] Scientists have detected elevated concentrations of microplastics in farmed mussels relative to wild populations.[59] Polypropylene and expanded polystyrene have been found in cultured mussels, and their presence most probably stems from the use of these polymers to manufacture ropes for the line culture of mussels and buoys.[60] Polyamide (nylon) and polyester are used extensively in fisheries.[61]

The quantity of microplastics present in fish has been seen to increase with the size of the animal, its proximity to urban areas, and the concentration of plastic debris in seawater and sediment.[62] This pattern indicates that both environmental and biological factors shape the degree of contamination in marine species. Studies have examined the presence and quantity of microplastics in fish from around the world, with numerous papers concentrating on the contamination of commercial marine and freshwater species.[63] The primary polymer types regularly detected in the marine environment are polyethylene and polypropylene, followed by polystyrene, polyamide, and PVC.[64]

Research indicates that the most prevalent polymer types detected in shellfish are polyethylenes (including LDPE and HDPE), followed by PET, polypropylene, polyamide, and polystyrene.[65] In fish, the most frequent polymers are polyethylene, polypropylene, polyamide, PET, and polystyrene in descending order, while polyethylene and polypropylene appear to dominate in fisheries and aquaculture products

[56] Garrido Gamarro & Costanzo, *supra* note 41, at 33.
[57] *Id.* at 33.
[58] *Id.*
[59] Jiana Li, Dongqi Yang, Lan Li, Khalida Jabeen & Huahong Shi, Microplastics in commercial bivalves from China, 207 Environmental Pollution 190–195 (2015), https://doi.org/10.1016/j.envpol.2015.09.018, at 194 (reporting an earlier study); Garrido Gamarro & Costanzo, *supra* note 41, at 33.
[60] Garrido Gamarro & Costanzo, *supra* note 41, at 33.
[61] *Id.*
[62] Razegheh Akhbarizadeh, Farid Moore & Behnam Keshavarzi, Investigating a probable relationship between microplastics and potentially toxic elements in fish muscles from northeast of Persian Gulf, 232 Environmental Pollution 154–163 (2018), https://doi.org/10.1016/j.envpol.2017.09.028, at 154; Christiana M. Boerger, Gwendolyn L. Lattin, Shelly L. Moore & Charles J. Moore, Plastic ingestion by planktivorous fishes in the North Pacific Central Gyre, 60 Marine Pollution Bulletin 2275–2278 (2010), https://doi.org/10.1016/j.marpolbul.2010.08.007, at 2276; Olgaç Güven, Kerem Gökdağ, Boris Jovanović & Ahmet Erkan Kıdeyş, Microplastic litter composition of the Turkish territorial waters of the Mediterranean Sea, and its occurrence in the gastrointestinal tract of fish, 223 Environmental Pollution 286–294 (2017), https://doi.org/10.1016/j.envpol.2017.01.025, at 286; Garrido Gamarro & Costanzo, *supra* note 41, at 33.
[63] Garrido Gamarro & Costanzo, *supra* note 41, at 33.
[64] *Id.*
[65] *Id.* at 34.

overall.[66] Their pervasive presence may be attributed to their extensive use and production, which amounts to 36 percent and 21 percent of global non-fiber plastic production for polyethylene and polypropylene, respectively.[67]

Most microplastics in fish sit in the gut, which is usually removed before eating, but particles have also been found in the edible muscle of fish, squid, crab, and prawns.[68] These findings indicate that treating the digestive tract as the sole reservoir of plastic could result in an underestimation of the actual amount that may be consumed. For canned fishery products, contamination may arise from inadequate organ removal or from processing.[69]

The contamination of fisheries products may be linked to the use of contaminated feed in aquaculture. Fishmeal, which is made from raw fish, has frequently been reported to contain synthetic particles retained in the fish's digestive tracts,[70] giving microplastics a pathway to farmed fish and other animals that are fed fishmeal.

3. Industrial Food Processing

Food processing facilities add microplastic contamination through machinery, equipment, and worker contact during the manufacturing stages that convert raw agricultural products into the packaged goods sold in American supermarkets. The employment of machinery and technologies made of, or incorporating, plastic materials may result in the transfer of parts of the polymers to the foods they contact.[71] For instance, a study of milk found that the synthetic particles in it were thermoplastic sulfone polymers, materials widely used in the filtration membranes of dairy processing, and the authors concluded that the particles likely came from abrasion of those membranes.[72] Equipment designed to filter or purify food can thus add contamination to the final product. In studies of mineral water, substances used in the production process were identified as one likely cause of the presence of xenoestrogens, chemicals that mimic the hormone estrogen.[73]

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[66] *Id.*
[67] Geyer et al., *supra* note 23, at 1; Garrido Gamarro & Costanzo, *supra* note 41, at 34.
[68] Akhbarizadeh et al., *supra* note 62, at 154; Luís Gabriel A. Barboza, Clara Lopes, Patrícia Oliveira et al., Microplastics in wild fish from North East Atlantic Ocean and its potential for causing neurotoxic effects, lipid oxidative damage, and human health risks associated with ingestion exposure, 717 Science of the Total Environment art. 134625 (2020), https://doi.org/10.1016/j.scitotenv.2019.134625, at 1; Damaris Benny Daniel, P. Muhamed Ashraf, Saly N. Thomas & K.T. Thomson, Microplastics in the edible tissues of shellfishes sold for human consumption, 264 Chemosphere art. 128554 (2021), https://doi.org/10.1016/j.chemosphere.2020.128554, at 1; Ali Karami, Abolfazl Golieskardi, Yu Bin Ho, Vincent Larat & Babak Salamatinia, Microplastics in eviscerated flesh and excised organs of dried fish, 7 Scientific Reports art. 5473 (2017), https://doi.org/10.1038/s41598-017-05828-6, at 1; Garrido Gamarro & Costanzo, *supra* note 41, at 33–34.
[69] Garrido Gamarro & Costanzo, *supra* note 41, at 34.
[70] *Id.* at 17.
[71] *Id.* at 19.
[72] Gurusamy Kutralam-Muniasamy, Fermín Pérez-Guevara, I. Elizalde-Martínez & V.C. Shruti, Branded milks – Are they immune from microplastics contamination?, 714 Science of the Total Environment art. 136823 (2020), https://doi.org/10.1016/j.scitotenv.2020.136823, at 1, 7–8.
[73] Martin Wagner & Jörg Oehlmann, Endocrine disruptors in bottled mineral water: total estrogenic burden and migration from plastic bottles, 16 Environmental Science and Pollution Research 278–286 (2009), https://doi.org/10.1007/s11356-009-0107-7, at 282–283.

One study of sea salt suggested that the polypropylene fibers it found may have come from clothing worn by workers during processing, or from wastewater pollution of the seawater used to make the salt.[74] Synthetic clothing worn in processing plants is therefore one plausible route by which fibers reach finished food. Similarly, a distinct drop in phthalate concentration was documented in Japan after a ban on the use of PVC gloves containing di(2-ethylhexyl) phthalate (DEHP) during production and cooking processes.[75] This Japanese experience offers direct evidence that worker protective equipment and food-contact materials employed during processing contribute measurably to the chemical contamination of finished food products, and that regulatory interventions targeting these sources can decrease consumer exposure. Section IV.B.2 describes how widely plastic is used in food processing and packaging.

4. Food Packaging

Plastic packaging constitutes a ubiquitous source of microplastic contamination in the American food supply, with nearly every packaged food product potentially adding to consumer exposure. Roughly 10 to 20 percent of all plastics produced are used for food and beverage packaging.[76] The primary polymers used in this sector include polyethylene (particularly LDPE), polystyrene, polypropylene, PET, polycarbonates, polyamide, and polyurethane used in adhesives, along with PVC.[77] Additional polymers include polytetrafluoroethylene (Teflon), polyvinylidene chloride (employed as a barrier layer), and ethylene copolymers.[78]

Studies of bottled water attribute at least part of the synthetic particles in it to the packaging, because the polymers identified are typically those that make up the bottle caps (including polypropylene and polyethylene), labels, carton linings, and the bottles themselves (including PET).[79] Ordinary use of

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[74] Monia Renzi & Andrea Blašković, Litter & microplastics features in table salts from marine origin: Italian versus Croatian brands, 135 Marine Pollution Bulletin 62–68 (2018), https://doi.org/10.1016/j.marpolbul.2018.06.065.

[75] Yukari Tsumura, Susumu Ishimitsu, Akiko Kaihara, Kimihiko Yoshii, Yumiko Nakamura & Yasuhide Tonogai, Di(2-ethylhexyl) phthalate contamination of retail packed lunches caused by PVC gloves used in the preparation of foods, 18 Food Additives and Contaminants 569–579 (2001), https://doi.org/10.1080/02652030120071, at 569, 578–579 (gloves as source; Japanese prohibition of DEHP-containing PVC gloves from June 14, 2000); Garrido Gamarro & Costanzo, *supra* note 41, at 19 (reporting the decrease after the ban).

[76] Joe Yates, Megan Deeney, Jane Muncke et al., Plastics matter in the food system, 6 Communications Earth & Environment art. 176 (2025), https://doi.org/10.1038/s43247-025-02105-7, at 1.

[77] Kanishka Bhunia, Shyam S. Sablani, Juming Tang & Barbara Rasco, Migration of Chemical Compounds from Packaging Polymers during Microwave, Conventional Heat Treatment, and Storage, 12 Comprehensive Reviews in Food Science and Food Safety 523–545 (2013), https://doi.org/10.1111/1541-4337.12028, at 525–528, 530; PlasticsEurope, Plastics – the Facts 2019: An analysis of European plastics production, demand and waste data (2019), at 20, 22–23, https://plasticseurope.org/wp-content/uploads/2021/10/2019-Plastics-the-facts.pdf; Garrido Gamarro & Costanzo, *supra* note 41, at 21.

[78] Bhunia et al., *supra* note 77, at 525–527.

[79] Garrido Gamarro & Costanzo, *supra* note 41, at 21; Sherri A. Mason, Victoria G. Welch & Joseph Neratko, Synthetic Polymer Contamination in Bottled Water, 6 Frontiers in Chemistry art. 407 (2018), https://doi.org/10.3389/fchem.2018.00407, at 1, 9–10; B.E. Oßmann, G. Sarau, H. Holtmannspötter, M. Pischetsrieder, S.H. Christiansen & W. Dicke, Small-sized microplastics and pigmented particles in bottled mineral water, 141 Water Research 307–316 (2018), https://doi.org/10.1016/j.watres.2018.05.027, at 307; Darena Schymanski, Christophe Goldbeck, Hans-Ulrich Humpf & Peter Fürst, Analysis of microplastics in water by micro-Raman spectroscopy: Release of plastic particles from different

packaging also releases particles. A 2025 systematic evidence map of 103 studies concluded that normal use of plastic food contact articles can release microplastics into foods and beverages, and one highly reliable study of bottle closures found that polypropylene particles in mineral water increased significantly with repeated opening of the bottle.[80] In a separate laboratory study, each opening and closing of a disposable plastic water bottle added an average of 553 particles per liter of water.[81] Another review, however, found that most particles produced by abrasion from opening screw caps stayed on the cap or at the cap-neck interface rather than entering the water.[82]

Most polymers employed in packaging materials may experience certain changes resulting from external conditions, and this can result in the release of chemical components and their transfer into food. Polymer-specific alterations take place after exposure to high temperatures, ultraviolet light, and changes in pH.[83] Migration of plastic components depends not only on the quality of the plastic material but also on the characteristics of the food and contact time. One study documented an increase in the migration of certain components of PET bottles into water at 60 degrees Celsius.[84] A temperature-dependent release of heavy metals was also identified in food cooked in polyethylene bags, with a total migration of 7 percent, 16 percent, 8 percent, and 48 percent for lead, cadmium, chromium, and cobalt respectively, yielding food concentrations of up to 121, 12, 9.5, and 15 parts per million after 5 hours of exposure at 95 degrees Celsius, particularly on the surface of food.[85]

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packaging into mineral water, 129 Water Research 154–162 (2018), https://doi.org/10.1016/j.watres.2017.11.011, at 154.

[80] Lisa Zimmermann, Birgit Geueke, Lindsey V. Parkinson, Christoph Schür, Martin Wagner & Jane Muncke, Food contact articles as source of micro- and nanoplastics: a systematic evidence map, 9 npj Science of Food art. 111 (2025), https://doi.org/10.1038/s41538-025-00470-3, at 1, 6–7 & tbl. 1.

[81] Tvisha Singh, Generation of microplastics from the opening and closing of disposable plastic water bottles, 19 Journal of Water and Health 488–498 (2021), https://doi.org/10.2166/wh.2021.025, at 488.

[82] European Food Safety Authority (EFSA), Eric Barthélémy, Ronan Cariou et al., Literature review on micro- and nanoplastic release from food contact materials during their use, EFSA Supporting Publication 2025:EN-9733 (approved Oct. 15, 2025), https://doi.org/10.2903/sp.efsa.2025.EN-9733, at 1, 26 (concluding that actual release from food contact materials is lower than many published studies report).

[83] Bhunia et al., *supra* note 77, at 523, 528; Zahra Pilevar, Akbar Bahrami, Samira Beikzadeh, Hedayat Hosseini & Seid Mahdi Jafari, Migration of styrene monomer from polystyrene packaging materials into foods: Characterization and safety evaluation, 91 Trends in Food Science & Technology 248–261 (2019), https://doi.org/10.1016/j.tifs.2019.07.020, at 248, 254; Garrido Gamarro & Costanzo, *supra* note 41, at 22.

[84] Cristina Bach, Xavier Dauchy, Isabelle Severin, Jean-François Munoz, Serge Etienne & Marie-Christine Chagnon, Effect of temperature on the release of intentionally and non-intentionally added substances from polyethylene terephthalate (PET) bottles into water: Chemical analysis and potential toxicity, 139 Food Chemistry 672–680 (2013), https://doi.org/10.1016/j.foodchem.2013.01.046, at 672, 679 (finding that higher storage temperature increased the release of formaldehyde, acetaldehyde, and antimony into the water, but that extracts of the water stored for 10 days at 60 degrees Celsius showed no toxic, DNA-damaging, or hormone-disrupting activity in laboratory tests).

[85] L. Musoke, N. Banadda, C. Sempala & J. Kigozi, The Migration of Chemical Contaminants from Polyethylene Bags into Food During Cooking, 9 Open Food Science Journal 14–18 (2015), https://doi.org/10.2174/1874256401509010014, at 14, 17.

Migration of diisooctyl phthalate, polyethylene glycol, phthalic anhydride, and stearamide was documented in vacuum-packed beef samples.[86] In a study that added a test chemical to low-density polyethylene film, migration of the chemical into pork and chicken increased with contact time, storage temperature, and fat content.[87]

These packaging-related contamination pathways impact virtually all processed and packaged foods in American commerce, establishing widespread and repeated exposure for consumers across multiple food categories throughout daily life.

C. Contaminated Products

Microplastic contamination through the multiple pathways discussed above represents a pervasive issue across the American food and beverage supply. Plastic particles have been detected in virtually every food category examined by researchers worldwide. The breadth of microplastic contamination across food categories demonstrates that American consumers face unavoidable exposure through normal diets. While estimates of total dietary intake vary substantially across studies due to differences in analytical methods, detection limits, and food consumption patterns examined, the available evidence indicates that tens of thousands to billions of microplastic particles enter the human body annually through food and beverage consumption alone.[88] A 2026 review that compiled particle counts from 193 studies of 13 categories of food and drink, adjusted to a common particle size range, estimated that an average adult ingests a median of about 51,000 particles a day, with fruits, vegetables, and grains contributing the most.[89] The pervasive nature of contamination across all major food and beverage categories examined establishes that microplastic ingestion represents an unavoidable consequence of participation in the modern American food system.

1. Seafood

Marine and freshwater organisms take in microplastics both directly and by eating prey that contain them, making seafood a documented source of human exposure to plastic particles. Research examining

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[86] Tatiane Melina Guerreiro, Diogo Noin de Oliveira, Carlos Fernando Odir Rodrigues Melo, Estela de Oliveira Lima & Rodrigo Ramos Catharino, Migration from plastic packaging into meat, 109 Food Research International 320–324 (2018), https://doi.org/10.1016/j.foodres.2018.04.026, at 320, 322–323.
[87] A. Sanches Silva, J.M. Cruz, R. Sendón García, R. Franz & P. Paseiro Losada, Kinetic migration studies from packaging films into meat products, 77 Meat Science 238–245 (2007), https://doi.org/10.1016/j.meatsci.2007.03.009, at 238–239.
[88] Kieran D. Cox, Garth A. Covernton, Hailey L. Davies, John F. Dower, Francis Juanes & Sarah E. Dudas, Human Consumption of Microplastics, 53 Environmental Science & Technology 7068–7074 (2019), https://doi.org/10.1021/acs.est.9b01517, at 7068 (estimating 39,000 to 52,000 particles a year from the foods and beverages studied); Josefa Domenech & Ricard Marcos, Pathways of human exposure to microplastics, and estimation of the total burden, 39 Current Opinion in Food Science 144–151 (2021), https://doi.org/10.1016/j.cofs.2021.01.004, at 147 tbl. 1, 148 (estimating 2.93 × 10^10 particles a year, and cautioning that the figure must be taken with caution because detection methods are not established). Omitted from the discussion in this section, but nonetheless relevant to FDA's analysis, is contamination by plastic-associated chemicals. Additives including antioxidants, plasticizers, and pigments are regularly detected in the American food system. These additives reach food both from plastic packaging and equipment and from the particles themselves. Sections I.B.3 and I.B.4 note several examples of their migration, but this petition does not examine the larger body of studies documenting it.
[89] Maria Hayder, Maud M. Laan & Annemarie P. van Wezel, Exposure to microplastics from food: Comparative analysis of food types and quantification techniques, 501 Journal of Hazardous Materials art. 140657 (2026), https://doi.org/10.1016/j.jhazmat.2025.140657, at 1, 4, 6.

commercial fish species has identified microplastic particles in digestive tracts at concentrations ranging from less than one to more than seven particles per fish.[90] Bivalve mollusks, which filter large volumes of water during feeding, show particularly high contamination levels.[91] One review estimates that people consume between approximately 500 and 32,750 microplastic particles annually through mollusk consumption alone.[92] Crustacean consumption contributes an additional estimated 322 to 19,511 particles per person annually.[93] The particles identified in seafood predominantly consist of polyethylene, polypropylene, PET, and polyamide polymers.[94] Estimates for fish run as high as 32,375 particles per person per year, based mostly on particles found in fish digestive tracts.[95]

Although most microplastics in fish are in the digestive tract, which is removed before eating, several studies have detected plastic particles in the edible muscle of commercially important species.[96] For smaller fish species consumed whole, such as anchovies and sardines, and for bivalves like mussels and oysters that are typically eaten including their digestive systems, people who eat them take in more of that contamination.[97] Estimated maximum yearly intake from all seafood is in the tens of thousands of particles per capita.[98] Farmed fish can also take in microplastics from fishmeal in their feed (Section I.B.2).

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[90] Garrido Gamarro & Costanzo, *supra* note 41, at 34 & tbl. 3 (at 36–41).

[91] Evangelos Danopoulos, Lauren C. Jenner, Maureen Twiddy & Jeanette M. Rotchell, Microplastic Contamination of Seafood Intended for Human Consumption: A Systematic Review and Meta-Analysis, 128 Environmental Health Perspectives art. 126002 (2020), https://doi.org/10.1289/EHP7171, at 126002-1, 126002-24 tbl. 5 (reported contents of 0 to 10.5 particles per gram in mollusks, 0.1 to 8.6 in crustaceans, and 0 to 2.9 in fish); M.F.M. Santana, L.G. Ascer, M.R. Custódio, F.T. Moreira & A. Turra, Microplastic contamination in natural mussel beds from a Brazilian urbanized coastal region: Rapid evaluation through bioassessment, 106 Marine Pollution Bulletin 183–189 (2016), https://doi.org/10.1016/j.marpolbul.2016.02.074, at 183 (75 percent of sampled filter-feeding mussels in one Brazilian estuary had ingested microplastics).

[92] Inneke Hantoro, Ansje J. Löhr, Frank G. A. J. Van Belleghem, Budi Widianarko & Ad M. J. Ragas, Microplastics in coastal areas and seafood: implications for food safety, 36 Food Additives & Contaminants: Part A 674–711 (2019), https://doi.org/10.1080/19440049.2019.1585581, at 693, 701 tbl. 6.

[93] *Id.* at 701 tbl. 6.

[94] Garrido Gamarro & Costanzo, *supra* note 41, at 34.

[95] Hantoro et al., *supra* note 92, at 693, 701 tbl. 6.

[96] Garrido Gamarro & Costanzo, *supra* note 41, at 34; Pouran Makhdoumi, Hooshyar Hossini & Meghdad Pirsaheb, A review of microplastic pollution in commercial fish for human consumption, 38 Reviews on Environmental Health 97–109 (2023), https://doi.org/10.1515/reveh-2021-0103, at 97, 99; Commission Report at 57.

[97] Commission Report at 57; Garrido Gamarro & Costanzo, *supra* note 41, at 33; Hantoro et al., *supra* note 92, at 701.

