Microplastics in Seafood and the Implications for Human Health

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Abstract

Purpose of Review We describe evidence regarding human exposure to microplastics via seafood and discuss potential health effects.

Recent Findings Shellfish and other animals consumed whole pose particular concern for human exposure. If there is toxicity, it is likely dependent on dose, polymer type, size, surface chemistry, and hydrophobicity.

Summary Human activity has led to microplastic contamination throughout the marine environment. As a result of widespread contamination, microplastics are ingested by many species of wildlife including fish and shellfish. Because microplastics are associated with chemicals from manufacturing and that sorb from the surrounding environment, there is concern regarding physical and chemical toxicity. Evidence regarding microplastic toxicity and epidemiology is emerging. We characterize current knowledge and highlight gaps. We also recommend mitigation and adaptation strategies targeting the life cycle of microplastics and recommend future research to assess impacts of microplastics on humans. Addressing these research gaps is a critical priority due to the nutritional importance of seafood consumption.

Keywords Microplastics · Toxicology · Ocean · Seafood · Fish · Human health impacts

Introduction

Since the 1960s, plastic production has increased by approximately 8.7% annually, evolving into a $600 billion global industry [1, 2]. Approximately eight million metric tons of plastics enter the oceans annually [2], and conservative estimates suggest 5.25 trillion plastic particles currently circulate in ocean surface waters [3•]. While some plastics enter oceans from maritime operations, 80% is suspected to originate from land-based sources [1]. Discarded plastic materials enter the marine environment as trash, industrial discharge, or litter through inland waterways, wastewater outflows, and transport by winds or tides [4]. Waste generation and waste leakage are inextricably linked and proportionally associated with economic development, local infrastructure, and legislation. Today, uncollected waste accounts for 75% of these land-based discharges, while the remaining 25% comes from within the waste management system [4].

When plastics enter the ocean, the rate of degradation and persistence of plastics varies by polymer, shape, density, and the purpose of the plastic itself [3•]. These characteristics also govern where in the water column plastics may be found. For example, more buoyant plastics are more likely to be carried by ocean currents and wind across the environment [3•]. Additionally, when plastics are exposed to natural forces like sunlight and wave action, plastics will degrade into microplastics—defined as plastic particles under 5 mm in size. This definition commonly includes plastic pieces in the nanoscale, < 1 μm in size. The extent of plastic degradation depends on factors including polymer type, age, and environmental conditions like weathering, temperature, irradiation, and pH [5]. Over time, plastic particles contaminate the marine ecosystem and the food chain, including foodstuffs intended for human consumption [6]. In vivo studies have
Background on Microplastics

Sources and Distribution

In the marine environment, microplastics are a heterogeneous group of particles (< 5 mm), varying in size, shape, and chemical composition. They are found in sediment, on the sea surface, in the water column, and in wildlife [7, 8]. Table 1 describes the most common plastic polymer types in the marine environment. Of these, the most common plastic types manufactured are polyethylene and polypropylene [7].

Microplastics are often categorized into primary and secondary types. Primary microplastics were originally produced to be < 5 mm in size, while secondary microplastics result from the breakdown of larger items. Microbeads in personal care products are an example of primary microplastics [9•]. While they are now being phased out globally, in 2015, an estimated eight billion microbeads were released into aquatic habitats from the USA daily [10]. Other sources of primary microplastics include industrial abrasives and pre-production plastic pellets used to make larger plastic items. Sources of secondary microplastics include microfibers from textiles, tire dust, and larger plastic items that degrade and, consequently, fragment into microplastic particles, mostly due to weathering degradation [11]. Even if humans halted plastic production and prevented plastic waste dumping, marine microplastics would continue to increase as larger plastic litter degrades into secondary microplastics [9•].

Physical and Chemical Properties

Microplastics in the marine environment are typically found as pellets, fragments, or fibers and are composed of diverse polymers [12], some denser than seawater and expected to sink to the seafloor. These include polyamide, polyester, polymerizing vinyl chloride (PVC), and acrylic, among others. Others are lighter than seawater and are often found floating at the surface, including polyethylene, polypropylene, and polystyrene.

