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Further Studies in Translatable Model Systems are Needed to Predict the Impacts of Human Microplastic Exposure

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Abstract

Microplastics are a pervasive environmental contaminant that have been found in many media including water sources, soils, and foodstuff. Due to the worldwide presence and persistence of microplastic debris, human exposure is inevitable. Human exposure occurs predominantly through ingestion, although dermal and inhalation exposures are probable. Microplastic single exposure studies in aquatic species and fish have shown various toxic effects including those on reproduction and survival. In addition to potential intrinsic toxicity, microplastics often have chemicals adsorbed to their surfaces. Studies report that these chemicals can have innate toxicity that is modulated by the composition of microplastics. Both the impacts of microplastics alone and co-exposures with adsorbed chemicals exhibit size dependent effects. Analysis of the current literature has revealed published studies predominantly investigate the toxicity of microplastic exposure in fish and other aquatic species, with limited knowledge about the effects in mammals and cell lines. Toxicity has been shown to vary widely between taxonomic groups, suggesting inferring human health relevance will require model systems where human routes of exposure can be mimicked. Although it may be difficult to extrapolate the results from aquatic model systems to relevant human health impacts, they may suggest effects to investigate. In order to best estimate the short- and long-term impacts of human microplastic exposure, it is imperative that studies in model systems with increased similarity to human anatomy and cellular processes be done.

Origins of Microplastics

Since the introduction of plastic products in the early 1900s, plastic use and therefore waste has continued to increase. The mismanagement of plastic debris frequently results in plastic pieces, including microplastics, entering the environment [1]. Microplastics are a ubiquitous environmental problem and have been detected on all seven continents [2]. In addition to being found worldwide, microplastics have been detected in numerous forms of media including but not limited to oceans [3], lakes [4], drinking water [5,6], sediments [7], soils [8], sugar [9], and table salt [10]. The prevalence and persistence of microplastics in the environment is likely due to their physical properties.

Microplastics are small pieces of plastic less than 5 mm in size [11-13]. There are two main types: primary and secondary microplastics. Primary microplastics are those that are manufactured at the microplastic size. This includes those that are produced for use in consumer products, as well as those that are byproducts of other goods. Products that intentionally contain microplastics include personal care products [14,15], drug delivery systems [16] and air blasting media [17], while synthetic clothing [18] contains unintentional microplastics. Secondary microplastics are those that are produced through the environmental degradation of larger pieces of plastic debris through processes such as physical abrasion [19] and photodegradation [20]. Recently, researchers in Italy reported a potential new class of secondary microplastics. They found that some benthic crustaceans are able to break ingested plastics into smaller pieces, producing microplastics [21]. The small size of microplastics in addition to their properties, such as resistance to corrosion, that make plastics desirable product components contribute to their pervasiveness in the environment [22]. Microplastics can further be degraded to nanoplastics (<0.1 μm), but particles <1.0 μm are rarely collected in environmental studies, further pointing to a knowledge gap in understanding the health impacts.

Routes of Human Microplastics Exposure

Due to the widespread nature and persistence of microplastics, human exposure is inevitable. Humans are exposed through ingestion and possibly through dermal exposures and inhalation. Ingestion occurs via activities including consumption of aquatic species containing microplastics and drinking and cooking with microplastic contaminated water. Microplastics have been detected in aquatic species such as fish [23] and mussels [24]. Organisms may confuse microplastics with food, especially if their food sources are on the same size scale as the microplastics present [25], or accidentally ingest microplastics while feeding or drinking [26]. Additionally, microplastics have been observed to transfer up trophic levels in the environment [27,28], and this property is what allows humans to uptake microplastics from food. Dermal contact with microplastics is a less likely route of exposure, but it is possible that swimming or showering in water that contains microplastics could result in microplastic uptake [29]. This is more likely if an individual has a barrier defect in their epidermal layer, such as those that occur as a result of UV radiation exposure [30]. Lastly, microplastics have been detected in samples of both indoor and outdoor air [31], suggesting humans may be regularly inhaling microplastics. Given the likelihood of human exposure, it is vital that translatable studies be performed.