[98] Danopoulos et al., *supra* note 91, at 126002-1, 126002-24 & tbl. 6; Lisbeth Van Cauwenberghe & Colin R. Janssen, Microplastics in bivalves cultured for human consumption, 193 Environmental Pollution 65–70 (2014), https://doi.org/10.1016/j.envpol.2014.06.010, at 65, 68; Hantoro et al., *supra* note 92, at 701 tbl. 6. The Minderoo-Monaco Commission concluded that the available evidence does not show seafood to be a major pathway for microplastics to reach people, because typically only one or two particles are found in an individual fish or shellfish and drinking water and air may contribute as much or more. It added that seafood may contribute more for people whose diets rely heavily on it, that its contribution could grow without corrective action, and that the contribution of nanoplastics, which are too small to count reliably, remains unclear. Commission Report at 57, 58 tbl. 3.2.

2. Meat and Dairy

Meat and dairy products represent two of the most widely consumed food categories in the American diet, and both carry documented microplastic and plastic chemical contamination arising from multiple points in their respective supply chains. For meat, a preliminary study of ten beef hamburgers found microplastics in every sample, with concentrations of individual polymers ranging from 200 to 38,400 particles per kilogram.[99] Eighteen different polymers were detected; polycarbonate was the most abundant.[100] One study detected extruded polystyrene particles on the surface of packaged chicken meat at concentrations ranging from 4 to 18.7 microplastic particles per kilogram, and found that rinsing the meat did not effectively remove the particles.[101] From those counts, and based on average meat consumption, the authors estimated that a person would ingest between 7 micrograms and 1.4 milligrams of these particles a day, depending on the brand.[102] Styrene monomer from polystyrene packaging also migrates into raw chicken, where it has been measured at concentrations of 2.6 nanograms per gram, and into raw beef, where it has been measured at 5.6 nanograms per gram.[103] The study of polyethylene film described in Section I.B.4 found that migration of the test chemical into pork and chicken increased with the meat's fat content.[104]

Dairy products have been found to contain microplastic particles across all stages of processing, from raw milk collected at the farm through finished consumer products. A study of microplastics of 5 micrometers or larger in raw and processed cow's milk found concentrations that ranged from 204 to 1,004 microplastic particles per 100 milliliters across the milk samples examined.[105] The dominant polymer types identified were polyethylene, polyester, polypropylene, polytetrafluoroethylene, and polystyrene, with smaller amounts of polyamide, polyurethane, polysulfone, and polyvinyl alcohol also detected across the sample set. Powdered milk products showed the highest contamination levels, reaching 1,004 particles per 100 milliliters after reconstitution, and the study observed a trend of increasing particle counts corresponding to the degree of processing undergone, from farm milk through liquid commercial products to reconstituted powders. For cheese specifically, concentrations of di(2-ethylhexyl) adipate, a plasticizer (a chemical added to make plastic flexible), have been detected on the surface of fatty cheese wrapped in PVC film at levels of 12.2 and 18.9 milligrams per square decimeter after 240 hours of contact, above the European Union's overall migration limit for plastic food-contact materials of 10 milligrams per square

[99] E. Visentin, G. Niero, F. Benetti et al., Preliminary characterization of microplastics in beef hamburgers, 217 Meat Science art. 109626 (2024), https://doi.org/10.1016/j.meatsci.2024.109626, at 1, 5 tbl. 1, 7.
[100] *Id.* at 1, 7 & tbl. 3.
[101] Mikaël Kedzierski, Benjamin Lechat, Olivier Sire, Gwénaël Le Maguer, Véronique Le Tilly & Stéphane Bruzaud, Microplastic contamination of packaged meat: Occurrence and associated risks, 24 Food Packaging and Shelf Life art. 100489 (2020), https://doi.org/10.1016/j.fpsl.2020.100489; *see also* Garrido Gamarro & Costanzo, *supra* note 41, at 25.
[102] Kedzierski et al., *supra* note 101, tbl. 2.
[103] Susan Genualdi, Patricia Nyman & Timothy Begley, Updated evaluation of the migration of styrene monomer and oligomers from polystyrene food contact materials to foods and food simulants, 31 Food Additives & Contaminants: Part A 723–733 (2014), https://doi.org/10.1080/19440049.2013.878040, at 723, 727 tbl. 2, 728.
[104] Sanches Silva et al., *supra* note 87, at 238; *see also* Garrido Gamarro & Costanzo, *supra* note 41, at 25.
[105] Paulo A. Da Costa Filho, Daniel Andrey, Bjorn Eriksen et al., Detection and characterization of small-sized microplastics (≥ 5 μm) in milk products, 11 Scientific Reports art. 24046 (2021), https://doi.org/10.1038/s41598-021-03458-7, at 1, 4, 6 tbl. 2, 7.

decimeter.[106] Styrene, which the National Toxicology Program lists as reasonably anticipated to be a human carcinogen, also leaches from disposable polystyrene cups into hot milk drinks: one study measured up to 8.30 micrograms per liter in hot milk and 8.65 in hot cocoa milk, more than in hot tea, and found that migration was already considerable within the first ten minutes and increased with the drink's temperature, fat content, and time in the cup.[107]

3. Fruits and Vegetables

A 2020 study reported plastic particles inside the edible portions of apples, pears, broccoli, lettuce, and carrots bought from local shops and a supermarket, after the produce had been washed and, except for the lettuce and broccoli, peeled.[108] Measuring only particles smaller than 10 micrometers, it reported mean concentrations of 195,500 particles per gram in apples, 189,550 in pears, 126,150 in broccoli, 101,950 in carrots, and 50,550 in lettuce.[109] The authors suggested that fruits carry more particles than vegetables because of the extensive vascular network of fruit and the larger root systems and greater age of fruit trees.[110] The study counted particles by electron microscopy and does not report the polymer of any particle, and a 2026 review of dietary exposure that included it found that, for fruits and vegetables, studies relying on visual observation reported drastically higher particle numbers than studies using other techniques.[111]

The particles likely enter through the roots from the soil and move within the plant, though particles that stick to plant surfaces and particles deposited from the air during growth also add to the total.[112] Applying that study's mean concentration to the 400 grams of fruits and vegetables a day that the World Health Organization recommends, one review estimated that a person would ingest about 19 billion particles a year from produce, more than from any other source it assessed.[113] The 2026 review likewise found that fruits, vegetables, and grains account for nearly all of the estimated number of particles people ingest with food, while cautioning that the produce results depend heavily on the analytical method used.[114]

4. Salt, Sugar, and Honey

Table salt is eaten daily and is used to preserve many processed foods.[115] A 2020 systematic review of ten studies found microplastics in most of the salt samples tested, and a study of twelve sea salt brands

[106] A.E. Goulas, K.I. Anifantaki, D.G. Kolioulis & M.G. Kontominas, Migration of di-(2-ethylhexyl)adipate plasticizer from food-grade polyvinyl chloride film into hard and soft cheeses, 83 Journal of Dairy Science 1712–1718 (2000), https://doi.org/10.3168/jds.S0022-0302(00)75040-5, at 1712.
[107] Mohammad-Reza Khaksar & Mahmoud Ghazi-Khansari, Determination of migration monomer styrene from GPPS (general purpose polystyrene) and HIPS (high impact polystyrene) cups to hot drinks, 19 Toxicology Mechanisms and Methods 257–261 (2009), https://doi.org/10.1080/15376510802510299, at 257, 260; Genualdi et al., *supra* note 103, at 723.
[108] Gea Oliveri Conti, Margherita Ferrante, Mohamed Banni et al., Micro- and nano-plastics in edible fruit and vegetables. The first diet risks assessment for the general population, 187 Environmental Research art. 109677 (2020), https://doi.org/10.1016/j.envres.2020.109677, at 2–3.
[109] *Id.* at 2, 5 tbl. 1.
[110] *Id.* at 5–6.
[111] *Id.* at 3; Hayder et al., *supra* note 89, at 1, 3 tbl. 1, 6–7.
[112] Jadhav & Medyńska-Juraszek, *supra* note 42, at 3; Garrido Gamarro & Costanzo, *supra* note 41, at 18.
[113] Domenech & Marcos, *supra* note 88, at 146–147 & tbl. 1; Commission Report at 58 tbl. 3.2.
[114] Hayder et al., *supra* note 89, at 1, 7, 9.
[115] Qun Zhang, Elvis Genbo Xu, Jiana Li et al., A Review of Microplastics in Table Salt, Drinking Water, and Air: Direct Human Exposure, 54 Environmental Science & Technology 3740–3751 (2020), https://doi.org/10.1021/acs.est.9b04535.

bought in Minneapolis found human-made particles, nearly all fibers, in every brand, averaging 212 particles per kilogram.[116] Sea salt is made by evaporating seawater, and its contamination likely reflects the plastic in the water used to make it; a study of 39 salt brands found that particle levels in sea salt tracked plastic emissions from nearby rivers and levels in nearby seawater, while cautioning that this is indirect evidence.[117] The 2020 review found 0 to 1,674 particles per kilogram in sea salt, 8 to 462 in lake salt, and 0 to 204 in rock and well salt.[118] Individual brands have been reported far above that range, including about 13,600 particles per kilogram in one Indonesian sea salt and 13,500 to 19,800 particles per kilogram in five Croatian sea salts, the latter from a study the review rated at high risk of bias.[119] Sea salts produced in Asia contained more particles on average than those produced on other continents.[120]

Rock salt, mined from underground deposits, contains fewer particles than sea salt, and the particles it does contain may enter during processing.[121] The polymers most often found in salt are polyethylene, polypropylene, and PET.[122] These are also common packaging materials, but the 39-brand study compared the particles in each salt with that product's packaging and concluded that packaging did not substantially affect the particles present.[123] A study of Italian and Croatian sea salts found more particles in low-cost brands than in high-cost ones, which the authors suggested may reflect less effective purification of the cheaper salts.[124] Estimates of annual intake from salt range from under 100 particles per person to as many as 73,000.[125]

Synthetic fibers and fragments have also been reported in honey. A study of 47 honeys from supermarkets and German beekeepers found 10 to 336 fibers and 2 to 82 fragments per kilogram, and found similar particles on the flowers bees visit, indicating that much of this material may be carried into the hive by bees; handling of the product may add more.[126] An earlier study by the same authors found fibers and fragments in all five commercial sugars it tested, the most in unrefined cane sugar (560 fibers and 540

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[116] Evangelos Danopoulos, Lauren Jenner, Maureen Twiddy & Jeanette M. Rotchell, Microplastic contamination of salt intended for human consumption: a systematic review and meta-analysis, 2 SN Applied Sciences art. 1950 (2020), https://doi.org/10.1007/s42452-020-03749-0, at 1, 12; Mary Kosuth, Sherri A. Mason & Elizabeth V. Wattenberg, Anthropogenic contamination of tap water, beer, and sea salt, 13 PLOS ONE art. e0194970 (2018), https://doi.org/10.1371/journal.pone.0194970, at 1, 4.
[117] Garrido Gamarro & Costanzo, *supra* note 41, at 35; Ji-Su Kim, Hee-Jee Lee, Seung-Kyu Kim & Hyun-Jung Kim, Global Pattern of Microplastics (MPs) in Commercial Food-Grade Salts: Sea Salt as an Indicator of Seawater MP Pollution, 52 Environmental Science & Technology 12819–12828 (2018), https://doi.org/10.1021/acs.est.8b04180, at 12819, 12826.
[118] Danopoulos et al., *supra* note 116, at 1, 10.
[119] Kim et al., *supra* note 117, at 12819, 12826; Renzi & Blašković, *supra* note 74; Danopoulos et al., *supra* note 116, at 10. A study of sea salts from India's two largest salt-producing states reported 23 to 115 particles per 200 grams. A. Vidyasakar, S. Krishnakumar, K. Suresh Kumar et al., Microplastic contamination in edible sea salt from the largest salt-producing states of India, 171 Marine Pollution Bulletin art. 112728 (2021), https://doi.org/10.1016/j.marpolbul.2021.112728, at 1.
[120] Kim et al., *supra* note 117, at 12823; Garrido Gamarro & Costanzo, *supra* note 41, at 35.
[121] Kim et al., *supra* note 117, at 12819, 12823, 12825; Garrido Gamarro & Costanzo, *supra* note 41, at 19, 35.
[122] Kim et al., *supra* note 117, at 12823.
[123] *Id.* at 12824.
[124] Renzi & Blašković, *supra* note 74.
[125] Commission Report at 58 tbl. 3.2; Danopoulos et al., *supra* note 116, at 13.
[126] Gerd Liebezeit & Elisabeth Liebezeit, Origin of Synthetic Particles in Honeys, 65 Polish Journal of Food and Nutrition Sciences 143–147 (2015), https://doi.org/10.1515/pjfns-2015-0025, at 143, 145–146.

fragments per kilogram), but did not determine whether the particles were synthetic or natural.[127] One estimate based on American diets put annual intake at about 73 particles from honey and 8,319 from sugar.[128]

5. Bottled Beverages

Both tap water and bottled water are pathways for microplastic ingestion. Bottled water is regulated by FDA, and municipal tap water by EPA. In a study of 259 bottles from eleven brands bought in nine countries, 93 percent of bottles contained microplastics, and particles larger than 100 micrometers were about twice as numerous as in the same research group's earlier study of tap water (10.4 versus 5.45 particles per liter).[129] Reported concentrations in bottled water range from none to more than ten thousand particles per liter, and far higher in a study that counted particles smaller than ten micrometers by electron microscopy.[130] Packaging is one source: in water from plastic bottles, the particles identified often match the bottle (PET) or the cap (polypropylene).[131] Another study estimated that a person who drinks two liters of bottled water a day would ingest about 9.9 billion particles a year from that source.[132] An earlier estimate based on American diets put the additional intake for a person who drinks only bottled water at about 90,000 particles a year, compared with about 4,000 for a person who drinks only tap water.[133]

Beer and other alcoholic beverages represent additional beverage categories with documented microplastic contamination. A 2025 study of drinks sold in France found microplastics in every category it tested, with mean levels of about 3 particles per liter in water, 28 to 45 in colas, teas, and lemonades, 83 in beer, and 8 in wine; the highest levels were in glass bottles, and most particles matched the paint on the metal caps.[134] A study of twelve American beer brands found an average of about 4 human-made particles per liter, nearly all fibers, after accounting for laboratory contamination.[135] Estimates of annual intake from alcoholic beverages run as high as 294 particles per person.[136] The presence of microplastics across

[127] Gerd Liebezeit & Elisabeth Liebezeit, Non-pollen particulates in honey and sugar, 30 Food Additives & Contaminants: Part A 2136–2140 (2013), https://doi.org/10.1080/19440049.2013.843025, at 2136, 2138–2139.
[128] Commission Report at 58 tbl. 3.2; Cox et al., *supra* note 88, at 7069, 7072.
[129] Mason et al., *supra* note 79, at 1, 9; Kosuth et al., *supra* note 116, at 1.
[130] Mason et al., *supra* note 79, at 1, 5 tbl. 2; Schymanski et al., *supra* note 79, at 154; Oßmann et al., *supra* note 79, at 307; P. Zuccarello, M. Ferrante, A. Cristaldi et al., Exposure to microplastics (<10 μm) associated to plastic bottles mineral water consumption: The first quantitative study, 157 Water Research 365–371 (2019), https://doi.org/10.1016/j.watres.2019.03.091, at 365 (mean of 5.42 × 10^7 particles per liter between 0.5 and 10 micrometers); Hayder et al., *supra* note 89, at 5.
[131] Schymanski et al., *supra* note 79, at 154; Oßmann et al., *supra* note 79, at 307; Mason et al., *supra* note 79, at 1, 6 (polypropylene the most common polymer, at 54 percent, consistent with bottle caps).
[132] Domenech & Marcos, *supra* note 88, at 147 tbl. 1 (9.89 × 10^9 particles a year from bottled water, based on a mean of 13.55 × 10^6 particles per liter and 2 liters a day), 148 (cautioning that the estimates must be taken with caution because detection methods are not established).
[133] Cox et al., *supra* note 88, at 7068.
[134] Iseline Chaïb, Périne Doyen, Pauline Merveillie, Alexandre Dehaut & Guillaume Duflos, Microplastic contaminations in a set of beverages sold in France, 144 Journal of Food Composition and Analysis art. 107719 (2025), https://doi.org/10.1016/j.jfca.2025.107719, at 1, 7.
[135] Kosuth et al., *supra* note 116, at 1.
[136] Commission Report at 58 tbl. 3.2; Cox et al., *supra* note 88, at 7069 (modeling alcohol consumption as beer); Domenech & Marcos, *supra* note 88, at 147 tbl. 1.

all major beverage categories indicates that liquid consumption represents an unavoidable pathway for plastic particle ingestion in the American diet.

II. Microplastic Detection in People

Over the past several years, scientific investigations have confirmed with increasing clarity that microplastics are present within nearly every biological system of the human body. These synthetic polymer particles have been discovered in feces, blood, lungs, liver, spleen, colon, placenta, meconium (the first feces of a newborn), breast milk, saliva, and even urine. At least 40 different polymers have been detected in human tissues, including polyethylene, polypropylene, PET, PVC, polystyrene, polycarbonate, polyoxymethylene, polymethyl methacrylate, polybutylene terephthalate, acrylonitrile butadiene styrene, polyamide, polyacrylonitrile, polyurethane, styrene-ethylene-butylene-styrene, polyester, and polysulfone, in particles reported to range from about 0.7 micrometers to 5 millimeters.[137]

A. Organs and Tissues

Recent studies confirm widespread contamination of human cardiovascular tissues with microplastics. Researchers have found these particles in atherosclerotic plaque (the fatty deposits that narrow arteries), in heart tissue, and in veins from patients undergoing medical procedures. In one study of plaque removed from the carotid arteries of 257 patients, polyethylene was detected in the plaque of 150 patients (58.4 percent), and 31 of those patients (12.1 percent) also had measurable polyvinyl chloride.[138] Cardiac tissue from open heart surgery patients showed substantial microplastic contamination. Epicardial fat averaged 79,980 particles per gram, and heart muscle averaged 49 particles per gram.[139] Blood samples from these patients contained 7.2 particles per milliliter before surgery and 34.8 particles per milliliter after, a change the authors say may partly reflect exposure during surgery.[140] Vein tissue from other patients showed microplastic contamination as well.[141]

Microplastics have also been detected in the colon, liver, kidney, and spleen. Colon tissue removed from surgical patients (colectomy specimens) contained a mean of 28.1 particles per gram, more than 96 percent of them fibers.[142] The first direct analysis of human liver tissue found that patients with cirrhosis

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[137] Gurusamy Kutralam-Muniasamy, V.C. Shruti, Fermín Pérez-Guevara & Priyadarsi D. Roy, Microplastic diagnostics in humans: "The 3Ps" Progress, problems, and prospects, 856 Science of the Total Environment art. 159164 (2023), https://doi.org/10.1016/j.scitotenv.2022.159164, at 8–9.

[138] Raffaele Marfella, Francesco Prattichizzo, Celestino Sardu et al., Microplastics and Nanoplastics in Atheromas and Cardiovascular Events, 390 New England Journal of Medicine 900–910 (2024), https://doi.org/10.1056/NEJMoa2309822, at 900. Petitioners rely on this study for detection and for the association with later cardiovascular events, not for its mass values, for the reason given in Section IV.B.3.

[139] Yunxiao Yang, Enzehua Xie, Zhiyong Du et al., Detection of Various Microplastics in Patients Undergoing Cardiac Surgery, 57 Environmental Science & Technology 10911–10918 (2023), https://doi.org/10.1021/acs.est.2c07179, at 10911; Adrian Goldsworthy, Liam A. O'Callaghan, Ciara Blum et al., Micro-nanoplastic induced cardiovascular disease and dysfunction: a scoping review, 35 Journal of Exposure Science & Environmental Epidemiology 746–769 (2025), https://doi.org/10.1038/s41370-025-00766-2, at 754 tbl. 2.

[140] Yang et al., *supra* note 139, at 10911, 10915 (patients are exposed to "medical devices with plastic components during cardiac surgery, which may alter the in vivo situations of MPs"); Goldsworthy et al., *supra* note 139, at 754 tbl. 2 (means of 7.2 and 34.8 particles per milliliter).

[141] Goldsworthy et al., *supra* note 139, at 746, 754.

[142] Yusof Shuaib Ibrahim, Sabiqah Tuan Anuar, Alyza A. Azmi et al., Detection of microplastics in human colectomy specimens, 5 JGH Open 116–121 (2021), https://doi.org/10.1002/jgh3.12457, at 116.