Plastic products are composed of monomers joined to make the polymer structure and additive chemicals. During production, plastic is processed with additives to provide specific properties [13]. Several thousand distinct additives are used, including plasticizers, flame retardants, pigments, antimicrobial agents, heat stabilizers, UV stabilizers, fillers, and flame retardants such as polybrominated diphenyl ethers (PBDEs) [13, 14••, 15]. Additives account for approximately 4% of the weight of microplastics [14••]. Once created, plastic polymers are described as non-toxic because they are not reactive and generally cannot easily transport across biological membranes due to their size [16]. However, non-polymeric substances, like chemical additives or residual monomers, can be hazardous to human health and the environment when they leach from the plastic polymer matrix [6]. As plastics progressively degrade, the surface area to volume ratio increases and additive chemicals are expected to leach [17]. Leached chemicals may bioaccumulate in animals from seawater [17]. For organisms that have directly ingested microplastics, the uptake rate of additive chemicals by an organism’ gastrointestinal tract is primarily influenced by the chemical fugacity gradient between the organisms’ tissues and the plastic, the gut retention...
time of the microplastics, and the material-specific kinetic factors [18].

In addition to additive chemicals being associated with plastic debris, microplastics in the ocean accumulate persistent organic pollutants (POPs) such as polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), and organochlorine pesticides like dichlorodiphenyltrichloroethane (DDT) or hexachlorobenzene (HCB) from the water [18, 19]. These have a greater affinity for plastic than water, and concentrations on microplastics are orders of magnitude greater than in surrounding water [19, 20]. PBDEs are human-made flame-retardant chemicals. PBDEs enter the marine environment mainly via discarded or leaked consumer goods or municipal waste. Plastic deposited on beaches from the marine environment have been found to contain from 0.03 to 50 ng/g PBDE [17].

The global distribution of chemicals in the marine environment may affect environmental and human health, but microplastics do not represent the only exposure pathway. In fact, microplastics may represent a relatively small contributor to the total risk as there are many other sources for chemical exposure [18]. For example, the total dietary intake of PCBs from microplastics is likely minimal compared to that from other sources, as identified in Table 2 [6]. For other chemicals, such as bisphenol A (BPA) or PBDEs, sources of exposure may be limited to or originate from microplastic degradation.

### Degradation of Marine Plastics

Plastic is persistent in the marine environment because it is manufactured to be durable. Still, plastic polymers can be degraded slowly by microorganisms (e.g., *Bacillus cereus*, *Micrococcus sp.*, or *Corynebacterium*), heat, oxidation, light, or hydrolysis, as identified in Table 3. The rate and extent of plastic degradation are determined by the environmental variables present.

### Microplastics in the Food Chain

#### Exposure to Microplastics by Marine Animals

A 2016 UN report documented over 800 animal species contaminated with plastic via ingestion or entanglement—a figure 69% greater than that reported in a 1977 review, which estimated only 247 contaminated species [21, 22]. Of these 800 species, 220 have been found to ingest microplastic debris *in natura* [6].

Plastic ingestion occurs across taxa within different trophic levels, including marine mammals, fish, invertebrates, and fish-eating birds [8, 9•]. Plastic particles are often found concentrated in an organism’s digestive tract during carcass dissection and laboratory research. With preference to smaller particles, micro- and nanoplastics can persist in the animal’s body [6, 9•, 11, 22, 23] and translocate from the intestinal tract to the circulatory system or surrounding tissue [6].

#### Human Exposure Pathways

Seafood consumption represents one pathway for human microplastic exposure. As of 2015, global seafood intake represented 6.7% of all protein consumed and approximately 17% of animal protein consumption [24]. Global per capita seafood consumption is over 20 kg/year; in the USA, it is 7 kg annually [25]. Global seafood trade in 2016 was $132.6 billion, and over 90% of US seafood was imported from geographic regions with significant waste leakage and pelagic plastic pollution [6]. Roughly half of seafood is farmed (e.g., aquaculture) and half is wild-caught. It is possible to control environmental conditions in aquaculture—by raising animals in ponds, tanks, or selected water bodies—and animals generally have shorter lifespans in aquaculture than in the wild, which could provide less opportunities and time for microplastic exposure and uptake. Due to few studies, there
is uncertainty about the differences in microplastics for farmed and wild fish and shellfish.