(scarring of the liver) had microplastic concentrations ranging from 4.6 to 11.9 particles per gram (3.2 to 9.9 after correction for blank samples), while all liver, kidney, and spleen samples from five people without liver disease tested negative, considering the limit of detection.[143] Microplastics detected in kidney tissue ranged from 1 to 29 micrometers in size.[144] In the liver study described above, spleen tissue from people without liver disease had a median of 1.1 particles per gram (0.9 after subtracting blank samples), a level the authors counted as negative considering the limit of detection.[145] These findings suggest that liver disease may alter microplastic retention or clearance dynamics.[146] Additional studies have noted higher microplastic burdens in tumor tissues compared to adjacent normal tissues, indicating that local disease processes may affect polymer accumulation.[147] Brain tissue has also been examined. A 2025 analysis of autopsy specimens from a New Mexico medical examiner's office measured plastic by mass in liver, kidney, and brain, and found more in brain tissue than in either of the other two organs. Liver and brain specimens collected in 2024 contained more plastic than those collected in 2016 ($p = 0.01$), and twelve brains from people who had been diagnosed with dementia contained more than any other group examined.[148]

Microplastics have also been found in reproductive tissue. More than eleven particles per gram were detected in testes.[149] Polystyrene was the most common polymer detected, and most were between 20 and 100 micrometers in size.[150] A study of female reproductive tissue from 60 patients also found

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[143] Thomas Horvatits, Matthias Tamminga, Beibei Liu et al., Microplastics detected in cirrhotic liver tissue, 82 eBioMedicine art. 104147 (2022), https://doi.org/10.1016/j.ebiom.2022.104147, at 1, 4.
[144] Sara Massardo, Daniela Verzola, Stefano Alberti et al., MicroRaman spectroscopy detects the presence of microplastics in human urine and kidney tissue, 184 Environment International art. 108444 (2024), https://doi.org/10.1016/j.envint.2024.108444, at 1.
[145] Horvatits et al., *supra* note 143, at 1, 4.
[146] Kutralam-Muniasamy et al., *supra* note 137, at 7, 9.
[147] *Id.*
[148] Alexander J. Nihart, Marcus A. Garcia, Eliane El Hayek et al., Bioaccumulation of microplastics in decedent human brains, 31 Nature Medicine 1114–1119 (2025), https://doi.org/10.1038/s41591-024-03453-1, at 1114, 1116, 1118. An erratum correcting a duplicated image was published at 31 Nature Medicine 1367 (2025), https://doi.org/10.1038/s41591-025-03675-x. Petitioners rely on this study for the fact of detection in brain tissue and for the direction of the change over time, not for its mass values. Most of the mass reported in brain tissue was polyethylene. A 2025 study assessing this technique concluded that pyrolysis gas chromatography–mass spectrometry is not currently a suitable method for measuring polyethylene or polyvinyl chloride in biological samples, because fats in tissue break down into the same compounds the method counts as plastic and enzymatic digestion reduces but does not remove the interference. Cassandra Rauert, Nathan Charlton, Angus Bagley, Sarah A. Dunlop, Christos Symeonides & Kevin V. Thomas, Assessing the Efficacy of Pyrolysis–Gas Chromatography–Mass Spectrometry for Nanoplastic and Microplastic Analysis in Human Blood, 59 Environmental Science & Technology 1984–1994 (2025), https://doi.org/10.1021/acs.est.4c12599, at 1984, 1985, 1992 (study funded by the Minderoo Foundation). The German Federal Institute for Risk Assessment, reviewing the Nihart study, concluded that the reported brain amounts appear implausibly high and that measurement artifacts leading to overestimated exposure cannot be ruled out. German Federal Institute for Risk Assessment (BfR), Microplastics in the brain: The BfR assesses a controversial study on microplastics and nanoplastics in human organs, Communication No. 056/2025 (Dec. 3, 2025), at 1, 4, 7, https://www.bfr.bund.de/en/notification/microplastics-in-the-brain/ (last visited Nov. 4, 2026).
[149] Qiancheng Zhao, Long Zhu, Jiaming Weng et al., Detection and characterization of microplastics in the human testis and semen, 877 Science of the Total Environment art. 162713 (2023), https://doi.org/10.1016/j.scitotenv.2023.162713, at 1.
[150] *Id.*

microplastics in every sample of three kinds of diseased tissue, including ovarian cysts and uterine tubes.[151] These tissues contained an average of 1.5 particles per gram, and the most common particles were polypropylene and polyethylene.[152]

B. Bodily Fluids

Human feces is among the most frequently studied substances for microplastic detection. One of the earliest human studies found microplastics in stool samples from all eight participants, who lived in different countries.[153] Each 10-gram sample contained a median of 20 microplastic particles, predominantly polypropylene and PET.[154] Additional studies have reinforced and expanded on these findings. Another study identified significantly higher microplastic concentrations in patients with inflammatory bowel disease than in healthy patients: 41.8 particles/g versus 28.0 particles/g, respectively.[155] A study of fecal samples from a coastal Indonesian population showed HDPE was the most common polymer detected, with a mean concentration of 9.2 micrograms of microplastic per gram of feces, ranging from 3.3 to 13.99 micrograms per gram.[156] These findings highlight both environmental exposure variability and host physiological factors influencing accumulation and elimination.

Human blood has also been shown to contain plastic particles, signifying systemic transport beyond the gastrointestinal tract. In one study, microplastics were detected in 77 percent of blood samples from healthy volunteers, at an average concentration of 1.6 micrograms per milliliter.[157] Polymers identified included PET, polypropylene, and polyethylene, and approximately half of the samples contained two or more types simultaneously. The authors concluded that this study of 22 donors demonstrated that plastic particles can be taken up into the human bloodstream. A 2025 study applied an improved version of the same mass-based method to whole blood from 102 adults, with added controls against contamination: blood was drawn into glass tubes by a plastic-free procedure, every batch included procedural blanks (samples run through the analysis without blood, to measure background contamination), and the method's recovery was tested in blood to which known amounts of plastic had been added rather than in a substitute material such as soil or water. Plastic was detected in every sample. Polyvinyl chloride was the most frequently detected polymer, found in 99 percent of samples, while polyethylene was not detected at all. In the twenty percent of samples where the total was high enough to measure precisely, it averaged

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[151] Chunlin Dong, Hongwen Xu, Yaying Lin et al., Microplastics detected in three types of female reproductive organs using micro-Raman spectroscopy, 285 Ecotoxicology and Environmental Safety art. 117099 (2024), https://doi.org/10.1016/j.ecoenv.2024.117099, at 1.
[152] *Id.*
[153] Philipp Schwabl, Sebastian Köppel, Philipp Königshofer et al., Detection of Various Microplastics in Human Stool: A Prospective Case Series, 171 Annals of Internal Medicine 453–457 (2019), https://doi.org/10.7326/M19-0618, at 453.
[154] *Id.*
[155] Zehua Yan, Yafei Liu, Ting Zhang, Faming Zhang, Hongqiang Ren & Yan Zhang, Analysis of Microplastics in Human Feces Reveals a Correlation between Fecal Microplastics and Inflammatory Bowel Disease Status, 56 Environmental Science & Technology 414–421 (2022), https://doi.org/10.1021/acs.est.1c03924, at 414.
[156] Arif Luqman, Husna Nugrahapraja, Ruri Agung Wahyuono et al., Microplastic Contamination in Human Stools, Foods, and Drinking Water Associated with Indonesian Coastal Population, 8 Environments art. 138 (2021), https://doi.org/10.3390/environments8120138, at 1.
[157] Heather A. Leslie, Martin J.M. van Velzen, Sicco H. Brandsma, A. Dick Vethaak, Juan J. Garcia-Vallejo & Marja H. Lamoree, Discovery and quantification of plastic particle pollution in human blood, 163 Environment International art. 107199 (2022), https://doi.org/10.1016/j.envint.2022.107199, at 1, 5. Petitioners rely on this study for detection, not for its mass values, for the reason given in Section IV.B.3.

386 nanograms per milliliter.[158] Another study of patients with acute coronary syndrome found microplastics in 100 percent of blood samples.[159] A study of clots retrieved from 30 patients during procedures for stroke, heart attack, or deep vein thrombosis identified microplastics in 80 percent of them.[160]

Urine, while infrequently studied, has shown evidence of microplastic contamination. The first detection of microplastic particles in urine was reported in 2023, although concentrations were not yet quantified.[161] A 2024 study found polyethylene and polystyrene to be the most common polymers in urine.[162] This suggests that microplastics may be partially filtered by the kidneys and excreted. In semen, polyethylene and PVC were the most common polymers detected.[163]

C. Fetuses and Newborns

Plastics in placental tissue have received particular attention due to the placenta's role in fetal development. The first researchers to document microplastics in the placenta found 12 polymer fragments between 5 and 10 micrometers in four of six samples analyzed.[164] A 2024 study examined 62 placentas and measured plastic by mass rather than counting particles. Plastic was found in all 62.[165] The reported totals ranged from 6.5 to 685 micrograms per gram of tissue, but more than half of that mass was polyethylene, a figure Petitioners do not rely on for the reason given in Section IV.B.3. Polyvinyl chloride and nylon each accounted for roughly a tenth of the mass, with nine further polymers making up another quarter.[166] The authors note two limitations. Their polymer standard did not include rayon, which their imaging analysis found in quantity, so their total understates the amount present to that extent. And the samples were stored and centrifuged in plastic tubes, although the authors treat contamination as a minor

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[158] Federica Nardella, Marthinus Brits, Martin J. M. van Velzen et al., Advancing pyrolysis-gas chromatography-mass spectrometry for the accurate quantification of micro- and nanoplastics in human blood, 5 Microplastics and Nanoplastics art. 48 (2025), https://doi.org/10.1186/s43591-025-00152-7, at 1, 3, 6–7 (pages as numbered in the published article, "Page X of 10"). The authors attribute the difference between their figures and those reported by earlier studies using this method to differences in sample preparation and quality control, and to the vulnerability of the marker compounds commonly used for polyethylene and polyvinyl chloride to interference from biological material.

[159] Yunxiao Yang, Feng Zhang, Zhili Jiang et al., Microplastics are associated with elevated atherosclerotic risk and increased vascular complexity in acute coronary syndrome patients, 21 Particle and Fibre Toxicology art. 34 (2024), https://doi.org/10.1186/s12989-024-00596-4, at 5.

[160] Tingting Wang, Zhiheng Yi, Xiaoqiang Liu et al., Multimodal detection and analysis of microplastics in human thrombi from multiple anatomically distinct sites, 103 eBioMedicine art. 105118 (2024), https://doi.org/10.1016/j.ebiom.2024.105118, at 1.

[161] Concetta Pironti, Valentina Notarstefano, Maria Ricciardi, Oriana Motta, Elisabetta Giorgini & Luigi Montano, First Evidence of Microplastics in Human Urine, a Preliminary Study of Intake in the Human Body, 11 Toxics art. 40 (2023), https://doi.org/10.3390/toxics11010040, at 1.

[162] Massardo et al., *supra* note 144, at 1.

[163] Zhao et al., *supra* note 149, at 1.

[164] Antonio Ragusa, Alessandro Svelato, Criselda Santacroce et al., Plasticenta: First evidence of microplastics in human placenta, 146 Environment International art. 106274 (2021), https://doi.org/10.1016/j.envint.2020.106274, at 3.

[165] Marcus A. Garcia, Rui Liu, Alex Nihart et al., Quantitation and identification of microplastics accumulation in human placental specimens using pyrolysis gas chromatography mass spectrometry, 199 Toxicological Sciences 81–88 (2024), https://doi.org/10.1093/toxsci/kfae021, at 81, 85.

[166] *Id.* Polyethylene made up the remaining 54 percent, and Petitioners do not rely on that figure, for the reason given in Section IV.B.3.

contributor because ten of the 62 samples came in below 20 micrograms per gram.[167] Another study evaluated placenta, the first feces of a newborn, infant feces, breast milk, and formula from 18 mother-infant pairs, identifying 16 plastic polymers and reporting that 74 percent of particles were between 20–50 micrometers.[168] This study presents one of the most comprehensive pictures of early-life microplastic exposure to date. Another study found microplastics larger than 50 micrometers in both the placenta and the first feces of newborns delivered by cesarean section.[169] Another found that the median concentration of PET in the feces of six infants was about fourteen times that in the feces of ten adults.[170]

Breast milk, a critical route of postnatal nutrition, is also contaminated with microplastics. Microplastic fragments and spheres were found in 76 percent of human milk samples from 34 women in Rome.[171] The highest concentration observed was 2.72 microplastics per gram. In the study of 18 mother-infant pairs described above, the seven breast milk samples had a median of 20.2 particles per gram.[172] Together with the findings in placenta and meconium, these studies suggest that infants are exposed to microplastics by more than one route, before and after birth.

Together, these studies, which used different methods on different tissues and fluids, show that microplastics enter the human body and have been found in most of the organs and fluids examined, in newborns as well as adults.

III. Health Harms from Microplastics[173]

The evidence in this section differs in kind and in strength, and Petitioners identify each study by its design. Most of the evidence comes from the laboratory: human cells, tissue models grown from human stem cells, and animals given measured doses. A smaller body of work examines people directly, through analysis of human tissue and through observational studies of patients. The one formal, graded assessment of this literature, a 2024 systematic review of three human observational studies and twenty-eight animal studies, concluded that microplastic exposure is "suspected" to harm digestive, reproductive, and respiratory health, and described research on human health effects as still at an early stage.[174] Section 402(a)(1) of the Act asks whether a substance may render food injurious to health, not whether injury in people has been proved.

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[167] *Id.* at 85, 87.
[168] Shaojie Liu, Jialin Guo, Xinyuan Liu et al., Detection of various microplastics in placentas, meconium, infant feces, breastmilk and infant formula: A pilot prospective study, 854 Science of the Total Environment art. 158699 (2023), https://doi.org/10.1016/j.scitotenv.2022.158699, at 1, 4.
[169] Thorsten Braun, Loreen Ehrlich, Wolfgang Henrich et al., Detection of Microplastic in Human Placenta and Meconium in a Clinical Setting, 13 Pharmaceutics art. 921 (2021), https://doi.org/10.3390/pharmaceutics13070921, at 1.
[170] Junjie Zhang, Lei Wang, Leonardo Trasande & Kurunthachalam Kannan, Occurrence of Polyethylene Terephthalate and Polycarbonate Microplastics in Infant and Adult Feces, 8 Environmental Science & Technology Letters 989–994 (2021), https://doi.org/10.1021/acs.estlett.1c00559, at 989.
[171] Antonio Ragusa, Valentina Notarstefano, Alessandro Svelato et al., Raman Microspectroscopy Detection and Characterisation of Microplastics in Human Breastmilk, 14 Polymers art. 2700 (2022), https://doi.org/10.3390/polym14132700, at 1, 8.
[172] Liu et al., *supra* note 168, at 1, 4.
[173] The health harms documented in this section represent a curated sample from a scientific literature that has grown substantially in recent years and continues to expand. Petitioners have focused on findings most directly relevant to ingestion, the pathway through which microplastic contamination of food and beverages reaches the human body. But the information herein is representative, not comprehensive.
[174] Chartres et al., *supra* note 9, at 22843, 22844, 22858.

A. Mechanisms of Harm

Once inside the human body, microplastics do not remain biologically inert. Rather, they interact dynamically with cells, tissues, chemicals, and microorganisms in ways that can initiate or amplify toxic effects. These interactions are shaped by the particles' physical and chemical properties, their environmental and biological history, and their capacity to serve as vectors for biological material, hazardous substances and microbial communities. Understanding these mechanisms is essential to assessing the risk they pose.

How toxic microplastics are depends on their size, shape, surface charge, and chemical properties.[175] Smaller particles, especially nanoplastics, have more surface area relative to their size and can more easily pass through barriers in the body like the intestinal lining, the placenta, and even the blood-brain barrier.[176] Their small size also makes it easier for them to enter cells. In cell-culture studies, most of which used short exposures at high concentrations, particles taken up by cells caused oxidative stress, mitochondrial damage, and membrane damage.[177] Shape also affects how toxic the particles are: irregularly shaped fragments cause more physical harm than smooth, round particles.[178] Plastic surfaces also change as the particles weather and degrade. Sunlight, physical wear, and chemical reactions make the surface rougher, create tiny cracks, and change the chemical makeup of plastics, making them more reactive and better able to attach to other substances.[179] As plastics break down into nanoscale pieces, they can reach tissues that larger particles cannot, and toxicity is expected to increase as particle size decreases.[180]

Once microplastics enter fluids in the body, they quickly attract a coating of proteins, fats, and other biological molecules to form what is called a biomolecular corona. This coating changes the particle's surface, which can affect how long it persists and how it interacts with cells. In laboratory studies, particles carrying such a coating were taken up by cells more readily than uncoated particles.[181]

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[175] Yue Li, Le Tao, Qiong Wang, Fengbang Wang, Gang Li & Maoyong Song, Potential health impact of microplastics: a review of environmental distribution, human exposure, and toxic effects, 1 Environment & Health 249–257 (2023), https://doi.org/10.1021/envhealth.3c00052, at 252; A.F.R.M. Ramsperger, J. Jasinski, M. Völkl et al., Supposedly identical microplastic particles substantially differ in their material properties influencing particle-cell interactions and cellular responses, 425 Journal of Hazardous Materials art. 127961 (2022), https://doi.org/10.1016/j.jhazmat.2021.127961, at 1–2.

[176] Klára Cverenkárová, Martina Valachovičová, Tomáš Mackuľak, Lukáš Žemlička & Lucia Bírošová, Microplastics in the Food Chain, 11 Life art. 1349 (2021), https://doi.org/10.3390/life11121349, at 12; Kornelia Kadac-Czapska, Eliza Knez & Małgorzata Grembecka, Food and human safety: the impact of microplastics, 64 Critical Reviews in Food Science and Nutrition 3502–3521 (2024), https://doi.org/10.1080/10408398.2022.2132212, at 3514; Ekaterina Brynzak-Schreiber, Elisabeth Schögl, Carolin Bapp et al., Microplastics role in cell migration and distribution during cancer cell division, 353 Chemosphere art. 141463 (2024), https://doi.org/10.1016/j.chemosphere.2024.141463, at 2.

[177] Concetta Pironti, Maria Ricciardi, Oriana Motta, Ylenia Miele, Antonio Proto & Luigi Montano, Microplastics in the environment: intake through the food web, human exposure and toxicological effects, 9 Toxics art. 224 (2021), https://doi.org/10.3390/toxics9090224, at 16; Li et al., *supra* note 175, at 252; Brynzak-Schreiber et al., *supra* note 176, at 2, 5.

[178] Li et al., *supra* note 175, at 252.

[179] Jadhav & Medyńska-Juraszek, *supra* note 42, at 6–7; Kadac-Czapska et al., *supra* note 176, at 3512–13.

[180] Kadac-Czapska et al., *supra* note 176, at 3502, 3514.

[181] Li et al., *supra* note 175, at 252; Ramsperger et al., *supra* note 175, at 2, 8–9.

Microplastics are also effective carriers for toxic substances. Their surfaces can attract and hold a wide range of persistent environmental pollutants, including polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), pesticides like DDT, pharmaceutical compounds, and heavy metals like cadmium, lead, copper, and chromium. Animal studies indicate that some of these chemicals can be released from swallowed particles in the digestive tract and taken up by the body.[182] Plastics also contain additives from manufacturing, such as bisphenol A (BPA), phthalates, flame retardants, and stabilizers, that can leak from the particle surface inside the body. These substances include known endocrine disruptors and carcinogens.[183] Ingested particles may therefore cause harm in two ways: through the particles themselves and through the chemicals they carry or release.[184]

In the environment, microbes frequently colonize plastics, attaching to their surfaces and forming stable, structured films of bacteria, known as biofilms, in what is often called the plastisphere.[185] These biofilms can include both benign and harmful microbes, such as *Vibrio cholerae*, *Escherichia coli*, *Aeromonas, Acinetobacter*, *Haemophilus*, and *Pseudomonas*.[186] The presence of a bacterial film may protect these pathogens from environmental stresses, help them survive longer, and may carry them into the body when contaminated seafood is eaten.[187] Biofilms on microplastics can also promote the transfer of genes between bacteria, including genes for antibiotic resistance, which may contribute to the broader public health problem of drug-resistant infections.[188]

These pathways provide a clear and plausible basis for concern about microplastic exposure. While the specific health outcomes are addressed in later sections of this petition, the mechanisms presented here show how these particles can cause harm once they are in the body.

B. Health Harms from Ingestion

1. Cardiovascular Effects

Microplastics have been linked to blood vessel disease, disruption of heart function, and problems with blood clotting. They have been detected in human heart and blood vessel tissue, and the studies described below found harmful changes in cells and animals and, in patients, an association with later heart attack, stroke, or death. In a prospective observational study, 257 patients who had plaque surgically removed from a carotid artery were followed for an average of 34 months. Among the 150 whose plaque contained detectable microplastics, 30 had a heart attack or stroke or died from any cause, compared with 8 of the 107 whose plaque did not. Adjusted for cardiovascular risk factors, that is a hazard ratio of 4.53 (95%

[182] Pironti et al., *supra* note 177, at 2, 17; Cverenkárová et al., *supra* note 176, at 4–5; Gabriel Enrique De-la-Torre, Microplastics: an emerging threat to food security and human health, 57 Journal of Food Science and Technology 1601–1608 (2020), https://doi.org/10.1007/s13197-019-04138-1, at 1601, 1604; Kadac-Czapska et al., *supra* note 176, at 3512–13.
[183] Cverenkárová et al., *supra* note 176, at 4–5, 13; De-la-Torre, *supra* note 182, at 1602, 1604–05; Pironti et al., *supra* note 177, at 2.
[184] Pironti et al., *supra* note 177, at 2, 16; Kadac-Czapska et al., *supra* note 176, at 3513.
[185] Cverenkárová et al., *supra* note 176, at 5; F.K. Mammo, I.D. Amoah, K.M. Gani et al., Microplastics in the environment: interactions with microbes and chemical contaminants, 743 Science of the Total Environment art. 140518 (2020), https://doi.org/10.1016/j.scitotenv.2020.140518, at 9; De-la-Torre, *supra* note 182, at 1604.
[186] Cverenkárová et al., *supra* note 176, at 6.
[187] Mammo et al., *supra* note 185, at 9–11; Cverenkárová et al., *supra* note 176, at 6; De-la-Torre, *supra* note 182, at 1604.
[188] Cverenkárová et al., *supra* note 176, at 5–6; Mammo et al., *supra* note 185, at 11.

confidence interval, 2.00 to 10.27); unadjusted, it is 2.84 (95% confidence interval, 1.50 to 5.40). The authors state that the results do not prove causality and that laboratory contamination cannot be firmly ruled out, and the patients were a single group of Italians with symptom-free carotid disease who may not represent the general population.[189] The two polymers the study measured, polyethylene and polyvinyl chloride, are also the two that a 2025 methods study found cannot currently be measured reliably in biological samples by the technique used, a limitation Section IV.B.3 addresses.[190] Another study found microplastics in 100 percent of blood samples from patients with acute coronary syndrome, with higher contamination levels matching up with more severe heart disease.[191] Those are observational findings in patients, and they cannot by themselves separate exposure from the disease it accompanies. A 2025 feeding study tested the same relationship under controlled conditions. Mice bred to develop artery disease were given polystyrene particles 1.1 micrometers across by mouth, at 10 milligrams per kilogram of body weight daily for nine weeks, while eating a low-fat, low-cholesterol diet. Plaque area at the root of the aorta was 63 percent greater in exposed male mice than in unexposed males, a statistically significant difference, with no difference in body weight, body fat, or blood cholesterol between the groups, and fluorescently labeled particles were found inside the plaques themselves. The same exposure did not significantly change plaque area in female mice. The dose was chosen to approximate human intake: the authors cite an estimate placing the mouse equivalent of human daily intake at 2.7 to 8.7 milligrams per kilogram, so the dose tested sits somewhat above that range. Because only one dose was tested, over nine weeks, the study does not show how the effect varies with dose or over a longer exposure.[192]

Microplastics contribute to hardening of the arteries, also called atherosclerotic disease. Studies using human blood vessel cells show that microplastic exposure causes significant oxidative damage and cell damage.[193] One study found some polystyrene nanoplastic particles at 20 micrograms per milliliter decreased the cells' energy production to 53 percent through damage to the mitochondria.[194] These effects impair normal function of the cells lining blood vessels and compromise the protective barrier against changes that lead to hardened arteries.[195] Smaller nanoplastic particles produce worse effects than larger microplastics: 5-micrometer polystyrene particles caused minimal effects on cells lining blood vessels, while smaller particles significantly decreased cell survival and increased harmful cellular responses.[196] Smaller, positively charged particles particularly disrupted how cells process energy and damaged cells.[197] Microplastics also promote the formation of oxidized low-density lipoproteins (LDLs), which play a

[189] Marfella et al., *supra* note 138, at 900, 905, 908.
[190] Rauert et al., *supra* note 148, at 1984, 1992.
[191] Yang et al., *supra* note 159, at 1, 5, 8.
[192] Ting-An Lin, Jianfei Pan, Mya Nguyen et al., Microplastic exposure elicits sex-specific atherosclerosis development in lean low-density lipoprotein receptor-deficient mice, 206 Environment International art. 109938 (2025), https://doi.org/10.1016/j.envint.2025.109938, at 1–2, 4, 7.
[193] Goldsworthy et al., *supra* note 139, at 746, 756.
[194] Yiqi Fu, Mengqi Fan, Liwang Xu, Hui Wang, Qinglian Hu & Yuanxiang Jin, Amino-Functionalized Polystyrene Nano-Plastics Induce Mitochondria Damage in Human Umbilical Vein Endothelial Cells, 10 Toxics art. 215 (2022), https://doi.org/10.3390/toxics10050215, at 8.
[195] Hee-Seop Lee, Darshika Amarakoon, Cheng-i Wei, Kyu Yong Choi, Dmitriy Smolensky & Seong-Ho Lee, Adverse effect of polystyrene microplastics (PS-MPs) on tube formation and viability of human umbilical vein endothelial cells, 154 Food and Chemical Toxicology art. 112356 (2021), https://doi.org/10.1016/j.fct.2021.112356.
[196] *Id.*
[197] Goldsworthy et al., *supra* note 139, at 756.

central role in hardening of the arteries.[198] Microplastic exposure has been reported to increase reactive oxygen species, unstable oxygen-containing molecules that damage cells,[199] and in a study of rats eating polystyrene it decreased protective high-density lipoproteins (HDLs) and increased harmful LDLs, although markers of oxidative stress did not change.[200] This combination makes it easier for oxidized LDL to form and for foam cells to develop within artery walls, a component of hardened plaques.[201] Immune cells in mice called macrophages exposed to nanoplastics absorb particles and accumulate fat, creating foam cells.[202] In human macrophage cells grown in culture, polystyrene particles smaller than 450 nanometers reduced cell survival at 50 to 500 micrograms per milliliter, caused oxidative stress at the highest concentration, and lowered the electrical potential across the cells' mitochondrial membranes.[203] This may contribute to unstable plaque centers that increase the risk the plaque will rupture.[204]

Laboratory studies using human blood have shown how plastic particles disrupt or increase blood clotting. Some polystyrene particles with chemically modified surfaces, such as carboxylated or amine-modified particles 100 nanometers in size, caused clotting and made clots stronger depending on their size and concentration.[205] Polystyrene nanoplastics damage a blood clotting protein called fibrinogen, with more particles causing more damage to how blood clots form.[206] The particles slow down clotting and change clot characteristics primarily through interactions with thrombin, the enzyme that converts fibrinogen into fibrin to form a clot.[207] The presence of microplastics in 80 percent of examined human clots from multiple locations may be associated with increased disease severity.[208]

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[198] Matthew Batty, Martin R. Bennett & Emma Yu, The Role of Oxidative Stress in Atherosclerosis, 11 Cells art. 3843 (2022), https://doi.org/10.3390/cells11233843, at 4; Goldsworthy et al., *supra* note 139, at 756.
[199] Goldsworthy et al., *supra* note 139, at 756.
[200] Udoka Chukwudubem Nnoruka, Chinedu Joseph Okonkwo, Ifenna Ilechukwu, Chioma Joy Okonkwo & Donatus Chuka Belonwu, Impact of polystyrene microplastic exposure on lipid profile and oxidative stress status of male and female Wistar rats, 37 Environmental Analysis, Health and Toxicology art. e2022024 (2022), https://doi.org/10.5620/eaht.2022024, at 1, 3, 5.
[201] Goldsworthy et al., *supra* note 139, at 756–757.
[202] Ida Florance, Seenivasan Ramasubbu, Amitava Mukherjee & Natarajan Chandrasekaran, Polystyrene nanoplastics dysregulate lipid metabolism in murine macrophages in vitro, 458 Toxicology art. 152850 (2021), https://doi.org/10.1016/j.tox.2021.152850, at 1.
[203] Shramana Koner, Ida Florance, Amitava Mukherjee & Natarajan Chandrasekaran, Cellular response of THP-1 macrophages to polystyrene microplastics exposure, 483 Toxicology art. 153385 (2023), https://doi.org/10.1016/j.tox.2022.153385, at 1.
[204] Goldsworthy et al., *supra* note 139, at 757; Pauline Puylaert, Michelle Zurek, Katey J. Rayner, Guido R.Y. De Meyer & Wim Martinet, Regulated Necrosis in Atherosclerosis, 42 Arteriosclerosis, Thrombosis, and Vascular Biology 1283–1306 (2022), https://doi.org/10.1161/ATVBAHA.122.318177, at 1284, 1285 fig. 1.
[205] Alexei Christodoulides, Abigail Hall & Nathan J. Alves, Exploring microplastic impact on whole blood clotting dynamics utilizing thromboelastography, 11 Frontiers in Public Health art. 1215817 (2023), https://doi.org/10.3389/fpubh.2023.1215817, at 1–2.
[206] Xiuxiu Wang, Juan Zhao, Shengli Ding & Hongyan Zhang, Interaction of polystyrene nanoplastics with human fibrinogen, 238 International Journal of Biological Macromolecules art. 124049 (2023), https://doi.org/10.1016/j.ijbiomac.2023.124049, at 1.
[207] Daniela Q. Tran, Nathan Stelflug, Abigail Hall, Tanmaye Nallan Chakravarthula & Nathan J. Alves, Microplastic Effects on Thrombin–Fibrinogen Clotting Dynamics Measured via Turbidity and Thromboelastography, 12 Biomolecules art. 1864 (2022), https://doi.org/10.3390/biom12121864, at 1, 9.
[208] Wang et al., *supra* note 160, at 1.

Microplastics directly damage heart muscle cells in several ways. Exposure of heart muscle cells to nanoplastics significantly decreased calcium levels inside cells, reduced energy production in mitochondria, and weakened contractions.[209] In mice given nanoplastics by mouth, the particles caused scarring in the heart and premature aging of heart muscle cells.[210] Studies in rats, in mice, and in laboratory-grown human heart tissue found signs of heart damage, and the mouse and heart-tissue study found thickening of the heart wall; in the heart-tissue experiments, the lowest concentration tested was chosen to match levels measured inside the human body.[211] The impact on heart development raises concerns for birth defects affecting the heart. In the same study, nanoplastic exposure reduced the stem cells' survival in a way that depended on dose and time, increased programmed cell death, damaged their mitochondria, raised oxidative stress, and reduced markers of their ability to renew themselves, and the exposed stem cells were less able to become heart muscle cells and to form heart-like tissue, with the heart muscle cells that did form showing irregular structure and weaker contraction.[212] Studies using stem cells similarly showed changes in which genes are turned on, affecting how heart valves develop.[213]

Microplastics also cause inflammation in heart and blood vessel cells. In a cell-culture study, cells from human heart arteries exposed to weathered microplastics showed signs of inflammation.[214] Exposure to sun and weather appears to make plastic materials more likely to cause inflammation. Different types of plastic caused different types of responses and inflammation depending on what chemicals they contained and their surface properties.[215] In human white blood cells exposed to polystyrene nanoplastics outside the body, different types of immune cells took up very different amounts: very little in lymphocytes and a great deal in monocytes.[216] After absorbing particles, immune cells experienced cellular stress, lower

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[209] Jingyan Li, Huimin Weng, Shuang Liu et al., Embryonic exposure of polystyrene nanoplastics affects cardiac development, 906 Science of the Total Environment art. 167406 (2024), https://doi.org/10.1016/j.scitotenv.2023.167406, at 1, 6, 8. The nanoplastic exposure was to human embryonic stem cells, from which the heart muscle cells were derived.

[210] Kaihao Wang, Yipeng Du, Peixin Li et al., Nanoplastics causes heart aging/myocardial cell senescence through the Ca2+/mtDNA/cGAS-STING signaling cascade, 22 Journal of Nanobiotechnology art. 96 (2024), https://doi.org/10.1186/s12951-024-02375-x, at 3, 20.

[211] Yue Zhou, Qian Wu, Yan Li, Yan Feng, Yan Wang & Wei Cheng, Low-dose of polystyrene microplastics induce cardiotoxicity in mice and human-originated cardiac organoids, 179 Environment International art. 108171 (2023), https://doi.org/10.1016/j.envint.2023.108171, at 1; Jialiu Wei, Xifeng Wang, Qian Liu et al., The impact of polystyrene microplastics on cardiomyocytes pyroptosis through NLRP3/Caspase-1 signaling pathway and oxidative stress in Wistar rats, 36 Environmental Toxicology 935–944 (2021), https://doi.org/10.1002/tox.23095, at 935.

[212] Li et al., *supra* note 209, at 6.

[213] Sanja Bojic, Matias M. Falco, Petra Stojkovic et al., Platform to study intracellular polystyrene nanoplastic pollution and clinical outcomes, 38 Stem Cells 1321–1325 (2020), https://doi.org/10.1002/stem.3244, at 1321–1322.

[214] T. Lomonaco, E. Persiani, D. Biagini et al., Type-specific inflammatory responses of vascular cells activated by interaction with virgin and aged microplastics, 282 Ecotoxicology and Environmental Safety art. 116695 (2024), https://doi.org/10.1016/j.ecoenv.2024.116695, at 1.

[215] *Id.* at 1, 8.

[216] Sandra Ballesteros, Josefa Domenech, Irene Barguilla, Constanza Cortés, Ricard Marcos & Alba Hernández, Genotoxic and immunomodulatory effects in human white blood cells after ex vivo exposure to polystyrene nanoplastics, 7 Environmental Science: Nano 3431–3446 (2020), https://doi.org/10.1039/D0EN00748J, at 3431.

survival rates, and died.[217] The particles caused structural changes and disrupted mitochondria.[218] Nanoplastic exposure caused DNA damage in some cell types but not others and changed many inflammation-related proteins.[219]

2. Endocrine Disruption

One of the most well-documented ways plastics harm human health is through endocrine disruption. Endocrine-disrupting chemicals are substances from outside the body that interfere with hormone signaling, leading to harmful effects on growth, metabolism, reproduction, and brain development. Among the most studied plastic-derived endocrine disruptors are BPA and phthalates. These substances are used as additives in plastic manufacturing and can leak from products into the environment and the human body through eating, breathing, and skin contact.[220] Once in the body, these chemicals can mimic or block natural hormones by binding to hormone receptors, changing hormone production, or interfering with how hormones are transported, broken down, and removed.[221]

BPA, for example, binds to estrogen receptors and can trigger estrogen-like responses even at very low doses, particularly during vulnerable windows such as fetal life and early infancy.[222] BPA can also interact with androgen and thyroid hormone receptors, disrupt receptors involved in fat metabolism, and increase aromatase activity, thereby changing sex hormone production.[223] Phthalates, including DEHP (the main plasticizer in PVC), benzyl butyl phthalate (used in foamed PVC for flooring materials), and diethyl phthalate (used in scented personal care products), have similar endocrine-disrupting properties.[224] They have been shown to stimulate estrogen-responsive gene activity, alter fat metabolism, and disrupt androgen signaling.[225] These effects are particularly pronounced during critical periods of development

[217] Koner et al., *supra* note 203, at 1.
[218] *Id.* at 1, 9.
[219] Ballesteros et al., *supra* note 216, at 3431.
[220] Philippa D. Darbre, Chemical components of plastics as endocrine disruptors: Overview and commentary, 112 Birth Defects Research 1300–1307 (2020), https://doi.org/10.1002/bdr2.1778, at 1302–03.
[221] *Id.* at 1300.
[222] *Id.* at 1302, 1304.
[223] *Id.* at 1303.
[224] *Id.* at 1303.
[225] Erin K. Maloney & David J. Waxman, trans-Activation of PPARα and PPARγ by Structurally Diverse Environmental Chemicals, 161 Toxicology and Applied Pharmacology 209–218 (1999), https://doi.org/10.1006/taap.1999.8809, at 209; Amanda Janesick & Bruce Blumberg, Minireview: PPARγ as the Target of Obesogens, 127 Journal of Steroid Biochemistry and Molecular Biology 4–8 (2011), https://doi.org/10.1016/j.jsbmb.2011.01.005; Catherine A. Harris, Pirkko Henttu, Malcolm G. Parker & John P. Sumpter, The Estrogenic Activity of Phthalate Esters in Vitro, 105 Environmental Health Perspectives 802–811 (1997), https://doi.org/10.1289/ehp.97105802; S. Jobling, T. Reynolds, R. White, M.G. Parker & J.P. Sumpter, A variety of environmentally persistent chemicals, including some phthalate plasticizers, are weakly estrogenic, 103 Environmental Health Perspectives 582–587 (1995), https://doi.org/10.1289/ehp.95103582, at 582; Kembra L. Howdeshell, Andrew K. Hotchkiss & L. Earl Gray Jr., Cumulative effects of antiandrogenic chemical mixtures and their relevance to human health risk assessment, 220 International Journal of Hygiene and Environmental Health 179–188 (2017), https://doi.org/10.1016/j.ijheh.2016.11.007.

and have been associated with reduced testosterone, genital malformations, and impaired fertility in animal models.[226]

Population-wide biomonitoring studies confirm that exposure to these endocrine disruptors is widespread. The CDC's National Health and Nutrition Examination Survey found detectable levels of BPA in 95.7 percent of urine samples collected from the U.S. population, with phthalates present in virtually all samples.[227] These chemicals are especially dangerous at low, chronic doses due to their non-monotonic dose-response curves, meaning that effects at low levels can be more severe or qualitatively different than at higher exposures.[228] This makes standard toxicological risk assessments ill-equipped to fully evaluate the dangers posed by endocrine-disrupting chemicals in plastics. Research also shows that newer bisphenol analogs, such as bisphenol S (BPS) and bisphenol F (BPF), were developed as replacements for BPA but share many of its endocrine-disrupting properties. These compounds still bind estrogen receptors and stimulate hormone-sensitive pathways in both cell-based and animal studies.[229] Their use as "BPA-free" alternatives does not guarantee reduced risk, and they may have similar potency depending on the context of exposure. Animal studies further corroborate the endocrine-disrupting potential of the plastic particles themselves. In marine medaka, a fish, exposure to 2-micrometer polystyrene microplastics at 2 to 200 micrograms per liter from the larval stage through adulthood disrupted the activity of the genes that regulate reproduction and steroid hormone production. Testosterone rose in exposed males while estradiol fell in both sexes, significantly lowering the ratio of estradiol to testosterone, and sperm counts in the testes were reduced.[230] In rats dosed by mouth, exposure to polystyrene nanoplastics and

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[226] Howdeshell et al., *supra* note 225.

[227] Hans-Joachim Lehmler, Buyun Liu, Manuel Gadogbe & Wei Bao, Exposure to Bisphenol A, Bisphenol F, and Bisphenol S in U.S. Adults and Children: The National Health and Nutrition Examination Survey 2013–2014, 3 ACS Omega 6523–6532 (2018), https://doi.org/10.1021/acsomega.8b00824, at 6525 (bisphenol A detection frequency); Darbre, *supra* note 220, at 1304 (phthalate metabolites detected in almost all human urine samples).

[228] Laura N. Vandenberg, Theo Colborn, Tyrone B. Hayes et al., Hormones and Endocrine-Disrupting Chemicals: Low-Dose Effects and Nonmonotonic Dose Responses, 33 Endocrine Reviews 378–455 (2012), https://doi.org/10.1210/er.2011-1050; Laura N. Vandenberg, Perinaaz R. Wadia, Cheryl M. Schaeberle, Beverly S. Rubin, Carlos Sonnenschein & Ana M. Soto, The mammary gland response to estradiol: Monotonic at the cellular level, non-monotonic at the tissue-level of organization?, 101 Journal of Steroid Biochemistry and Molecular Biology 263–274 (2006), https://doi.org/10.1016/j.jsbmb.2006.06.028, at 263, 268–269.

[229] Xiyan Mu, Ying Huang, Xuxing Li et al., Developmental Effects and Estrogenicity of Bisphenol A Alternatives in a Zebrafish Embryo Model, 52 Environmental Science & Technology 3222–3231 (2018), https://doi.org/10.1021/acs.est.7b06255, at 3222; Hiroyuki Kojima, Shinji Takeuchi, Seigo Sanoh et al., Profiling of bisphenol A and eight of its analogues on transcriptional activity via human nuclear receptors, 413 Toxicology 48–55 (2019), https://doi.org/10.1016/j.tox.2018.12.001, at 48; Yin Li, Lalith Perera, Laurel A. Coons et al., Differential in Vitro Biological Action, Coregulator Interactions, and Molecular Dynamic Analysis of Bisphenol A (BPA), BPAF, and BPS Ligand–ERα Complexes, 126 Environmental Health Perspectives art. 017012 (2018), https://doi.org/10.1289/EHP2505, at 017012-1.