Because of their small size, microplastics can be ingested by a wide variety of marine organisms. Ingestion may be direct or indirect via trophic transfer (e.g., up the food web). Microplastic ingestion has been documented in planktonic organisms and larvae at the bottom of the food chain [25–28], in small and large invertebrates [6, 7, 11, 29, 22] and in fish [6]. Trophic transfer of microplastics was observed in the predatory Crucian carps [30].

Microplastics are found in many species intended for human consumption including invertebrates, crustaceans, and fish [23, 31•]. Plastic particles are often found concentrated in an organisms’ digestive tracts such that bivalves and small fish consumed whole are more likely to expose microplastics to the human diet [9••]. For example, Fig. 1 illustrates movement of plastic from bivalve mollusks to the human diet. Van Cauwenberghe and Janssen [23] found farmed mussels had significantly higher microplastic concentrations (178 microfibers) than wild-caught mussels (126 microfibers) [23]. Additionally, Rochman et al. identified the presence of microplastics (> 500 μm) in commercially sold, wild-caught fish from markets in Makassar, Indonesia (28% of fish processed contained microplastics), and California, USA (25% of commercial fish processed contained microplastics) [31•]. Karami et al. investigated the potential presence of microplastics in dried fish tissue: excised organs (viscera and gills) and eviscerated flesh (whole fish, excluding the viscera and gills) [32]. In four of 30 commonly consumed dried fish species, 36 of 61 isolated foreign particles were identified as

### Table 2  Comparing the estimated total dietary exposure to contaminants and additives directly from microplastics in seafood

| Compound          | Highest concentration in microplastics | Calculated intake from microplastics (pg/kg bw/day) | Total intake from the diet (pg/kg bw/day) | Ratio intake microplastic/total dietary intake (pg/kg bw/day) (%) |
|-------------------|----------------------------------------|---------------------------------------------------|----------------------------------------|----------------------------------------------------------------|
| **Contaminants**  |                                        |                                                   |                                        |                                                                  |
| Non-dioxin like PCBs | 2970                                   | 0.3                                               | –                                      | –                                                                |
| EFSA, 2012        | –                                      | –                                                 | 4300*                                  | 0.007                                                            |
| JECFA, 2016       | –                                      | –                                                 | 1000*                                  | 0.03                                                             |
| PAHs              | 44,800                                 | 4.5                                               | –                                      | –                                                                |
| EFSA, 2008        | –                                      | –                                                 | 28,800*                                | 0.02                                                             |
| JECFA, 2006       | –                                      | –                                                 | 4000*                                  | 0.1                                                              |
| DDT               | 2100                                   | 0.2                                               | –                                      | –                                                                |
| EFSA, 2006        | –                                      | –                                                 | 5000*                                  | 0.004                                                            |
| JECFA, 1960       | –                                      | –                                                 | 100,000,000*                           | 0.0000002                                                        |
| **Additives/monomers** |                                       |                                                   |                                        |                                                                  |
| Bisphenol A       | 200                                    | 0.02                                              | –                                      | –                                                                |
| EFSA, 2015a       | –                                      | –                                                 | 130,000*                               | 0.00002                                                          |
| FAO/WHO, 2011      | –                                      | –                                                 | 400,000*                               | 0.000005                                                         |
| PBDEs             | 50                                     | 0.005                                             | –                                      | –                                                                |
| EFSA, 2011        | –                                      | –                                                 | 700*                                   | 0.0007                                                           |
| JECFA, 2006       | –                                      | –                                                 | 185*                                   | 0.003                                                            |
| NP                | 2500                                   | 0.3                                               | NA1                                    | –                                                                |
| OP                | 50                                     | 0.005                                             | NA1                                    | –                                                                |

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PCBs polychlorinated biphenyls, PAHs polycyclic aromatic hydrocarbons, DDT dichlorodiphenyltrichloroethane, PBDEs polybrominated diphenyl ethers, NP nonylphenol, OP octylphenol

a Lowest intake of six indicators of non-dioxin like PCBs, representing about 50% of all non-dioxin like PCBs

b Median intake (EFSA, 2008)

c Mean intake of benzo[a]pyrene (JECFA)

d Lowest intake, DDT, and related compounds (EFSA, 2006)

e Average intake adults (EFSA, 2015a)

f Lowest intake FAO/WHO

g Lowest intake, sum of BDE-47, BDE-209, BDE-153, and BDE-154 (EFSA, 2011)

h Lowest intake JECFA

i NA: dietary intake not available from EFSA or JECFA

j Provisional tolerable daily intake (JECFA)
plastic polymers [32]. In young and adult fish, Yifeng et al. demonstrated microplastic particle translocation from digestive tracts to the gills and liver of zebrafish (*Danio rerio*), a common prey fish [33]. Microplastic particle translocation is also documented in European seabass and the common goby (*Pomatoschistus microps*) [34]. Together, these studies demonstrate the presence of microplastics, not the chemical constituents, in some seafood and indicate that the challenge could be widespread due to ubiquity in the environment and translocation potentially moving particles to animal parts typically eaten by humans.

Because water and salt are often extracted from the natural world, researchers investigated whether products made with these ingredients were also contaminated with nano- and microplastics. They investigated and found microplastics in beer [35], honey [36], and sea salt [37]. While the origin of these contaminants is uncertain, potential sources include atmospheric emission and uptake of microplastics by the basic components of the food products, impurities introduced by processing materials, and the contaminants present in packaging [35]. Increasingly, scientific evidence outlines multiple pathways of microplastic exposure via food including

| Degradation process       | Explanation                                                                 |
|---------------------------|-----------------------------------------------------------------------------|
| Biodegradation            | Decomposition of organic materials by microorganisms                        |
| Photo degradation         | Action of light or photons, usually sunlight (UVA or greater, > 320 nm)     |
| Thermooxidative degradation | Slow oxidative, molecular deterioration at moderate temperatures            |
| Thermal degradation       | High temperature cause molecular deterioration (not an environmental mechanism) |
| Hydrolysis                | Reaction with water                                                          |

Because they filter water, bivalves (such as mussels, oysters, clams and others) can absorb and excrete microplastic present in the sea water where they are cultivated. After harvesting, shellfish are usually kept in clean water to get rid of contaminants. The shellfish expel some microplastics, while others remain inside, reach the market and end up on the consumer's plate.
evidence that microplastics are present in species which contribute to global marine fisheries [6]. Accordingly, microplastics pose an emerging food safety concern [6]. International scientific committees such as the Joint FAO/WHO Expert Committee on Food Additives (JECFA) has not evaluated the food safety concern posed by microplastics [6]; however, state-level environmental protection agencies have begun assessing the public health implications of microplastics and nanoplastics [38].

Human health effects depend on exposure concentrations. Due to data gaps in microplastic research, there is insufficient information to assess the true amount of microplastics humans may be exposed to via food. Researchers have predicted that the total microplastic intake from salts is at most 37 particles per individual annually [37]. Researchers have also estimated that a top European shellfish consumer eats approximately 11,000 plastic particles annually [23, 32, 37]. The implications are unknown.

Microplastic exposure can confer exposure to associated chemicals. Few studies have assessed the relative contribution of microplastic exposure to additives or chemicals found in organisms, versus alternative exposure pathways [39]. The EFSA monitors six indicators for non-dioxin-like PCBs in food to assess average total dietary exposure to PCBs. While the portion of exposure from microplastics is unknown, fish, meat, and dairy contribute the greatest dietary exposure to PCBs, demonstrating a route of persistent exposure to animal tissue and trophic transfer [40]. It was determined that total dietary PCB intake ranged from 1 to 83 ng PCB/kg bodyweight (bw) per day [41]. The average dietary intake of PAHs, using benzo[a]pyrene as the reference marker, ranged from 4 to 10 ng/kg b.w. per day [41].

The US FDA residue limit for PCBs in fish and shellfish is 0.2 ppm for infants and juveniles and 2 ppm for adults, corresponding to developmental effects, hormonal disruption, immune system, thyroid effects, and cancer [40]. The FDA has not established a limit governing the concentration of PAH content in foodstuffs [42]. In animals, the US Environmental Protection Agency has identified a reference dose for oral benzo[a]pyrene exposure, the most studied PAH, at 0.0003 mg/kg/day [42]. The oral reference dose applies to food and water and estimates the concentration at which adverse effects on human health are known to occur. Additional studies are needed to understand the biological processes influencing the release of chemicals associated with microplastic ingestion, and all routes of chemical exposure [43].