[230] Jun Wang, Mingyi Zheng, Lin Lu, Xuefu Li, Zhenzhong Zhang & Shaoguo Ru, Adaptation of life-history traits and trade-offs in marine medaka (Oryzias melastigma) after whole life-cycle exposure to polystyrene microplastics, 414 Journal of Hazardous Materials art. 125537 (2021), https://doi.org/10.1016/j.jhazmat.2021.125537, at 5–6 (sperm count at 6, with the supporting histology at Supplementary Fig. S8A). The authors state that the exposure concentrations were environmentally relevant, citing reported microplastic concentrations in seawater of 3 to 23 micrograms per liter and up to 4,500 micrograms per liter in heavily contaminated regions. Those are concentrations measured in seawater, not estimates of human dietary intake.

polystyrene microplastics reduced testosterone, follicle-stimulating hormone, and luteinizing hormone, with lower sperm count, motility, and viability and dose-dependent damage to testicular tissue.[231]

In addition to reproductive effects, endocrine-disrupting plastics are implicated in metabolic and neurological dysfunction. BPA can interfere with thyroid hormone signaling, which is essential for brain development and metabolic regulation.[232] Plastic additives also interfere with the receptors that regulate fat metabolism. Phthalates act on peroxisome proliferator-activated receptors, which sense fats and help control how the body stores and uses them, and early-life exposure to BPA or BPS has predisposed animals to weight gain.[233] These outcomes are particularly relevant when exposures occur during gestation or early life, potentially programming the endocrine system toward chronic dysfunction that persists into adulthood.[234] Because Americans are exposed to mixtures of these chemicals, the effects on hormone signaling pathways can be additive and overlapping.[235]

3. Gastrointestinal Disruption

Microplastic exposure through ingestion creates multiple harmful effects on the digestive system, including damage to the intestinal barrier, inflammation, cellular stress, disruption of the gut bacteria, and changes in digestive function. Once microplastics reach the digestive system, larger particles above 150 micrometers remain attached to the intestinal mucus layer and directly contact the cells lining the intestine, while smaller particles can cross the mucus barrier and potentially enter the bloodstream.[236] Because the body's digestive system cannot break down plastics, the particles pass through the gut largely

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[231] Fatemeh Amereh, Mohammad Babaei, Akbar Eslami, Simin Fazelipour & Mohammad Rafiee, The emerging risk of exposure to nano(micro)plastics on endocrine disturbance and reproductive toxicity: From a hypothetical scenario to a global public health challenge, 261 Environmental Pollution art. 114158 (2020), https://doi.org/10.1016/j.envpol.2020.114158 (male rats; polystyrene nanoplastics averaging 38.92 nanometers, by oral gavage at 1, 3, 6, and 10 mg/kg/day for 35 days; reduced testosterone, luteinizing hormone and follicle-stimulating hormone, with lesion severity following the dose; held copy is an accepted manuscript without printed pagination); Muhammad Umar Ijaz, Sabahat Shahzadi, Abdul Samad et al., Dose-Dependent Effect of Polystyrene Microplastics on the Testicular Tissues of the Male Sprague Dawley Rats, 19 Dose-Response art. 15593258211019882 (2021), https://doi.org/10.1177/15593258211019882 (male Sprague Dawley rats; polystyrene microplastics by oral gavage at 2, 20, 200, and 2,000 mg/L for 60 days; reduced plasma and intra-testicular testosterone, follicle-stimulating hormone and luteinizing hormone; reduced sperm count, motility and viability; dose-dependent testicular damage).

[232] Yelena B. Wetherill, Benson T. Akingbemi, Jun Kanno et al., In vitro molecular mechanisms of bisphenol A action, 24 Reproductive Toxicology 178–198 (2007), https://doi.org/10.1016/j.reprotox.2007.05.010, at 187.

[233] Darbre, *supra* note 220, at 1303–04; Maloney & Waxman, *supra* note 225, at 209; Janesick & Blumberg, *supra* note 225 (held copy is an NIH author manuscript without printed pagination).

[234] Darbre, *supra* note 220, at 1304.

[235] *Id.* at 1305.

[236] Carlo Covello, Federica Di Vincenzo, Giovanni Cammarota & Marco Pizzoferrato, Micro(nano)plastics and Their Potential Impact on Human Gut Health: A Narrative Review, 46 Current Issues in Molecular Biology 2658–2677 (2024), https://doi.org/10.3390/cimb46030168, at 2661; Jonathan J. Powell, Nuno Faria, Emma Thomas-McKay & Laetitia C. Pele, Origin and Fate of Dietary Nanoparticles and Microparticles in the Gastrointestinal Tract, 34 Journal of Autoimmunity J226–J233 (2010), https://doi.org/10.1016/j.jaut.2009.11.006, at J230; Tamara S. Galloway, Micro- and Nano-plastics and Human Health, in Marine Anthropogenic Litter 343–366 (Melanie Bergmann, Lars Gutow & Michael Klages eds., Springer 2015), https://doi.org/10.1007/978-3-319-16510-3_13, at 351.

unaltered; most are excreted in feces, while a portion is retained in the intestine for several days and a smaller fraction crosses the gut wall into the bloodstream.[237] Ingestion of contaminated food and water is a main route by which these particles enter the body.[238]

Microplastic particles cause direct toxicity to intestinal cells through multiple mechanisms. Studies using human intestinal cells have demonstrated that polystyrene microparticles between 4.8 and 5.8 micrometers produce significant cell toxicity in both human and mouse cells at concentrations of 1 milligram per milliliter.[239] In human colon cells grown in culture, exposure to polyethylene microplastics ranging from 30 to 140 micrometers reduced cell survival to 76 percent of unexposed cells at 1,000 milligrams per liter.[240] These particles generate cellular stress in two ways: reactive molecules that form on the particle surface, and a reduction in the intestinal cells' own antioxidant and detoxification capacity. In human colon cells, polyethylene microplastics alone raised reactive oxygen species by 47 percent at 100 milligrams per liter and by 65 percent at 1,000 milligrams per liter, compared with unexposed cells.[241] The resulting increase in harmful molecules inside cells causes damage to mitochondria including increased calcium in mitochondria, loss of the electrical charge across their membranes, release of factors that promote cell death, and reduced ATP production.[242] These mitochondrial disruptions trigger cellular death, contributing to loss of the intestinal lining's integrity.[243]

The intestinal barrier serves as a critical defense system protecting against bacterial invasion and maintaining the body's internal balance, but microplastic exposure compromises this protective barrier in multiple ways. Research in mouse models has shown that exposure to polystyrene microparticles at 5 micrometers for six weeks resulted in reduced production of protective mucus and damage to the intestinal barrier.[244] Studies using polystyrene particles at both 0.5 and 50 micrometers demonstrated decreased mucus secretion in exposed animals compared to unexposed controls.[245] The reduction in this protective mucus layer leaves the intestinal surface vulnerable to mechanical damage and chemical

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[237] Covello et al., *supra* note 236, at 2661; Galloway, *supra* note 236, at 351.
[238] Covello et al., *supra* note 236, at 2659, 2660.
[239] Virginia Mattioda, Valerio Benedetti, Carlotta Tessarolo et al., Pro-Inflammatory and Cytotoxic Effects of Polystyrene Microplastics on Human and Murine Intestinal Cell Lines, 13 Biomolecules art. 140 (2023), https://doi.org/10.3390/biom13010140, at 2, 5.
[240] Wantang Huang, Hua Yin, Yuanyu Yang, Lizhu Jin, Guining Lu & Zhi Dang, Influence of the Co-exposure of Microplastics and Tetrabromobisphenol A on Human Gut: Simulation in Vitro with Human Cell Caco-2 and Gut Microbiota, 778 Science of the Total Environment art. 146264 (2021), https://doi.org/10.1016/j.scitotenv.2021.146264, at 3.
[241] Laura Rubio, Ricard Marcos & Alba Hernández, Potential Adverse Health Effects of Ingested Micro- and Nanoplastics on Humans. Lessons Learned from in Vivo and in Vitro Mammalian Models, 23 Journal of Toxicology and Environmental Health, Part B 51–68 (2020), https://doi.org/10.1080/10937404.2019.1700598, at 58; Covello et al., *supra* note 236, at 2662; Huang et al., *supra* note 240, at 5.
[242] Amrita Banerjee & Weilin L. Shelver, Micro- and Nanoplastic Induced Cellular Toxicity in Mammals: A Review, 755 Science of the Total Environment art. 142518 (2021), https://doi.org/10.1016/j.scitotenv.2020.142518, at 13.
[243] Covello et al., *supra* note 236, at 2660, 2666; Banerjee & Shelver, *supra* note 242, at 13.
[244] Yuanxiang Jin, Liang Lu, Wenqing Tu, Ting Luo & Zhengwei Fu, Impacts of Polystyrene Microplastic on the Gut Barrier, Microbiota and Metabolism of Mice, 649 Science of the Total Environment 308–317 (2019), https://doi.org/10.1016/j.scitotenv.2018.08.353, at 308, 311.
[245] Liang Lu, Zhiqin Wan, Ting Luo, Zhengwei Fu & Yuanxiang Jin, Polystyrene Microplastics Induce Gut Microbiota Dysbiosis and Hepatic Lipid Metabolism Disorder in Mice, 631–632 Science of the Total Environment 449–458 (2018), https://doi.org/10.1016/j.scitotenv.2018.03.051, at 449, 452.

irritation from digestive contents. Microplastic particles cause intestinal cells to die, which directly increases how easily substances pass through the intestinal wall. In mice given polystyrene particles of 50, 500, and 5,000 nanometers, reactive oxygen species triggered the death of cells lining the gut, and that cell death in turn increased intestinal permeability.[246] A 2026 study tested nanoplastics at doses drawn from estimates of human intake, giving mice polystyrene particles about 600 nanometers across in their drinking water for 90 days. At the lowest dose, 0.1 milligrams per kilogram of body weight daily, the protective mucus layer in the colon changed in quality and the expression of tight junction genes in the small intestine fell, although overall gut permeability did not measurably change.[247]

Microplastics trigger inflammation throughout the gastrointestinal tract. Mouse studies demonstrated that oral exposure to polyethylene microplastics ranging from 10 to 150 micrometers caused visible inflammation in both the colon and the first part of the small intestine when examined under a microscope.[248] The inflammatory response involves increased production of immune system proteins including toll-like receptor 4, activator protein 1, and interferon regulatory factor 5, indicating activation of key immune signaling pathways.[249] This microplastic exposure raises blood levels of interleukin-1 alpha, an inflammatory signaling molecule.[250] In the same study, exposure reduced two populations of immune cells in the spleen: regulatory T cells, which restrain inflammation, and Th17 cells, which drive it. The balance between the two did not change.[251] Studies of polystyrene nanoplastics at 100 nanometers in mice with chronic colitis showed that particle exposure worsened intestinal inflammation through activation of MAPK signaling pathways.[252] The inflammatory cascade triggered by microplastic exposure may contribute to chronic digestive disorders and worsen pre-existing conditions.

The large surface area of microplastic particles enables them to function as carriers for toxic substances. The particles carry chemical plasticizers including phthalates and other toxic additives that can leak from the plastic surface under the body's normal physiological conditions.[253] Research has shown that polyethylene spheres between 45 and 53 micrometers contaminated with phthalate chemicals caused

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[246] Boxuan Liang, Yizhou Zhong, Yuji Huang et al., Underestimated Health Risks: Polystyrene Micro- and Nanoplastics Jointly Induce Intestinal Barrier Dysfunction by ROS-Mediated Epithelial Cell Apoptosis, 18 Particle and Fibre Toxicology art. 20 (2021), https://doi.org/10.1186/s12989-021-00414-1, at 7.
[247] Chloé Liebgott, Melanie Mobley, Sophie Miguel et al., A Western-style diet shapes the gut and liver responses to low-dose, fit-for-purpose polystyrene nanoplastics in mice, 13 Environmental Science: Nano 405–426 (2026), https://doi.org/10.1039/D5EN00866B, at 405, 412. The study measured particles in gut contents but did not detect them in organs, so it does not establish passage out of the gut, and the effects it reports were not ordered by dose.
[248] Boqing Li, Yunfei Ding, Xue Cheng et al., Polyethylene Microplastics Affect the Distribution of Gut Microbiota and Inflammation Development in Mice, 244 Chemosphere art. 125492 (2020), https://doi.org/10.1016/j.chemosphere.2019.125492, at 1, 5, 8.
[249] *Id.*
[250] *Id.* at 4.
[251] *Id.* at 5, 8.
[252] Juan Ma, Yin Wan, Lingmin Song et al., Polystyrene Nanobeads Exacerbate Chronic Colitis in Mice Involving in Oxidative Stress and Hepatic Lipid Metabolism, 20 Particle and Fibre Toxicology art. 49 (2023), https://doi.org/10.1186/s12989-023-00560-8, at 1.
[253] John N. Hahladakis, Costas A. Velis, Roland Weber, Eleni Iacovidou & Phil Purnell, An Overview of Chemical Additives Present in Plastics: Migration, Release, Fate and Environmental Impact During Their Use, Disposal and Recycling, 344 Journal of Hazardous Materials 179–199 (2018), https://doi.org/10.1016/j.jhazmat.2017.10.014, at 184.

impaired intestinal barrier function in mouse models after 30 days of exposure.[254] The ability of microplastics to deliver chemical toxins amplifies their harmful effects on gastrointestinal health beyond the direct physical and oxidative damage caused by the particles themselves.

Microplastic exposure substantially alters the composition and function of the gut microbiota, the trillions of beneficial bacteria living in the digestive tract. The human gut microbiota comprises diverse microbial communities essential for absorbing nutrients, defending against pathogens, supporting immune system development, and regulating metabolism.[255] It is composed primarily of five bacterial phyla: *Firmicutes*, *Bacteroidetes*, *Actinobacteria*, *Proteobacteria*, and *Verrucomicrobia*.[256] Disruption of these microbial ecosystems, termed dysbiosis, has been linked to numerous digestive and body-wide disorders.[257] Multiple animal studies have documented that microplastic exposure causes dysbiosis characterized by reduced microbial diversity and altered abundance of key bacterial groups. Studies using polystyrene microparticles at 5 micrometers showed altered overall microbial diversity, with significant changes in 15 bacterial genera.[258] Research examining both 0.5 and 50 micrometer polystyrene particles demonstrated decreased relative abundance of Firmicutes and alpha-Proteobacteria in feces, along with significant changes in the richness and diversity of intestinal bacteria.[259] Exposure to polyethylene microplastics between 10 and 150 micrometers increased the total number of microbial species and bacterial abundance while altering which types were present, with significant increases in Staphylococcus (which includes some harmful species) and decreases in Parabacteroides (which is generally beneficial).[260]

The specific bacterial groups affected by microplastic exposure include species essential for maintaining intestinal health. Studies have documented increases in the bacterial phylum *Actinobacteria* and several genera including *Lactobacillus*, *Adlercreutzia*, *Butyricimonas*, and *Parabacteroides* following polyethylene microplastic exposure.[261] Changes in Proteobacteria abundance have been reported across multiple studies, with some showing increases in relative abundance of this group.[262] Microplastic exposure also shifts the balance between the two largest bacterial groups in the gut. In mice fed polyethylene terephthalate particles, *Bacteroidetes* fell by about ten percent and *Firmicutes* rose by about eleven percent, the direction of change that researchers have associated with the development of

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[254] Yongfeng Deng, Zehua Yan, Ruqin Shen et al., Microplastics Release Phthalate Esters and Cause Aggravated Adverse Effects in the Mouse Gut, 143 Environment International art. 105916 (2020), https://doi.org/10.1016/j.envint.2020.105916, at 1, 2.
[255] William Fusco, Manuel Bernabeu Lorenzo, Marco Cintoni et al., Short-Chain Fatty-Acid-Producing Bacteria: Key Components of the Human Gut Microbiota, 15 Nutrients art. 2211 (2023), https://doi.org/10.3390/nu15092211, at 2.
[256] Gregory P. Donaldson, S. Melanie Lee & Sarkis K. Mazmanian, Gut Biogeography of the Bacterial Microbiota, 14 Nature Reviews Microbiology 20–32 (2016), https://doi.org/10.1038/nrmicro3552, fig. 1.
[257] Fusco et al., *supra* note 255, at 2.
[258] Jin et al., *supra* note 244, at 308, 311.
[259] Lu et al., *supra* note 245, at 449, 452.
[260] Li et al., *supra* note 248, at 1, 4.
[261] Deng et al., *supra* note 254, at 5.
[262] Ping Jiang, Ge-hui Yuan, Bao-rong Jiang et al., Effects of Microplastics (MPs) and Tributyltin (TBT) Alone and in Combination on Bile Acids and Gut Microbiota Crosstalk in Mice, 220 Ecotoxicology and Environmental Safety art. 112345 (2021), https://doi.org/10.1016/j.ecoenv.2021.112345, at 6; Jiyan Qiao, Rui Chen, Mengjie Wang et al., Perturbation of Gut Microbiota Plays an Important Role in Micro/Nanoplastics-Induced Gut Barrier Dysfunction, 13 Nanoscale 8806–8816 (2021), https://doi.org/10.1039/d1nr00038a, at 8812; Jin et al., *supra* note 244, at 311.

obesity.[263] Research examining PET microplastics ranging from 2 to 631 micrometers found decreased abundance of *Bacteroidetes* and increased abundance of *Firmicutes*, accompanied by increased *Lactobacillus* and decreased *Parabacteroides*.[264] Microplastic exposure has been shown to reduce the abundance of bacteria that help maintain tight connections between intestinal cells, creating additional harmful effects through disruption of the gut bacterial community.[265] The changes in gut bacteria caused by microplastics may compromise the protective functions of beneficial bacteria and create conditions favorable to colonization by harmful organisms and body-wide inflammation. The direction of change is not consistent across studies: *Parabacteroides* declined after exposure to polyethylene and to polyethylene terephthalate, but rose after exposure to polyethylene carrying phthalate additives.[266]

Research indicates a correlation between the amount of microplastics in feces and the severity of inflammatory bowel disease in human populations. A study examining patients with inflammatory bowel disease found significantly higher concentrations of microplastics in the feces of patients with inflammatory bowel disease than in healthy individuals, with mean values of 41.8 particles per gram in patients versus 28.0 particles per gram in controls.[267] The microplastics detected included 15 different polymer types, with PET and polyamide representing the most abundant forms.[268] The study observed that higher microplastic concentrations correlated with more severe disease activity, suggesting that microplastic exposure may contribute to disease progression or that inflammatory conditions cause microplastics to remain longer in the intestinal tract.[269] This human data complements experimental findings in models showing that polystyrene nanoplastics worsen inflammation and intestinal injury in animals with pre-existing chronic colitis.[270]

Across human cell cultures, animal studies, and an observational study of people, microplastic exposure damages the cells lining the intestine, degrades the mucus and tight-junction barriers that protect them, provokes inflammation, and disrupts the bacterial communities the gut depends on. The one systematic review to grade this literature rated the evidence that microplastics suppress digestive immune function as high quality, and concluded that microplastic exposure is suspected to be a digestive hazard to humans, including a suspected link to colon cancer.[271] Section 402(a)(1) of the Act does not require more. It asks whether a substance may render food injurious to health, and evidence of this kind and strength answers that question.

4. Liver Toxicity

The liver is a major site of microplastic accumulation following ingestion, and exposure to these particles produces harmful effects on liver cells through several biological pathways. Research using human liver cells has consistently shown that microplastic particles cause oxidative stress, disrupt the liver's energy

[263] Jin Liu, Min Lv, Anqi Sun et al., Exposure to Microplastics Reduces the Bioaccumulation of Sulfamethoxazole but Enhances Its Effects on Gut Microbiota and the Antibiotic Resistome of Mice, 294 Chemosphere art. 133810 (2022), https://doi.org/10.1016/j.chemosphere.2022.133810, at 3.
[264] *Id.* at 2, 3, 5.
[265] Qiao et al., *supra* note 262, at 8806.
[266] Li et al., *supra* note 248, at 4; Liu et al., *supra* note 263, at 5; Deng et al., *supra* note 254, at 5.
[267] Yan et al., *supra* note 155, at 414.
[268] *Id.*
[269] *Id.*
[270] Ma et al., *supra* note 252, at 1.
[271] Chartres et al., *supra* note 9, at 22843, 22854.

production systems, interfere with fat processing, and trigger inflammatory responses.[272] These effects occur in ways that depend on both the size of the particles and the amount of exposure, with smaller nanoplastic particles generally producing more severe damage than larger microplastic particles.[273]

Studies using human liver cell lines have documented that microplastic exposure generates excessive amounts of harmful molecules called reactive oxygen species within liver cells.[274] This oxidative stress damages the mitochondria, leading to changes in their structure, loss of their ability to maintain proper electrical charge across their membranes, and reduced activity of key energy-producing components.[275] These mitochondrial problems restrict the liver's ability to produce ATP, and create structural abnormalities in the internal compartments of mitochondria.[276] The resulting energy shortage impairs the liver's capacity to carry out its normal detoxification and metabolic functions.