Today, evidence is mounting suggesting that microplastic ingestion or its associated chemicals pose a threat to marine animals [9, 14–41]. Understanding whether microplastic exposures impact human health requires standardized and reproducible methods for sampling, exposure characterization, ecological assessment, and human health assessment. There is no standard operating procedure for sampling occurring on beaches, in subtidal sediments, in biota, or within the water column.

Toxicity to Humans

Microplastics may cause harm to humans via both physical and chemical pathways. While it is not possible to completely disentangle these, we separate them for the purpose of this discussion.

Potential Physical Effects of Microplastics

Microplastics are ubiquitous in the marine environment and are increasingly contaminating species in the marine environment. Given levels of seafood consumption worldwide, it is inevitable that humans are exposed to microplastics at some level. The human body’s excretory system eliminates microplastics, likely disposing of > 90% of ingested micro- and nanoplastic via feces [14–41]. Factors affecting retention and clearance rates are the size, shape, polymer type, and additive chemicals of microplastics ingested by humans [6]. The severity of adverse effects resulting from exposures depends on the nature of the toxic chemical, exposure characteristics, individual susceptibility, and hazard controls. The physical effects of accumulated microplastics are less understood than the distribution and storage of toxicants in the human body, but preliminary research has demonstrated several potentially concerning impacts, including enhanced inflammatory response, size-related toxicity of plastic particles, chemical transfer of adsorbed chemical pollutants, and disruption of the gut microbiome [44].

Surface functional groups, size, shape, surface charge, buoyancy, and hydrophobicity predict microplastic uptake [45]. Mammalian systems modeling suggests that microplastics with certain characteristics can translocate across living cells, such as M cells or dendritic cells, to the lymphatic and/or circulatory system, accumulate in secondary organs, and impact the immune system and cell health [14–41, 43, 45–51]. Microplastics may contact the airway or gastrointestinal epithelium demonstrating several routes of uptake and translocation, such as endocytic pathways and persorption [44]. Medical literature related to the impact of micro- and nanoplastics originating from surgical procedures and inhalation provides insight into the kinetic movement of plastics in humans [6]. For example, micro- and nanoplastics released from surgical materials mimic the effects of absorbed particles in the bloodstream and tissue [6], while inhaled particles interact with the same type of epithelial tissue as that involved during ingestion. For example, microbes colonized on the surface of ingested microplastics may serve as a vector of harmful bacteria when ingested, potentially resulting in direct...
physiological effects (nutritional, toxicological, immunological, or developmental) on marine animals. Wright and Kelly predict that ingested microplastics may cause inflammation in tissue, cellular proliferation, and necrosis and may compromise immune cells [44•]. While laboratory research has demonstrated that plastic microspheres ingested by blue crabs (Callinectes sapidus) stimulate hemocyte aggregation and reduce their respiratory function [52]. Moreover, after ingesting microspheres, blue mussels experienced an immune response and the formation of granulomas [53]. The Japanese medaka (Oryzias latipes) experienced hepatic stress after ingesting virgin polyethylene fragments [54]. Factors influencing the biological and ecological impact of microplastics include presence, sizes, and frequency of engagement between biota-microparticles. More research is needed to further inform a risk assessment of the impact of microplastics on seafood and consequently human health. It would be valuable to conduct a risk analysis monitoring microparticles and the related chemical concentrations in seafood, particularly shellfish to identify the potential biological consequences of microplastic exposure. Additionally, in this stage, it is important to monitor consumer consumption rates of seafood, particularly bivalves. This information will inform a risk evaluation and management or mitigation strategies connecting sources and drivers of microplastic pollution. This approach integrates a systems perspective that employs precautionary measures to reduce the threat of harm posed by microparticles to the environment and to humans given present uncertainty.

Nanoplastic movement provides insight into the movement and potential effects of non-degradable particles in the human body. The potential health risks of micro- and nanoplastics could be evaluated similar to those of engineered nanoparticles [14••]. Following oral exposure, nanoplastics are transported by M cells, specialized epithelial cells of the mucosa, from the gut into the blood where they are carried through the lymphatic system and into the liver and gall bladder [55]. Their size and hydrophobicity enable their passage through the placenta and blood-brain barrier and into the gastrointestinal tract and lungs, potential sites for harm to occur [56]. Their large surface area to volume ratio makes them potentially very chemically reactive, more so than some microplastics. Research studies have demonstrated toxicity in vitro to lung cells, liver, and brain cells [9•]. The systemic distribution from oral exposure to nanoplastics has been shown to have numerous effects: cardiopulmonary responses, alterations of endogenous metabolites, genotoxicity, inflammatory responses, oxidative stress, effects on nutrient absorption, gut microflora, and reproduction [14••, 36, 46]. Parallel research into nanoparticle movement and toxicity provides insight into threats posed by microplastics and nanoplastics.