Microplastic exposure also disrupts how the liver processes and stores fats. Multiple studies have reported that exposure to these particles causes fat to build up inside liver cells, potentially speeding up the development of fatty liver disease.[277] In liver organoids grown from human stem cells, a 72-hour exposure to polystyrene microplastics interfered with the activity of multiple genes responsible for controlling fat metabolism and the production of associated proteins, resulting in fat accumulation.[278] In a 90-day drinking-water study in mice, more fat accumulated in the liver and the fat droplets changed in size on both a standard and a Western-style diet.[279] Other studies using these organoids found that microplastic exposure altered the processing of sulfur-containing amino acids, disrupted iron balance in liver tissue,

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[272] Zahra Beyzaei, Bita Geramizadeh, Zahra Bagheri, Sara Karimzadeh & Ralf Weiskirchen, Microplastics in focus: a silent disruptor of liver health — a systematic review, 16 Frontiers in Pharmacology art. 1721644 (2025), https://doi.org/10.3389/fphar.2025.1721644, at 1, 5, 10–11.
[273] *Id.* at 7.
[274] *Id.* at 5, 7.
[275] *Id.* at 10.
[276] Siyi Lin, Hongna Zhang, Chen Wang et al., Metabolomics Reveal Nanoplastic-Induced Mitochondrial Damage in Human Liver and Lung Cells, 56 Environmental Science & Technology 12483–12493 (2022), https://doi.org/10.1021/acs.est.2c03980, at 12483, 12488 (human liver cell line, 80 nm particles); Wei Cheng, Hange Chen, Yue Zhou et al., Aged fragmented-polypropylene microplastics induced ageing statues-dependent bioenergetic imbalance and reductive stress: in vivo and liver organoids-based in vitro study, 191 Environment International art. 108949 (2024), https://doi.org/10.1016/j.envint.2024.108949, at 1, 6 (liver organoids grown from human stem cells, and mice).
[277] Yujie Shi, Runyang Hong, Zhencheng Fan et al., Chronic environmental exposure to polystyrene microplastics increases the risk of nonalcoholic fatty liver disease, 511 Toxicology art. 154067 (2025), https://doi.org/10.1016/j.tox.2025.154067, at 1 (mice given 10 mg/L in drinking water for six months, and human liver cells at 25 μg/mL); Jiwon Ahn, Kajung Ryu, Hyerin Kim et al., Polystyrene Nanoplastics Exacerbate HFD-induced MASLD by Reducing Cathepsin Activity and Triggering Large Vacuole Formation via Impaired Lysosomal Acidification, 21 International Journal of Biological Sciences 3867–3885 (2025), https://doi.org/10.7150/ijbs.108268, at 3867 (human liver cell lines, and mice on a high-fat diet); Albert Menéndez-Pedriza, Joaquim Jaumot & Carmen Bedia, Lipidomic analysis of single and combined effects of polyethylene microplastics and polychlorinated biphenyls on human hepatoma cells, 421 Journal of Hazardous Materials art. 126777 (2022), https://doi.org/10.1016/j.jhazmat.2021.126777, at 1 (human liver cell culture).
[278] Beyzaei et al., *supra* note 272, at 8–9 (reporting that 500 to 1,000 ng/mL polystyrene microplastics over 72 hours interfered with gene panels regulating lipid metabolism and the associated protein expression in liver organoids grown from human stem cells, producing significant lipid accumulation).
[279] Liebgott et al., *supra* note 247, at 405, 414.

and increased fat breakdown products.[280] Aged microplastic particles that have undergone environmental weathering showed higher toxicity compared to newly manufactured particles, disturbing the liver's chemical balance further and reducing its energy supply.[281]

Beyond oxidative damage and fat accumulation, microplastics trigger inflammatory responses and cause liver cell death. Studies have documented increased levels of inflammatory signaling molecules and activation of cell death pathways in liver cells exposed to these particles.[282] At higher exposure levels and with prolonged contact, microplastics caused direct toxic effects including cell death through multiple mechanisms and the formation of abnormal structures in cell nuclei that can lead to scarring of liver tissue.[283]

Limited available evidence from human tissue studies provides support for these laboratory findings. Analysis of liver samples from patients with cirrhosis detected microplastic particles at concentrations ranging from 4.6 to 11.9 particles per gram of tissue (3.2 to 9.9 after correction for blank samples), while all liver, kidney, and spleen samples from the five individuals without liver disease tested negative, considering the limit of detection.[284] This suggests that diseased livers may accumulate more plastic particles than healthy livers, though whether this accumulation contributes to disease progression or results from the disease itself remains unclear. A prospective observational study of 1,057 pregnancies found that maternal microplastic exposure was associated with elevated levels of liver enzymes in umbilical cord blood, including alkaline phosphatase, aspartate aminotransferase, and gamma-glutamyl

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[280] Wei Cheng, Yue Zhou, Hange Chen et al., The iron matters: Aged microplastics disrupted the iron homeostasis in the liver organoids, 906 Science of the Total Environment art. 167529 (2024), https://doi.org/10.1016/j.scitotenv.2023.167529, at 1, 2 (liver organoids grown from human stem cells; aged microplastics at 50 ng/mL, which the authors state is equivalent to the human intake level, disrupted the genes handling iron transport, storage, and use); Beyzaei et al., *supra* note 272, at 1, 8–9.

[281] Cheng et al., *supra* note 276, at 1, 6; Cheng et al., *supra* note 280, at 1, 2; Beyzaei et al., *supra* note 272, at 8–9.

[282] Yuhan Li, Yulun Jian, Jinjin Zhou et al., Molecular regulatory networks of microplastics and cadmium mediated hepatotoxicity from NAFLD to tumorigenesis via integrated approaches, 300 Ecotoxicology and Environmental Safety art. 118431 (2025), https://doi.org/10.1016/j.ecoenv.2025.118431, at 1; Asha Guraka, Graham Souch, Richard Duff, David Brown, Wolfgang Moritz & Ali Kermanizadeh, Microplastic-induced hepatic adverse effects evaluated in advanced quadruple cell human primary models following three weeks of repeated exposure, 364 Chemosphere art. 143032 (2024), https://doi.org/10.1016/j.chemosphere.2024.143032, at 1; Tugce Boran, Ozge Sultan Zengin, Zehra Seker et al., An evaluation of a hepatotoxicity risk induced by the microplastic polymethyl methacrylate (PMMA) using HepG2/THP-1 co-culture model, 31 Environmental Science and Pollution Research 28890–28904 (2024), https://doi.org/10.1007/s11356-024-33086-3, at 28890; Amrita Banerjee, Lloyd O. Billey, Amy M. McGarvey & Weilin L. Shelver, Effects of polystyrene micro/nanoplastics on liver cells based on particle size, surface functionalization, concentration and exposure period, 836 Science of the Total Environment art. 155621 (2022), https://doi.org/10.1016/j.scitotenv.2022.155621; Rong Shen, Keren Yang, Xin Cheng et al., Accumulation of polystyrene microplastics induces liver fibrosis by activating cGAS/STING pathway, 300 Environmental Pollution art. 118986 (2022), https://doi.org/10.1016/j.envpol.2022.118986, at 1.

[283] Shen et al., *supra* note 282, at 1, 3–4 (human liver cell line, 100 nm particles at 1,000 μg/L); Beyzaei et al., *supra* note 272, at 7, 11.

[284] Horvatits et al., *supra* note 143, at 1, 4 (proof-of-concept case series: liver tissue from six patients with cirrhosis and liver, kidney, and spleen tissue from five individuals without underlying liver disease; blank samples were analyzed alongside each run, and the cirrhosis range of 4.6 to 11.9 particles per gram corresponds to 3.2 to 9.9 particles per gram after blank correction).

transferase.[285] The study measured particles in the placenta rather than in the fetus, and reports an association.

The available evidence demonstrates that microplastic exposure harms the liver through interconnected mechanisms involving oxidative damage, energy disruption, fat metabolism problems, and inflammatory responses. These effects have been documented across multiple types of human liver cells and tissue models, though most of that work used concentrations above those measured in human liver tissue.

5. Metabolic Disorders

A 2025 review of 127 studies, most of them in cells and animals, concluded that microplastic exposure may impair glucose regulation and contribute to insulin resistance, while noting that evidence in people remains limited.[286] Studies in zebrafish larvae and in mice have found that polystyrene nanoplastics disrupt glucose metabolism.[287] In mice fed a high-fat diet, exposure to microplastics raised fasting blood glucose and insulin resistance more than the diet alone.[288]

Studies examining glucose metabolism show that both short-term and long-term microplastic exposure alters blood glucose levels in experimental models.[289] Mice given polystyrene nanoplastics by mouth developed elevated fasting blood glucose compared with controls.[290] Surface chemistry influences toxicity: in mice, positively charged amino-modified particles disrupted glucose regulation more than either pristine or negatively charged carboxyl-modified particles.[291] These disruptions result in impaired glucose tolerance and insulin tolerance alongside elevated fasting blood glucose levels.[292]

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[285] Chuanzhuo Wang, Hua Chang, Huan Wang, Hui Li, Silu Ding & Fang Ren, Exposure to microplastics during pregnancy and fetal liver function, 294 Ecotoxicology and Environmental Safety art. 118099 (2025), https://doi.org/10.1016/j.ecoenv.2025.118099, at 1.

[286] Yuqing Song, Shun Lyu, Ruoxi Wang et al., Micro- and nanoplastic exposure and metabolic disruption: A review on their emerging role in the pathogenesis of diabetes, 40 NanoImpact art. 100590 (2025), https://doi.org/10.1016/j.impact.2025.100590, at 1, 5.

[287] Nadja R. Brun, Patrick van Hage, Ellard R. Hunting et al., Polystyrene nanoplastics disrupt glucose metabolism and cortisol levels with a possible link to behavioural changes in larval zebrafish, 2 Communications Biology art. 382 (2019), https://doi.org/10.1038/s42003-019-0629-6, at 1 (zebrafish larvae); Yunyi Wang, Ke Xu, Xiao Gao et al., Polystyrene nanoplastics with different functional groups and charges have different impacts on type 2 diabetes, 21 Particle and Fibre Toxicology art. 21 (2024), https://doi.org/10.1186/s12989-024-00582-w, at 1 (mice, 80 nm particles at 5 mg/kg of body weight daily for nine weeks).

[288] Song et al., *supra* note 286, at 5, 9 (collecting in vivo studies in which mice fed a high-fat diet and exposed to microplastics showed greater increases in fasting blood glucose and in insulin resistance than mice fed a high-fat diet alone).

[289] *Id.* at 5.

[290] Xingpei Fan, Xiangjuan Wei, Hailong Hu et al., Effects of oral administration of polystyrene nanoplastics on plasma glucose metabolism in mice, 288 Chemosphere art. 132607 (2022), https://doi.org/10.1016/j.chemosphere.2021.132607, at 1, 3 (mice, 5 and 15 mg/kg of body weight); Dingjie Huang, Ying Zhang, Jianglan Long et al., Polystyrene microplastic exposure induces insulin resistance in mice via dysbacteriosis and pro-inflammation, 838 Science of the Total Environment art. 155937 (2022), https://doi.org/10.1016/j.scitotenv.2022.155937, at 1, 3.

[291] Wang et al., *supra* note 287, at 1, 7 (mice, 80 nm particles at 5 mg/kg of body weight daily for nine weeks).

[292] Song et al., *supra* note 286, at 5.

Insulin resistance represents a consistently documented outcome of microplastic exposure across multiple studies.[293] Measurements of insulin resistance show significantly higher levels in microplastic-exposed animals, indicating insulin problems throughout the body.[294] This resistance worsens with increasing particle doses and longer exposure times.[295] Analysis shows that microplastic exposure disrupts insulin signaling, with abnormal changes to the proteins that carry the insulin signal inside liver cells.[296]

Microplastic exposure substantially disrupts liver metabolism, with particles accumulating in liver tissue and triggering fat buildup.[297] Exposed mice develop more fat in the liver, along with inflammation and oxidative stress.[298] Microplastics also disturb the gut–liver axis, blunting the insulin signaling that normally governs how the liver stores and uses energy.[299]

Pancreatic function deteriorates under microplastic exposure through mechanisms that damage insulin-producing cells.[300] In mice given 80-nanometer particles daily for nine weeks, the insulin-producing clusters of cells in the pancreas lost their normal structure, and the damage was worst with the positively charged particles.[301] In obese mice on a high-fat diet, polystyrene microplastics given by mouth worsened the mice's already impaired glucose metabolism and insulin resistance, and promoted systemic inflammation.[302]

Diet strongly affects how toxic microplastics are, with high-fat diets making metabolic harm worse through combined effects. Animals eating high-fat diets while exposed to microplastics show significantly greater increases in fasting blood sugar and insulin resistance compared to those receiving microplastics or high-fat diet alone.[303] This combination makes inflammation worse, speeds up fat

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[293] *Id.* at 8.
[294] Huang et al., *supra* note 290, at 1, 3, 5 (mice given 80 mg/kg of body weight daily by gavage for ten weeks, a dose the authors state is equivalent to 4.3 grams per week in a human); Wang et al., *supra* note 287, at 1, 7 (mice, 80 nm particles at 5 mg/kg of body weight daily for nine weeks).
[295] Fan et al., *supra* note 290, at 1, 3 (mice given polystyrene nanoplastics by mouth at 5 and 15 mg/kg of body weight; fasting plasma glucose rose significantly in both dose groups from week 16 onward, and glucose stayed elevated through the insulin tolerance test only in the 15 mg/kg group).
[296] *Id.* at 1, 3, 8.
[297] Song et al., *supra* note 286, at 6, 9.
[298] Huang et al., *supra* note 290, at 1, 3; Chunzhen Shi, Xiaohong Han, Wei Guo et al., Disturbed Gut-Liver axis indicating oral exposure to polystyrene microplastic potentially increases the risk of insulin resistance, 164 Environment International art. 107273 (2022), https://doi.org/10.1016/j.envint.2022.107273, at 4–5.
[299] Shi et al., *supra* note 298, at 1 (mice).
[300] Song et al., *supra* note 286, at 5.
[301] Wang et al., *supra* note 287, at 1, 8 (mice, 5 mg/kg of body weight daily for nine weeks); Manjin Xu, Huixia Niu, Lizhi Wu et al., Impact of Microplastic Exposure on Blood Glucose Levels and Gut Microbiota: Differential Effects under Normal or High-Fat Diet Conditions, 14 Metabolites art. 504 (2024), https://doi.org/10.3390/metabo14090504, at 1, 4.
[302] Aden Geonhee Lee, Sora Kang, Hye Ji Yoon, Suyeol Im, Seung Jun Oh & Youngmi Kim Pak, Polystyrene Microplastics Exacerbate Systemic Inflammation in High-Fat Diet-Induced Obesity, 24 International Journal of Molecular Sciences art. 12421 (2023), https://doi.org/10.3390/ijms241512421, at 1 (mice on a high-fat diet).
[303] Ziye Yang, Huajiang Dong, Yifei Gao et al., Airborne Nanoplastics Exposure Inducing Irreversible Glucose Increase and Complete Hepatic Insulin Resistance, 58 Environmental Science & Technology 3108–3117 (2024), https://doi.org/10.1021/acs.est.3c06468, at 3108 (whole-body inhalation exposure in mice, not oral exposure); Xu et al., *supra* note 301, at 1, 4 (mice); Takuro Okamura, Masahide

buildup in the liver, and disrupts gut barrier function more severely than either exposure alone.[304] A 90-day study gave mice polystyrene nanoplastics in their drinking water at doses the authors chose to approximate estimated human intake. Glucose tolerance worsened on a Western-style diet at 1 milligram per kilogram of body weight daily, while the same exposure produced no comparable effect on standard feed. The effects did not rise steadily with dose.[305] In one study, mice given polystyrene by mouth while on a high-fat diet developed worse insulin resistance, larger fat cells, and more fat in the liver than mice on the high-fat diet alone.[306]

6. Additional Systemic Health Effects

Beyond the organ systems discussed above, studies in animals have found effects of microplastic exposure on immune function, the nervous system, and reproduction.

Microplastic particles trigger immune responses throughout the body following ingestion. In adult zebrafish held for seven days in water containing polyethylene microplastics, expression of the genes that make up the intestinal immune network for mucosal immunoglobulin production increased. The effect appeared at the two highest concentrations tested, 100 and 1,000 micrograms per milliliter.[307] Exposure to polyethylene increased release of the inflammatory molecule interleukin-1 alpha and reduced the share of regulatory T cells and Th17 cells among helper T cells.[308]

Microplastic exposure causes brain and nerve toxicity through processes that impair neural development and function. Inhibition of acetylcholinesterase activity, an enzyme important for nerve signaling, is among the most frequently reported nerve-damaging effects following microplastic exposure in bivalves, crustaceans and fish. The review that reports this notes that data from mammals given microplastics are scarce, and that differences in how aquatic animals and mammals are exposed make the comparison an imperfect one.[309] A study of juvenile European seabass, a marine fish eaten by people, found that microplastic exposure in the surrounding water inhibits acetylcholinesterase activity, increases fat oxidation in brain tissue, and changes the activity of energy-related enzymes, ultimately producing nerve toxicity.[310] Chronic exposure to polystyrene at environmentally relevant concentrations of 100 and 1,000

Hamaguchi, Yuka Hasegawa et al., Oral Exposure to Polystyrene Microplastics of Mice on a Normal or High-Fat Diet and Intestinal and Metabolic Outcomes, 131 Environmental Health Perspectives art. 027006 (2023), https://doi.org/10.1289/EHP11072, at 1 (mice); Huang et al., *supra* note 290, at 1, 3, 5 (mice given 80 mg/kg of body weight daily by gavage for ten weeks, a dose the authors state is equivalent to 4.3 grams per week in a human).

[304] Yang et al., *supra* note 303, at 3108 (whole-body inhalation exposure in mice, not oral exposure); Okamura et al., *supra* note 303, at 1 (mice).

[305] Liebgott et al., *supra* note 247, at 405, 414.

[306] Huang et al., *supra* note 290, at 3–4.

[307] Yin Yuan, Marisol S. Sepúlveda, Baoliang Bi et al., Acute polyethylene microplastic (PE-MPs) exposure activates the intestinal mucosal immune network pathway in adult zebrafish (Danio rerio), 311 Chemosphere art. 137048 (2023), https://doi.org/10.1016/j.chemosphere.2022.137048, at 2.

[308] Li et al., *supra* note 248, at 1, 5, 8.

[309] Minne Prüst, Jonelle Meijer & Remco H. S. Westerink, The plastic brain: neurotoxicity of micro- and nanoplastics, 17 Particle and Fibre Toxicology art. 24 (2020), https://doi.org/10.1186/s12989-020-00358-y, at 11–12 (pages 11–12 of 16).

[310] Luís Gabriel Antão Barboza, Luís Russo Vieira, Vasco Branco et al., Microplastics cause neurotoxicity, oxidative damage and energy-related changes and interact with the bioaccumulation of mercury in the European seabass, Dicentrarchus labrax (Linnaeus, 1758), 195 Aquatic Toxicology 49–57 (2018), https://doi.org/10.1016/j.aquatox.2017.12.008, at 49, 51.

micrograms per liter damages the blood-brain barrier and causes learning and memory problems in mice.[311]

Microplastic exposure produces reproductive and developmental toxicity through effects on reproductive cell development and embryo quality. Research shows that polystyrene exposure affects development of female follicles and maturation of egg cells, reducing egg quality in mice.[312] Studies indicate that microplastics may cause harmful effects on pregnancy outcomes by causing immune system disorders; the study cited delivered the particles by injection rather than by mouth.[313] A 2024 systematic review examined five rodent studies of sperm and sperm-related outcomes after oral exposure to polystyrene. It found consistent declines in living sperm, sperm concentration and sperm motility, and increases in sperm malformation, and rated the quality of that body of evidence "high," the review's top rating.[314] In mice given polyethylene microplastics by mouth for 90 days, the number of live births per dam fell and the sex ratio of the pups shifted, and pups in the highest-dose group weighed less at birth than controls. The effects did not follow the dose: pups in the lowest-dose group weighed more than controls, and the authors state that the shift in sex ratio was not dose-dependent.[315] In that study, the treated mothers also had significantly higher blood levels of immunoglobulin A, an antibody, and the authors suggested that immunoglobulin A levels may serve as a marker for harmful effects following microplastic exposure.[316] These animal studies point to possible effects on reproduction and on the next generation; Section IV.B.3 describes a 2025 review that rated the mammalian studies of these outcomes at high risk of bias.

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[311] Haibo Jin, Chen Yang, Chengyue Jiang et al., Evaluation of Neurotoxicity in BALB/c Mice following Chronic Exposure to Polystyrene Microplastics, 130 Environmental Health Perspectives art. 107002 (2022), https://doi.org/10.1289/EHP10255, at 107002-1. Male BALB/c mice were given drinking water containing polystyrene particles of 0.5, 4, and 10 micrometers for 180 consecutive days.

[312] Zhiqiang Liu, Qingrui Zhuan, Luyao Zhang, Lin Meng, Xiangwei Fu & Yunpeng Hou, Polystyrene microplastics induced female reproductive toxicity in mice, 424 Journal of Hazardous Materials art. 127629 (2022), https://doi.org/10.1016/j.jhazmat.2021.127629, at 1, 4. Female mice were given polystyrene microplastics by oral gavage at 30 milligrams per kilogram of body weight daily for 35 days. The number of antral follicles was significantly reduced; the numbers of primordial, primary and secondary follicles were not.

[313] Jianing Hu, Xiaoli Qin, Jinwen Zhang et al., Polystyrene microplastics disturb maternal-fetal immune balance and cause reproductive toxicity in pregnant mice, 106 Reproductive Toxicology 42–50 (2021), https://doi.org/10.1016/j.reprotox.2021.10.002, at 42, 49. Pregnant mice received 250 micrograms of polystyrene by intraperitoneal injection on two days of gestation. The authors state that they chose that route over the oral route to shorten the exposure window, that this was not a physiological route of exposure, and that the total dose was above environmental concentrations. *Id.* at 49.

[314] Chartres et al., *supra* note 9, at 22843, 22855.