Potential Effects of Chemical Additives

Chemical additives in plastic may cause toxic effects. Moreover, the ability for microplastics to accumulate POPs raises concern that microplastics could transfer hazardous POPs to marine animals and subsequently humans [6]. Chemical partitioning between microplastics and animal tissue is a dynamic process; there are few studies that model variables and mechanisms like bioaccumulation, kinetics, and the physicochemical properties of marine microplastics [57].

Direct exposures to POPs and other chemicals associated with microplastics may affect biological systems and pose specific threats to juvenile humans and animals, including at low doses [9•, 40]. Current guidelines for toxicity testing of chemical components use high contaminant concentrations from a single substance to estimate risk at lower exposure levels or to make low-dose extrapolations. This method fails to capture concerns related to low-dose contaminants or mixed groups of contaminants. Additionally, this method makes it challenging to account for non-linear dose relationships. As a result, these methods fail to generate data that captures the potential threat posed by chemicals associated with microplastics.

Ingestion is a common interaction between biota and microplastics. The fate and impact of microplastics and their associated chemicals vary across species and environments [6]. Laboratory studies demonstrate increased toxicity from the combination of microplastics and associated chemicals [51, 58]. It is difficult to evaluate whether toxicological impacts translate to humans, however [59]. In animals, the quantity of chemicals from microplastics is suspected to be minimal compared to that from other components of the diet [6]. Microplastics and their constituents may exert localized particle toxicity, but chronic exposure producing a cumulative effect is of greater concern. In summary, further work is required to estimate the dose of chemicals to humans from microplastics in seafood and the related effects, including studies of seafood intake, chemical characterization in seafood, and kinetic studies.

Epidemiology

In human medicine, microplastics are used as carriers of medications into body tissues [60]. A report commissioned by the House of Commons Environmental Audit Committee of the UK Parliament speculates that the additives and contaminants of concern, when adsorbed to marine microplastics, would act similarly to micrometals used in medical procedures, which transfer to human tissues [60], though there is insufficient data demonstrating this [61].
We do not fully understand how microplastics interact with human biological tissue. For example, if there is an adverse interaction, the effects may be apparent and significant to the individual, but without sufficient and extensive epidemiological studies, impacts may be difficult to detect at a population level. There is a significant correlation between urine BPA levels and both cardiovascular disease and type 2 diabetes [62]. BPA exposures in humans occur both from low-dose exposures to microplastics and both low- and high-dose exposures from non-microplastic sources via inhalation of air and dust or ingestion of foodstuffs. Research is needed to thoroughly assess the risk of microplastics and nanoplastic dietary exposure.

Microplastics and their constituents may exert localized particle toxicity, but chronic exposure producing a cumulative effect is of greater concern. To address research gaps, it is recommended that scientists evaluate the relative impact of microplastics as an exposure pathway. Further, it would be valuable to identify sorbed contaminant bioavailability and use biomonitoring methods to contextualize safe toxicological exposure parameters for chronic exposure to microplastics and their constituents [7].

**Mitigation of and Adaptation to Risks**

The above sections have described the state of evidence linking microplastics to potential human and animal health risk. Microplastics, chemical toxicity, and chronic exposure to microplastics may pose risk to human health, especially with increasing direct exposure to plastic and localized chemicals. And, while significant gaps remain, complimentary bodies of evidence indicate likely exposures and potential hazards from both particles and associated chemicals. The impact of microplastics on human health is uncertain, but cannot be ignored, and presents one justification to mitigate the increasing influx of plastic into the environment.
Governments, industry, and civil society all have important roles to play.

Multiple global agreements and domestic policies govern protection of the marine environment; Table 4 identifies several notable policies. Since the enactment of The United Nations Law of the Sea in 1982, a coastal country has sovereign rights extending 200 nautical miles from its shoreline. It is, therefore, the responsibility of governments in those locations to determine who may use this area and how. With diverse cultures, priorities, and opinions present in each coastal country, levels of protection differ considerably.