[315] Eun-Jung Park, Ji-Seok Han, Eun-Jun Park et al., Repeated-oral dose toxicity of polyethylene microplastics and the possible implications on reproduction and development of the next generation, 324 Toxicology Letters 75–85 (2020), https://doi.org/10.1016/j.toxlet.2020.01.008, at 75, 79, 83 tbl.3. Mice were given polyethylene microplastics of about 17 micrometers by gavage at 0.125, 0.5, and 2 milligrams per day for 90 days; five mice of each sex per dose were mated for the reproductive screen. Seneca Fitch, John Rogers, Sue Marty et al., Systematic review of potential developmental and reproductive toxicity of microplastics, 207 Toxicological Sciences 289–305 (2025), https://doi.org/10.1093/toxsci/kfaf108, at 299, 302, rates this study at high risk of bias, noting the absence of a dose trend in pup body weight and inconsistent reporting of the number of animals per group. That review was supported by the American Chemistry Council, which was involved in conceptualizing it and reviewed the final manuscript, and four of its authors are employed by companies that produce plastics.

[316] Park et al., *supra* note 315, at 75.

C. Heightened Risk to Children

Children face unique and heightened risks from microplastic contamination in food and beverages. In November 2025, a majority of the members of EPA's Children's Health Protection Advisory Committee ("CHPAC") conveyed to EPA Administrator Zeldin that "the science is clear that plastic pollution can harm children's health and raise risks of developmental disabilities, birth defects, cancers, and other serious diseases."[317] Early-life exposures to microplastics through contaminated food and beverages may contribute to disease burden throughout the lifespan.[318] The Committee applied the seven health hazards of highest concern for children's health that it had identified previously: reproductive toxicity, developmental toxicity including developmental neurotoxicity, carcinogenicity, endocrine toxicity including metabolism-disrupting chemicals, respiratory toxicity and potential effects on lung development, immunotoxicity, and toxicity through preconception or in utero exposures.[319] These hazard categories encompass the range of potential health impacts from dietary exposure to microplastics and their associated chemicals during pregnancy and early childhood. There is a heightened risk to children because exposure begins before birth and continues through infancy, which Section II.C documents; the developing endocrine and nervous systems are more sensitive to interference than adult systems; and plastic-associated chemicals are associated with harm to children in the epidemiological literature set out below.[320]

Children are especially vulnerable to plastic-associated chemical exposures for biological reasons. Endocrine-disrupting chemicals interfere with the natural hormones responsible for growth, reproduction, and developmental processes.[321] The endocrine signaling system governs all facets of early human development, including physical growth, organ formation, and development of the brain, reproductive system, and immune system. Exposures during pregnancy and in the first two years after birth are particularly dangerous because they can cause changes within developing cells and organs that disrupt organ formation and increase risk of disease and disability in childhood and across the lifespan.[322]

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[317] CHPAC Letter A, *supra* note 8, at 1. Letter A is the response of 23 of the Committee's 25 members; one member submitted a separate letter, Letter B, in the same document. Letter A defines plastic pollution to encompass the full life cycle of plastics, including plastic-associated chemicals as well as microplastics. *Id.*

[318] Commission Report at 89.

[319] CHPAC Letter A, *supra* note 8, at 2.

[320] Chartres et al., *supra* note 9, at 22858. A single member of the Committee submitted a separate letter questioning this review, on the ground that it reported identifying no studies of microplastic exposure using human subjects. That statement appears in the review's description of how it amended its eligibility criteria after its first database search in July 2022; the authors searched again in April 2024, and the published review includes three human observational studies. *Id.* at 22843, 22848. The same letter describes the review's sensitivity analysis as showing no statistically significant differences across multiple domains; what the review reports is that its findings did not change when polystyrene and polypropylene results were separated or when only one result per study was counted, which is a test of robustness rather than a null result. *Id.* at 22858. Children's Health Protection Advisory Committee, Letter B to Administrator Lee Zeldin, Plastics and Children's Health Charge Response (Nov. 4, 2025), U.S. EPA Docket No. EPA-HQ-OA-2025-0261-0029, at 2.

[321] Commission Report at 79.

[322] Maria Elisabeth Street & Sergio Bernasconi, Endocrine-disrupting chemicals in human fetal growth, 21 International Journal of Molecular Sciences art. 1430 (2020), https://doi.org/10.3390/ijms21041430, at 5; Commission Report at 79.

Endocrine-disrupting chemicals in a pregnant woman's body can be transmitted to her child through the placenta during pregnancy and through breast milk during lactation.[323]

The developing brain is especially sensitive to toxic chemical exposures from food. Brain development begins shortly after conception when cells start dividing and multiplying rapidly, ultimately producing approximately 100 billion neurons by birth.[324] The complexity of brain development creates extreme vulnerability to toxic chemicals and other harmful environmental exposures.[325] Exposures to even small doses of toxic chemicals during critical developmental stages can have lifelong consequences for brain function and neurological health.[326] These windows of vulnerability are unparalleled in adults, and brain damage caused by exposure to toxic chemicals early in life may be chronic, irreversible, and difficult to treat.[327]

Evidence shows plastic-associated chemicals are neurotoxic. Prenatal exposures to PCBs are associated with lower intelligence quotient scores and difficulties with motor skills, attention, and memory.[328] Prenatal exposure to phthalates has been associated with increased risk for autism spectrum disorder, aggression, depression, conduct and attention problems, externalization of problems, and diminished

[323] Sudipta Dutta, Diana K. Haggerty, Daniel A. Rappolee & Douglas M. Ruden, Phthalate exposure and long-term epigenomic consequences: a review, 11 Frontiers in Genetics art. 405 (2020), https://doi.org/10.3389/fgene.2020.00405, at 14; Anne Denuzière & Jean-François Ghersi-Egea, Cerebral concentration and toxicity of endocrine disrupting chemicals: the implication of blood-brain interfaces, 91 NeuroToxicology 100–118 (2022), https://doi.org/10.1016/j.neuro.2022.04.004.
[324] Commission Report at 80.
[325] Commission Report at 80.
[326] Commission Report at 80.
[327] Commission Report at 80.
[328] Audrey-Anne Ethier, Gina Muckle, Sandra Jacobson, Pierre Ayotte, Joseph Jacobson & Dave Saint-Amour, Assessing new dimensions of attentional functions in children prenatally exposed to environmental contaminants using an adapted Posner paradigm, 51 Neurotoxicology and Teratology 27–34 (2015), https://doi.org/10.1016/j.ntt.2015.07.005; Joseph L. Jacobson, Sandra W. Jacobson & Harold E.B. Humphrey, Effects of exposure to PCBs and related compounds on growth and activity in children, 12 Neurotoxicology and Teratology 319–326 (1990), https://doi.org/10.1016/0892-0362(90)90050-M; Sietske A. Berghuis, Shalini D. Soechitram, Marrit M. Hitzert, Pieter J.J. Sauer & Arend F. Bos, Prenatal exposure to polychlorinated biphenyls and their hydroxylated metabolites is associated with motor development of three-month-old infants, 38 NeuroToxicology 124–130 (2013), https://doi.org/10.1016/j.neuro.2013.07.003; Hestien J.I. Vreugdenhil, Caren I. Lanting, Paul G.H. Mulder, E. Rudy Boersma & Nynke Weisglas-Kuperus, Effects of prenatal PCB and dioxin background exposure on cognitive and motor abilities in Dutch children at school age, 140 The Journal of Pediatrics 48–56 (2002), https://doi.org/10.1067/mpd.2002.119625; Svati Patandin, Caren I. Lanting, Paul G.H. Mulder, E. Rudy Boersma, Pieter J.J. Sauer & Nynke Weisglas-Kuperus, Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age, 134 The Journal of Pediatrics 33–41 (1999), https://doi.org/10.1016/S0022-3476(99)70369-0.

executive functioning.[329] Certain phthalates are associated with poorer motor skills in children.[330] Mothers with higher exposure to phthalates during pregnancy are three times more likely than other women to have children diagnosed with attention deficit hyperactivity disorder.[331] In a study of prenatal phthalate exposure and cognitive function among 328 mothers and their children, the children whose mothers had the highest concentration of certain phthalates scored significantly lower on both perceptual reasoning (by 3.9 points) and verbal comprehension (by 4.4 points).[332] In the same study, children born to women in the top quarter of exposure to two phthalates scored 6.6 and 7.6 points lower on full-scale IQ at age seven than children born to women in the bottom quarter.[333] These findings suggest that the adverse impacts of prenatal phthalate exposure on cognitive function can persist into the early school years, negatively impacting academic performance.[334]

CHPAC's analysis of the health harms children experience from plastic identified several specific chemicals tied to poor outcomes. Bisphenol A is associated with changes in children's reproductive development, type 2 diabetes, insulin resistance, polycystic ovary syndrome, obesity, hypertension, and cardiovascular disease.[335] The Committee reported similar concerns for the bisphenols used in its place, including BPS and BPF.[336] Phthalates are associated with increased risks of pregnancy complications including miscarriages, preterm birth, hypertensive disorders of pregnancy, and decreased birth weight. These chemicals are also associated with changes in children's reproductive development, precocious puberty in girls, adverse effects on child neurodevelopment, insulin resistance, elevated blood pressure,

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[329] Maede Ejaredar, Elias C. Nyanza, Kayla Ten Eycke & Deborah Dewey, Phthalate exposure and childrens neurodevelopment: a systematic review, 142 Environmental Research 51–60 (2015), https://doi.org/10.1016/j.envres.2015.06.014; Anne-Louise Ponsonby, Christos Symeonides, Richard Saffery et al., Prenatal phthalate exposure, oxidative stress-related genetic vulnerability and early life neurodevelopment: a birth cohort study, 80 NeuroToxicology 20–28 (2020), https://doi.org/10.1016/j.neuro.2020.05.006; Elizabeth G. Radke, Joseph M. Braun, Rebecca M. Nachman & Glinda S. Cooper, Phthalate exposure and neurodevelopment: a systematic review and meta-analysis of human epidemiological evidence, 137 Environment International art. 105408 (2020), https://doi.org/10.1016/j.envint.2019.105408; Stephanie M. Engel, Amir Miodovnik, Richard L. Canfield et al., Prenatal phthalate exposure is associated with childhood behavior and executive functioning, 118 Environmental Health Perspectives 565–571 (2010), https://doi.org/10.1289/ehp.0901470.

[330] Arin A. Balalian, Robin M. Whyatt, Xinhua Liu et al., Prenatal and childhood exposure to phthalates and motor skills at age 11 years, 171 Environmental Research 416–427 (2019), https://doi.org/10.1016/j.envres.2019.01.046; Radke et al., *supra* note 329.

[331] Stephanie M. Engel, Gro D. Villanger, Rachel C. Nethery et al., Prenatal phthalates, maternal thyroid function, and risk of attention-deficit hyperactivity disorder in the Norwegian mother and child cohort, 126 Environmental Health Perspectives art. 057004 (2018), https://doi.org/10.1289/EHP2358, at 057004-1.

[332] Pam Factor-Litvak, Beverly Insel, Antonia M. Calafat et al., Persistent associations between maternal prenatal exposure to phthalates on child IQ at age 7 years, 9 PLOS ONE art. e114003 (2014), https://doi.org/10.1371/journal.pone.0114003, at 7, 9. The two phthalate metabolites concerned are monobenzyl phthalate and mono-isobutyl phthalate.

[333] *Id.* at 6, 10.

[334] Commission Report at 81.

[335] CHPAC Letter A, *supra* note 8, at 4. Letter A states that these outcomes are drawn from systematic reviews and meta-analyses of moderate quality, except for polycystic ovary syndrome, which rests on a high-quality review. *Id.*

[336] *Id.*

endometriosis, reduced sperm quality and asthma.[337] Reviewing epidemiological, toxicological, and mechanistic data, EPA found that some phthalates cause adverse effects on the developing male reproductive system.[338] The Centers for Disease Control and Prevention's National Biomonitoring Program finds BPA and phthalates in the urine of nearly all people tested, with children aged 6 to 11 years having the highest levels, followed by adolescents aged 12 to 19 years.[339]

IV. FDA Should Measure Microplastics in Food, Set Action Levels, and Establish Enforceable Tolerances

Petitioners request that FDA validate and publish methods for measuring microplastics in food, establish an action level for microplastics in each food or class of foods as validated methods become available, set at the lowest level that can be obtained, and establish a tolerance under section 406 of the Act. The requests apply to microplastics of every polymer type. Section IV.A describes the legal framework within which FDA would act. Section IV.B explains why microplastics are poisonous or deleterious substances that may render food injurious to health and why their presence in food cannot be avoided by good manufacturing practice. Section IV.C explains why the requested actions are the ones that framework makes available now, and how each should be carried out.

A. Legal Framework

Two provisions of the Act govern poisonous or deleterious substances in food. Section 402(a)(1) sets the standard for when such a substance makes food adulterated. Section 406 addresses added substances that food producers cannot avoid and allows FDA to limit them by regulation. FDA carries out both provisions through Part 109 of its regulations, which provides three instruments for setting a numerical level for a contaminant in food: tolerances, regulatory limits, and action levels.[340] Sections IV.A.1 and IV.A.2 describe these provisions and instruments and how FDA has used them for other contaminants.

1. The Statutory Standards

Section 402(a)(1) of the Act, 21 U.S.C. § 342(a)(1), establishes that a food shall be deemed adulterated if "it bears or contains any poisonous or deleterious substance which may render it injurious to health."[341] Courts have read the "may render" language to ask whether injury to the consumer is reasonably possible.[342] The standard turns on whether the substance is added. Food containing an added substance is

[337] *Id.* at 5. Letter A states that these outcomes are drawn from systematic reviews and meta-analyses of moderate quality, except for asthma, which rests on a high-quality review. *Id.*
[338] *Id.*
[339] Commission Report at 78.
[340] 21 U.S.C. §§ 342(a)(1), 346; 21 C.F.R. §§ 109.4(a)–(c), 109.6.
[341] 21 U.S.C. § 342(a)(1).
[342] *Lexington Mill*, 232 U.S. at 411 ("[I]t is not required that the article of food containing added poisonous or other added deleterious ingredients must affect the public health. . . . it is intended that if any flour, because of any added poisonous or other deleterious ingredient, may possibly injure the health of any of these, it shall come within the ban of the statute."); *United States v. Anderson Seafoods, Inc.*, 622 F.2d 157, 159 (5th Cir. 1980); *United States v. 1,200 Cans, Pasteurized Whole Eggs*, 339 F. Supp. 131, 135 (N.D. Ga. 1972) ("when a statute is worded in the subjunctive, the test is whether the result is reasonably possible").

adulterated if the substance "may render" it injurious; food containing a substance that is not added is not adulterated if the quantity "does not ordinarily render it injurious to health."[343]

Section 406 of the Act, 21 U.S.C. § 346, governs added poisonous or deleterious substances, and what it requires depends on whether the substance can be avoided. An added poisonous or deleterious substance that is not required in producing a food and can be avoided by good manufacturing practice is deemed unsafe, and food containing it is adulterated under section 402(a)(2)(A).[344] When the substance is required or cannot be avoided, the statute directs FDA to issue regulations, called tolerances, "limiting the quantity therein or thereon to such extent as [FDA] finds necessary for the protection of public health," and any quantity above the limit is also unsafe under section 402(a)(2)(A).[345] While such a tolerance is in effect, food bearing the substance is not adulterated under section 402(a)(1) by reason of that substance.[346] A tolerance therefore replaces the section 402(a)(1) standard for the substance and foods it covers.

In *Young v. Community Nutrition Institute*, a foundational Supreme Court decision on FDA's authority over unavoidable contaminants, the Court upheld FDA's longstanding reading that section 406 leaves the agency to decide whether to set a tolerance at all.[347] The Court also held that when no tolerance has been set, Congress intended the general adulteration provision to apply: section 402(a) "remains available to the FDA to prevent the shipment of any food '[i]f it bears or contains any poisonous or deleterious substance which may render it injurious to health.'"[348] FDA is thus "given the choice between employing the standard of § 346 and the standard of § 342(a)."[349] When FDA has not set a tolerance, it may therefore act against an unavoidable contaminant under section 402(a)(1).

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[343] For substances not added to food, the statute provides that "such food shall not be considered adulterated under this clause if the quantity of such substance in such food does not ordinarily render it injurious to health." 21 U.S.C. § 342(a)(1). For a substance that is not added, the government must therefore show that the substance would "*ordinarily* render (the food) injurious to health," rather than that it may do so. *Anderson Seafoods*, 622 F.2d at 159. The same court held that "added" means "artificially introduced, or attributable in some degree to the acts of man," after rejecting FDA's argument that a contaminant could be added without any connection to human acts. *Id.* at 160. FDA's regulations treat a substance as added unless it is "an inherent natural constituent of a food and is not the result of environmental, agricultural, industrial, or other contamination." 21 C.F.R. § 109.3(c)–(d). FDA's guidance applies the *Anderson Seafoods* understanding: "added" means "resulting from the hand of man." U.S. Food & Drug Admin., Ctr. for Food Safety & Applied Nutrition, Action Level for Inorganic Arsenic in Apple Juice: Guidance for Industry (June 2023), Docket No. FDA-2012-D-0322, at 3 n.2 [*hereinafter* Arsenic in Apple Juice Guidance]. Microplastics are synthetic polymers that reach food through environmental, agricultural, industrial, and packaging contamination, so they are "added" under both the regulation and the *Anderson Seafoods* test. Therefore, the treatment of non-added substances is omitted from this analysis. *See Young v. Cmty. Nutrition Inst.*, 476 U.S. 974, 978 (1986) (noting the parties' agreement that aflatoxin, although naturally and unavoidably present in some foods, is treated as "added" under § 346).

[344] 21 U.S.C. §§ 342(a)(2)(A), 346.

[345] 21 U.S.C. § 346.

[346] *Id.*

[347] *Young*, 476 U.S. at 979–81.

[348] *Id.* at 982 (citing *Lexington Mill*, 232 U.S. at 411).

[349] *Id.* at 984.

2. Action Levels and Tolerances

As relevant to this petition, FDA's regulations in Part 109 provide two instruments for setting a numerical level for an added poisonous or deleterious substance, and each has its own criteria.

First, tolerances are appropriate when "the substance cannot be avoided by good manufacturing practice" and "the tolerance established is sufficient for the protection of the public health, taking into account the extent to which the presence of the substance cannot be avoided and the other ways in which the consumer may be affected by the same or related poisonous or deleterious substances," and "[n]o technological or other changes are foreseeable in the near future that might affect the appropriateness of the tolerance established."[350] A tolerance may prohibit any detectable amount of the substance in food.[351]

Second, action levels may be established in two situations: when the criteria for tolerances are met "except that technological or other changes that might affect the appropriateness of the tolerance are foreseeable in the near future," or, "without regard to the criteria in paragraph (b) of this section or in section 406 of the act," at "a level at which the Food and Drug Administration may regard the food as adulterated within the meaning of section 402(a)(1) of the act."[352]

The instruments also differ in legal effect and in how they are set. Tolerances are set by regulation and bind FDA, industry, and the courts.[353] An action level is set less formally, to "define a level of contamination at which a food may be regarded as adulterated." FDA must announce each action level in the Federal Register with an invitation for public comment, and it withdraws an action level if a tolerance for the same substance and use is established.[354] When FDA amended its regulations in 1990, it explained that action levels "constitute prosecutorial guidance rather than substantive rules," that they "do not create a legal immunity from prosecution," and that they "are not binding on the agency, the courts, or the regulated industry."[355]

FDA has used these instruments for many environmental contaminants in food. The agency has set action levels for heavy metals in particular foods, including methylmercury in fish, inorganic arsenic in apple juice, and lead in processed food intended for babies and young children, each marking a level at which FDA "may regard" the food as adulterated within the meaning of section 402(a)(1), and in October 2026 it proposed action levels for cadmium in processed food intended for babies and young children.[356] The

[350] 21 C.F.R. §§ 109.4(a), 109.6(b)(1)–(3).
[351] 21 C.F.R. § 109.4(a).
[352] 21 C.F.R. § 109.6(d).
[353] 21 C.F.R. §§ 109.4(a)–(c), 109.6(b)–(d); 21 U.S.C. § 346; U.S. Food & Drug Admin., Action Levels for Added Poisonous or Deleterious Substances in Food, Final Rule, 55 Fed. Reg. 20,782, 20,783–84 (May 21, 1990) (Docket No. 88N-0006) (regulatory limits "will be binding on the food and feed industry as well as on FDA and the courts"; action levels "are not binding on the agency, the courts, or the regulated industry").
[354] 21 C.F.R. §§ 109.4(c)(1)–(2), 109.6(d).
[355] 21 C.F.R. § 109.4(c)(1); 55 Fed. Reg. at 20,782–83; *see Young*, 476 U.S. at 977 (describing action levels before the 1990 amendments).
[356] 21 C.F.R. § 109.6(d); U.S. Food & Drug Admin., Ctr. for Food Safety & Applied Nutrition, Guidance for Industry: Action Levels for Poisonous or Deleterious Substances in Human Food and Animal Feed (Aug. 2000), Docket No. FDA-2020-D-1956, https://www.fda.gov/regulatory-information/search-fda-guidance-documents/guidance-industry-action-levels-poisonous-or-deleterious-substances-human-food-and-animal-feed (last visited Nov. 9, 2026) (entry for mercury); Arsenic in Apple Juice Guidance, *supra* note 343, at 3; U.S. Food & Drug Admin., Human Foods Program, Action Levels for Lead in Processed

action level for methylmercury in fish is 1.0 parts per million,[357] and the action level for arsenic in apple juice is 10 parts per billion.[358] For lead in candy, FDA has recommended a maximum level of 0.1 parts per million.[359] In its Closer to Zero program and its guidance on arsenic in apple juice and lead in candy, FDA has based its levels on toxicological data and on what manufacturers can achieve, and for lead it has described its policy as reducing exposure "to the lowest level that can practically be obtained."[360] FDA has also set an action level for aflatoxin, a toxin produced by a mold that grows in some foods.[361] It has set tolerances under section 406 for PCBs, persistent environmental contaminants that build up in the food chain, in several foods, including fish and shellfish.[362]

B. Basis for Action Levels and Tolerances

1. Microplastics Are Poisonous or Deleterious Substances That May Render Food Injurious to Health

Microplastics satisfy the statutory definition of poisonous or deleterious substances that may render food injurious to health under section 402(a)(1) of the Act. These particles are not inert materials but rather biologically active contaminants that produce documented harm through multiple pathways. Most ingested particles are thought to leave the body in the feces, but particles up to about 150 micrometers can be absorbed through the intestinal lining, and the smallest can reach deep into organs.[363] There, as the experimental studies described in Section III show, they can provoke inflammation and damage cells and tissues. Chemical toxicity arises from the leaching of plastic additives including bisphenol compounds, phthalate plasticizers, flame retardants, and stabilizers, as well as from the release of environmental pollutants that adsorb to particle surfaces, including heavy metals, persistent organic pollutants, and pharmaceutical residues.[364] Particles in the nanoscale range cross biological barriers including the

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Food Intended for Babies and Young Children: Guidance for Industry (Jan. 2025), Docket No. FDA-2022-D-0278, at 3–4 [*hereinafter* Lead in Baby Food Guidance]; U.S. Food & Drug Admin., Action Levels for Cadmium in Processed Food Intended for Babies and Young Children; Draft Guidance for Industry; Availability, 91 Fed. Reg. 66,796 (Oct. 23, 2026) (Docket No. FDA-2025-D-4023).