Industry also plays a critical role in reducing microplastic prevalence throughout the supply chain, in the form of primary microplastics used in industrial processes and secondary microplastics. Extended producer responsibility (EPR), a stewardship policy targeting corporations marketing consumer goods, holds manufacturers responsible for the post-consumer phase of plastic packaging [68]. IKEA, for example, has integrated EPR policies into its business model by promoting material reuse and recycling throughout its supply chain and consumer experience. The company indicates in their Sustainability Summary Report F17 that 590,258 t of waste was produced in 2017 across their supply chain of which 83% was recycled or incinerated for energy recovery [69].

Other companies are utilizing focused upcycling strategies in their supply chain by directly removing, recycling, and reclaiming plastic from the marine environment to create textile fibers which are then processed and manufactured into yarn for consumer goods. Adidas, for example, partnered with Parley for the Oceans in 2015, to manufacture sneakers and clothing from plastic pollution in the Maldives using a zero-waste 3D printing technique. In 2017, Adidas sold one million pairs of Parley collaborated shoes, equivalent to 16.5 million plastic bottles and 14.3 t of nylon gill nets [70, 71]. Unifi, Bureo, CityPlace, Method, and G-Star RAW clothing have also taken steps to reduce ocean plastic pollution through Ocean Plastic Programs [A.I.R., Avoid, Intercept, and Redesign] [72]. While these are not EPR stewardship policies, A.I.R. is a step in the right direction.

Another approach to mitigation is beach cleanup programs. These are generally organized by nongovernmental organizations (NGOs) globally and aim both to raise awareness about marine debris and to remove materials that could cause harm and gradually degrade into microplastics. The International Coastal Cleanup (ICC) coordinated by the Ocean Conservancy, a US NGO, is one of the largest operational organizers of these programs, providing significant financial and social input [64]. The ICC engages 70 countries globally in an annual September weekend litter survey and beach clean-up activity [64]. From the 2016 event, 790,000 volunteers participated in collecting 18 million pounds of trash across over 25,000 miles of shoreline [64].

The extent to which these efforts influence marine plastic pollution or protect the environment is unknown. It is also unclear how measures aimed at preventing plastic pollution leakage compare with reactive measures such as beach cleanups, in terms of cost-effectiveness.

Conclusions

We know that humans ingest microplastics. Considering the totality of research findings on microplastics to date, we know that shellfish and other marine organisms consumed with intact GI tracts pose particular concern because they accumulate and retain microplastics. The toxicity associated with consuming microplastics is likely dependent on size, associated chemicals, and dose. Our collective understanding is limited regarding the sources, fate, exposure, bioavailability, and toxicity of microplastics and their associated chemicals in the marine environment. Current knowledge is mostly based on research conducted within the last decade; however, interest in studying microplastics is growing. The following are key research needs for microplastics and their effects on human health:

- Assess microplastics’ impact on ecological systems and food safety and improve understanding of potential toxicological mechanisms and public health effects.
- Identify, if possible, lower risk species, production methods, or regions, and interactions of microplastics with nutrients and various seafood processing and cooking methods, in order to promote adjustments rather than consumer avoidance of seafood.
- Standardize data collection methods for microplastic occurrence in the environment and food stuffs, followed by exposure assessment for dietary intake.
- Standardize data collection assessing major seafood production types and seafood producing countries.
- Collect data on presence, identity and quantity of degraded plastic in food, and data on the translocation of microplastics through the aquatic food web and human food system.
- Develop methods to assess physical and chemical changes of micro- and nanoplastics when interacting with biological systems.
- Collect toxicity exposure data evaluating mixtures of various additives/monomers.
- Collect toxicological data on the most common polymers and their relative contributions to microplastic contamination.
- Develop specific biomonitoring processes and body burden measurements for additives and monomers.
- Research the toxicokinetics and toxicity of micro- and nanoplastics and their associated chemical compounds, to determine local gastrointestinal (GI) tract effects in animals and humans.
While much remains to be learned, filling these gaps is essential for advancing the dual goals of promoting seafood consumption and protecting consumers from negative health effects from microplastics in the marine environment.

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Compliance with Ethical Standards

Conflict of Interest Madeleine Smith, David C. Love, Chelsea M. Rochman, and Roni A. Neff declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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