[357] U.S. Food & Drug Admin., Ctr. for Food Safety & Applied Nutrition, Fish and Fishery Products Hazards and Controls Guidance app. 5, at A5-9 tbl. A-5 (June 2022 ed.), https://www.fda.gov/Seafood.

[358] Arsenic in Apple Juice Guidance, *supra* note 343.

[359] Lead in Candy Supporting Document, *supra* note 4, § I.

[360] Closer to Zero, *supra* note 4 ("We propose action levels based on toxicological data and what is achievable by manufacturers."); Arsenic in Apple Juice Guidance, *supra* note 343, at 3; U.S. Food & Drug Admin., Guidance for Industry: Lead in Candy Likely To Be Consumed Frequently by Small Children: Recommended Maximum Level and Enforcement Policy (Nov. 2006), Docket No. FDA-2005-D-0084, https://www.fda.gov/regulatory-information/search-fda-guidance-documents/guidance-industry-lead-candy-likely-be-consumed-frequently-small-children (last visited Nov. 9, 2026) ("FDA considers the recommended maximum lead level to be achievable with the use of good manufacturing practices in the production of candy and candy ingredients and to be protective of human health."); Lead in Candy Supporting Document, *supra* note 4, § III (describing FDA's policy of reducing lead exposure "to the lowest level that can practically be obtained").

[361] *Young*, 476 U.S. at 977–78.

[362] 21 C.F.R. § 109.30.

[363] Cverenkárová et al., *supra* note 176, at 12 (stating that more than 90 percent of ingested microplastics is assumed to be excreted in the feces, that absorption through the intestinal epithelium probably occurs only for particles up to 150 micrometers, and that particles smaller than 1.5 micrometers can penetrate deep into organs).

[364] *Id.* at 4–5.

intestinal lining, blood-brain barrier, and placental barrier, distributing throughout the body and accumulating in critical organs.[365] Detection of plastic particles in human blood, liver, kidney, spleen, brain, placenta, breast milk, cardiovascular tissue, and gastrointestinal tract, described in Section II, demonstrates that ingested particles achieve systemic distribution rather than passing harmlessly through the digestive system.

Several lines of the evidence in Section III come from people. In the prospective observational study discussed in Section III.B.1, patients whose carotid plaque contained detectable microplastics had a higher rate of heart attack, stroke, or death from any cause over an average of 34 months than patients whose plaque did not, with an adjusted hazard ratio of 4.53 and an unadjusted hazard ratio of 2.84. The authors do not claim that the particles caused those events, and Petitioners do not rely on the study for that proposition.[366] What it shows is an association, in people, between plastic in arterial tissue and subsequent cardiovascular events, which is evidence that microplastics may render food injurious to health within the meaning of section 402(a)(1) of the Act. The limitations of the method used to measure the particles are set out in Section IV.B.3.

A study of patients with inflammatory bowel disease found significantly higher concentrations of microplastics in feces compared to healthy individuals, with higher concentrations correlating with more severe disease activity, though the authors note that the disease may cause particles to stay longer in the gut rather than the reverse.[367] Analysis of liver samples from patients with cirrhosis detected microplastic particles at concentrations ranging from 4.6 to 11.9 particles per gram of tissue, while liver, kidney, and spleen samples from five people without liver disease tested negative.[368] Population-wide biomonitoring by the CDC found detectable levels of BPA in 95.7 percent of urine samples from the U.S. population, showing near-universal exposure to a plastic additive that particles can carry and release (Section III.A) and that is associated with the endocrine effects described in Section III.B.2.[369] That measurement covers bisphenol A from every source, not only from particles. Experimental studies using human cells and tissues demonstrate that microplastic exposure generates oxidative stress, damages mitochondria, disrupts cellular function, triggers inflammatory responses, and interferes with normal liver metabolism and detoxification processes, and animal studies show that it alters gut bacteria, although most of these studies used concentrations above those measured in human tissue.

Children face heightened risks from microplastic contamination in food due to biological vulnerabilities during development. A majority of the members of EPA's Children's Health Protection Advisory Committee conveyed to EPA Administrator Zeldin that "the science is clear that plastic pollution can harm children's health and raise risks of developmental disabilities, birth defects, cancers, and other serious diseases."[370] Detection of microplastics in human placenta, meconium, infant feces, and breast milk confirms that exposure begins before birth and continues through early development, and one study found a median concentration of PET in the feces of six infants about fourteen times that in the feces of

[365] *Id.* at 12.
[366] Marfella et al., *supra* note 138, at 900, 905, 908.
[367] Yan et al., *supra* note 155, at 414.
[368] Horvatits et al., *supra* note 143, at 1, 4 (proof-of-concept case series of six patients with cirrhosis and five individuals without underlying liver disease).
[369] Lehmler et al., *supra* note 227, at 6525.
[370] CHPAC Letter A, *supra* note 8, at 1. Letter A defines plastic pollution to encompass the full life cycle of plastics, including plastic-associated chemicals as well as microplastics. *Id.*

ten adults.[371] Epidemiological studies document associations between prenatal exposures to plastic-associated chemicals and lower intelligence quotient scores, increased risk for autism spectrum disorder, attention deficit hyperactivity disorder, and diminished cognitive function that persists into the school years. A study of 1,057 pregnancies found that higher levels of plastic in the placenta were associated with higher liver enzyme levels in umbilical cord blood, which the authors read as a sign of potential effects on fetal liver function.[372] The endocrine system governs all facets of early human development, including physical growth, organ formation, and development of the brain, reproductive system, and immune system, making exposures during pregnancy and the first years after birth particularly dangerous because they can cause changes within developing cells and organs that increase risk of disease and disability across the lifespan.

The record does not depend on any single study. Microplastics are present across the food supply (Section I.C), and they are absorbed and found in human blood, organs, placenta, and breast milk (Section II). In people, plastic in carotid plaque has been associated with later cardiovascular events, and more plastic in feces with more severe inflammatory bowel disease (Sections III.B.1 and III.B.3). In human cells, tissue models, and animals given measured doses, microplastics cause oxidative stress, inflammation, damage to the intestinal barrier, and disruption of liver and metabolic function (Section III.B). How large the risk is at current dietary exposure is not yet known, for the reasons set out in Section IV.B.3. Section 402(a)(1) does not require that it be known. It asks whether a substance "may" render food injurious to health, and the best available scientific evidence shows that microplastics certainly may. They are therefore poisonous or deleterious substances within the meaning of the Act.

2. Microplastic Contamination Is Not Avoidable by Good Manufacturing Practice

Section 406 of the Act and FDA's regulations ask whether an added poisonous or deleterious substance "cannot be avoided by good manufacturing practice."[373] For microplastics it cannot. Much of the plastic in food reaches it before any processor handles it. Sewage sludge spread on farm fields is estimated to add 125 to 850 tons of microplastics a year to agricultural soils for every million inhabitants;[374] plastic mulch and polymer-coated fertilizer add more (Section I.B.1). Experiments show that crop plants can take up the smallest particles through their roots and move them into the shoots.[375] Fish and shellfish take in particles from the water and sediment, from fishing and aquaculture gear, and from fishmeal made of other fish that have eaten plastic.[376] A farmer cannot remove particles already in the soil, and a fishery cannot remove them from the sea.

Particles are also added after harvest, by processing equipment and packaging, and some of those sources can and should be reduced. For example, Japan's ban on PVC gloves in food preparation lowered phthalate levels in prepared food (Section I.B.3), and a processor can change a filter membrane or a bottle closure that sheds particles into food. But plastic is widely used throughout food processing and packaging. A 2025 systematic evidence map of 103 studies concluded that the normal, intended use of plastic food contact articles, a category that includes processing equipment as well as packaging, can release microplastics into food, and its authors found food in glass jars, cans, and cartons likely to contact

[371] Zhang et al., *supra* note 170, at 989, 991; Liu et al., *supra* note 168, at 1; Ragusa et al., *supra* note 171, at 1.
[372] Wang et al., *supra* note 285, at 1.
[373] 21 U.S.C. § 346; 21 C.F.R. § 109.6(b)(1), (c)(1).
[374] Nizzetto et al., *supra* note 46, at 10777.
[375] Jadhav & Medyńska-Juraszek, *supra* note 42, at 3.
[376] Garrido Gamarro & Costanzo, *supra* note 41, at 17, 33.

plastic through a closure gasket or an inner coating.[377] The same review notes that ultra-processed foods, which pass through more processing steps and therefore more contact with plastic equipment, make up more than half of the diet in the United States, and that early studies find more microplastics in them than in minimally processed foods.[378] Replacing one source leaves the others in place, and none of these changes removes the particles that arrive from the field or the sea.

3. Limitations of the Evidence

Although the available evidence on microplastics demonstrates that they most certainly "may" render food injurious to health, the field is still relatively young. As 21 C.F.R. § 10.30(b) requires, Petitioners explain the limits below.

The first limitation is measurement. There is currently no universally accepted way to find and count microplastics in food or in human tissue, and the methods in use have shortcomings in both directions. FDA's analysts have catalogued the ways they undercount. Methods that rely on visual inspection of a sample miss particles smaller than a millimeter. Some methods that identify a particle by its chemical fingerprint cannot resolve anything below ten micrometers. The chemical pretreatment used to clear away food or tissue destroys some polymers outright. Small particles are lost in the step that floats plastic off from heavier material. Each of these causes analysis to report less plastic than the sample holds. The same authors describe the opposite issue. Plastic is hard to tell apart from natural molecules of similar structure, false positives are common enough that earlier estimates for some foods have been called greatly overestimated, and where those false positives come from is still poorly understood.[379] FDA's compendium of laboratory methods contains no validated method for microplastics in food.[380]

Many studies of microplastics in blood, placenta, and the brain measure plastic by weight rather than counting particles. The technique heats the sample and identifies each polymer by the smaller molecules it forms after breaking apart. Body fat breaks into the same molecules as polyethylene. A 2025 study set out to test the technique on blood and concluded that it is not currently suitable for measuring polyethylene or PVC in biological samples: the interference can be reduced, but not removed. That study also noted that earlier work on human samples using this technique had mostly reported high polyethylene and PVC levels, without making clear whether the interference had been accounted for.[381] The point reaches the studies this petition cites that measured polyethylene or polyvinyl chloride by this technique: the carotid plaque study (Section III.B.1), the brain study (Section II.A), the blood and blood clot studies (Section II.B), and the placenta study (Section II.C). But Petitioners do not ask FDA to adopt any published concentration as a finding of fact. Whatever this measurement problem means for particular studies and polymers, the evidence is firm: plastic is detected across food categories, across laboratories, and by many methods. The petition rests on that consistency and on the biological findings in Section III, not on the precision of any one figure. The remedy requested accounts for the problem directly. Petitioners ask FDA first to validate methods and measure microplastics in food and to set a numerical level for a food based on those methods.

The second limitation is arguably the health evidence itself, although this area of research is rapidly growing. Most existing evidence comes from cells and from non-human animals. The Commission

[377] Zimmermann et al., *supra* note 80, at 1, 2.
[378] *Id.* at 7.
[379] Duncan et al., *supra* note 13, at 4346, 4348; Duncan et al., *supra* note 14, at S-2, S-4 to S-5, S-7, S-9.
[380] Duncan et al., *supra* note 13, at 4351.
[381] Rauert et al., *supra* note 148, at 1984, 1985, 1991, 1992 (study funded by the Minderoo Foundation).

Report says as much: studies of human health effects are held back by the absence of a standard way to measure exposure, which is still being developed, so most of what is known rests on animal and laboratory work.[382] The European Commission, in adopting the drinking-water method referenced in Section IV.C.1, stated that current data provide "limited conclusive scientific evidence on the adverse effects of microplastics on human health," because information on the biological effects of microplastics and on exposure to them remains substantially limited.[383] The 2024 systematic review that graded this literature concluded "suspected," and lowered its own rating because the available studies were few and small. Its authors list the gaps plainly. The rodent studies covered only three polymer types in manufactured form, plus tire wear particles as the single secondary source. The particles tested were nearly all spheres. Plastic additives were not accounted for. Studies finding no effect may never have been published. And the human evidence is sparse.[384] A second systematic review, published in 2025, goes further. Funded by the American Chemistry Council, with several authors employed by plastics manufacturers, it appraised 24 mammalian studies of reproductive and developmental effects and rated every one at high risk of bias under the National Toxicology Program's framework, and concluded that this body of work cannot support conclusions about reproductive toxicity.[385] The review's own conclusion is narrower than its critique. It states that developmental and reproductive effects cannot be ruled out, and that the evidence is insufficient for deriving toxicological benchmarks. Both statements are consistent with what Petitioners ask FDA to find: that microplastics may render food injurious to health.

Given full weight, these limitations simply suggest that the full size of the harm is not yet known. They do not establish that there is no harm. That distinction is what section 402(a)(1) turns on. The Supreme Court held in *United States v. Lexington Mill & Elevator Co.*, construing the "may render" language of the Food and Drugs Act of 1906 that section 402(a)(1) carried forward, that the statute reaches a food containing an added poisonous or deleterious ingredient that *may possibly* injure a consumer's health, and does not require proof that the food has affected the public health.[386] A standard written in those terms is built to operate before the science is finished. The evidence in Section III meets it. Microplastics are in every category of the American food supply that has been examined. They are in human tissue. And the biological effects documented in human cells, in tissue models, in animals given measured doses, and in observational studies of patients point consistently toward harm. One further point follows from the measurement problem. Several of the analytical limits FDA identifies cause analysts to miss particles that are there, and they fall hardest on the smallest particles, which are the ones most able to cross into tissue.

[382] Commission Report at 89.
[383] Commission Delegated Decision (EU) 2024/1441 of 11 March 2024 supplementing Directive (EU) 2020/2184 of the European Parliament and of the Council by laying down a methodology to measure microplastics in water intended for human consumption, OJ L, 2024/1441, 21.5.2024 (notified under document C(2024) 1459), recital (2), http://data.europa.eu/eli/dec_del/2024/1441/oj (last visited Nov. 4, 2026) [*hereinafter* EU Drinking Water Methodology].
[384] Chartres et al., *supra* note 9, at 22858–59.
[385] Fitch et al., *supra* note 315, at 289, 293, 299, 302. The review was supported by the American Chemistry Council, which was involved in conceptualizing it and reviewed the final manuscript, and four of its authors are employed by companies that produce plastics.
[386] *Lexington Mill*, 232 U.S. at 411. *See also Young*, 476 U.S. at 982–83 (describing *Lexington Mill* as "discussing proper interpretation of the language that became § 342(a)").

C. The Requested Actions

1. Validated Methods and a Survey of the Food Supply

Every numerical action level or tolerance FDA could set for microplastics depends on a method that can measure them in the food it covers. FDA claims that "there are no standardized methods for how to detect, quantify, or characterize microplastics and nanoplastics," and that it is working to advance the science through analysis of testing methods.[387] Petitioners therefore ask FDA to expeditiously validate and publish such methods. Methods with defined quality controls already exist for one major route of exposure—drinking water.[388] Petitioners ask FDA to develop comparable methods for each food or class of foods, beginning with those that contribute most to dietary exposure, and to revise the methods as they improve. With those methods, FDA should measure microplastics across the food supply and publish the results, including the share that comes from packaging and processing materials.

2. Action Levels

Petitioners ask FDA to establish action levels for microplastics now to ensure that there are some measures in place to provide some protection of public health from microplastics while FDA pursues tolerance levels for them. An action level may be set at a level at which FDA may regard the food as adulterated under section 402(a)(1), without regard to the tolerance criteria, and FDA's action levels for lead and arsenic in food use that formulation.[389]

As methods become validated for a food or class of foods, FDA should establish an action level for microplastics in that food and announce it in the Federal Register with an invitation for public comment, as its regulations require.[390] Each action level should be set at the lowest level that can be obtained in that food, and lowered as methods and manufacturing practices improve. That is consistent with FDA's stated policy of reducing lead exposure to the lowest level that can practically be obtained, and with its Closer to Zero approach of lowering levels over time (Section IV.A.2), and it ties each level to what good manufacturing practice can achieve, which is the measure Part 109 uses (Section IV.B.2).

3. A Tolerance

Once validated methods are in place for a food or class of foods, FDA should set a tolerance for microplastics in that food under section 406, at a level no less protective of public health than the action level it replaces. In doing so, FDA must take into account "the extent to which the use of such substance is required or cannot be avoided in the production of each such article."[391] While a tolerance is in effect, food bearing an added amount of the substance is not "considered to be adulterated within the meaning

[387] FDA Microplastics Web Page, *supra* note 1.
[388] In 2024 the European Commission adopted a methodology, addressed to the European Union's member states, for measuring microplastics in drinking water that specifies various parameters for detection. EU Drinking Water Methodology, supra note 383, arts. 1–2; *id*. Annex § 2 & pts. (3), (6), (8), (10). The California State Water Resources Control Board has also adopted standard operating procedures for measuring microplastics in drinking water by Raman and infrared spectroscopy. California State Water Resources Control Board, Microplastics, https://www.waterboards.ca.gov/drinking_water/certlic/drinkingwater/microplastics.html (last visited Nov. 22, 2026), at 1, 3.
[389] 21 C.F.R. § 109.6(d); Lead in Baby Food Guidance, *supra* note 356, at 4; Arsenic in Apple Juice Guidance, *supra* note 343, at 3.
[390] 21 C.F.R. § 109.4(c)(2).
[391] 21 U.S.C. § 346.

of" section 402(a)(1), so the tolerance would take the place of the action level for the foods it covers, and FDA would withdraw the action level.[392]

D. Conclusion

The evidence in this petition shows that microplastics in food are poisonous or deleterious substances that may render it injurious to health within the meaning of section 402(a)(1) of the Act, 21 U.S.C. § 342(a)(1). Microplastics are found across the food supply and in human tissue. In people, they have been associated with cardiovascular events and with more severe inflammatory bowel disease. In laboratory and animal studies, they cause inflammation, oxidative stress, and harm to the heart, liver, gut, and reproductive, endocrine, and metabolic systems. Because they reach food through soil, water, feed, processing, and packaging, no diet avoids them. While the severity of risk at current exposures is not yet fully understood, as Section IV.B.3 explains, the statute requires FDA to act now because it is clear that at a minimum microplastics "may" render food injurious to health.

FDA has authority under section 402(a)(1) of the Act and 21 C.F.R. §§ 109.4(c) and 109.6(d) to set action levels marking the levels at which it may regard food containing microplastics as adulterated, and under section 406 to set tolerances. It has regulated other environmental contaminants, including heavy metals and PCBs, through the instruments described in Section IV.A. Microplastic contamination arises from environmental, agricultural, processing, and packaging pathways, which have led to a pervasive microplastic pollution and contamination problem, which food producers cannot alone eliminate through good manufacturing practice, as Section IV.B.2 explains. To address this escalating threat to public health, Petitioners therefore ask FDA to validate and publish methods for measuring microplastics in food and to measure them across the food supply; to establish an action level for each food or class of foods with these validated methods, at the lowest level that can be obtained; and to set tolerances under section 406 for each food or food class that are no less protective of public health than the action levels they replace. The evidence that microplastics may render food injurious to health supports each of these steps.

[392] 21 U.S.C. § 346; 21 C.F.R. § 109.6(d).