Impacts of Microplastics on Biological Systems

Microplastics have been studied in a variety of model systems, however the literature shows a bias towards fish and other aquatic species. These single exposure studies have demonstrated a wide variety of toxic effects. An inexhaustive list of these effects includes an increase in reactive oxygen species (ROS) production in copepods and rotifers [32,33], a disruption of lipid homeostasis due to a decrease in HDL levels in catfish [34], and a decrease in intestinal calcium concentrations in *Caenorhabditis elegans* [35]. Additionally, microplastics were observed to exhibit size dependent effects. Exposure to smaller microplastics in *Tigriopus japonicus*, a small aquatic crustacean, and the rotifer, *Brachionus koreanus*, resulted in greater reductions in survival [36] and lifespan [33], respectively, when compared to exposure to larger microplastics. Despite this variety in effects observed, these studies do not vary widely in model system used.

In addition to the plastic polymers that comprise the microplastic itself, many microplastic particles contain hydrophobic organic contaminants (HOCs) adsorbed to their surfaces. Some are chemical additives introduced during the manufacturing process to impart desired properties to the plastics being produced, such as plasticizers, flame retardants and pigments [37]. Additionally, once in the environment, microplastics tend to accumulate other contaminants such as metals [38], bacteria [39], and persistent organic pollutants (POPs) [40]. On their own these contaminants exhibit toxicity that may be modulated by co-exposure with microplastics. The results of microplastic-contaminant co-exposures reveals the toxic effects observed are distinct from exposure to the microplastics alone. A 2018 review that characterized results based on taxonomic group, plastic type, and contaminant present suggests that the toxicity of the contaminant is modulated by microplastic type present [41]. For example, when studies in Mollusca were analyzed the effect profiles of polycyclic aromatic hydrocarbons (PAHs) in the presence of polyethylene (PE), polystyrene (PS), or polyvinyl chloride (PVC) exhibited different toxic effects [41]. Reasons for this were not given but it is likely related to the solubility and permeability of the PAH in the bulk plastic. Similarly, to exposures to microplastics alone, co-exposures with contaminants show size dependent effects. A study that measured the estrogenic equivalency of populations of different sized microplastics containing endocrine disrupting contaminants, revealed a general trend where an increase in estrogenic activity was associated with a decrease in microplastic size [42]. Just like the microplastic single exposures, there is a clear bias in the literature towards studies in fish and aquatic species.

The general consensus thus far is that microplastics induce toxicity alone and in co-exposures. Because of the lack of mammal or cell exposure studies this conclusion cannot yet be applied to humans. A survey of the primary literature results of a Google scholar search of "microplastics toxicity" revealed 71% of the articles analyzed studied fish or other aquatic species. This same survey found only three and five percent of articles analyzed studied effects in cell lines and mammals, respectively. This is especially problematic as a different 2018 review that compiled the results of 43 studies of microplastic exposure found that effect sizes varied widely between taxonomic groups [43], suggesting it is extremely imperative a proper model system be used to estimate effects of human exposure. Additionally, the emerging routes of exposure of inhalation and dermal exposure cannot reliably be studied in aquatic species due to their lack of skin or lungs comparable to those of humans. Therefore, it is imperative that microplastic exposure studies be performed in a model system with relatively conserved biological processes, such as barrier function or cell signaling, and organ systems compared to humans. Without using these types of model organisms, it is a stretch to apply knowledge gained from studies when hypothesizing the short- and long-term effects of human microplastic exposure.

Existing studies using human cell models are sparse [44-46] and they use model particles rather than testing materials recovered from the environment. Overall, the conditions tested in these studies showed little to no impacts on cell viability with microplastic exposure, with the exception of human dermal fibroblasts treated with 3 μm polystyrene particles at a very high dose of 1000 μg/ml microplastics [45]. Beyond viability the cellular effects observed varied between studies. ROS production was elevated in response to microplastic exposure in once study [46], while another found all but one exposure had no significant

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effect on ROS production [44]. Similarly, cytokine and histamine secretion varied widely between treatments [44,45]. These inconsistencies between human cell studies further motivate the need additional comprehensive studies with both commercial and environmental microplastic particle samples.

Conclusion

Microplastics are a ubiquitous environmental contaminant that inevitably leads to human exposure. Despite the high likelihood that humans are exposed, the majority of research on microplastic exposure has focused on the effects in aquatic species. Exposure to microplastics with and without the presence of organic contaminants has been shown to result in numerous toxic effects in these aquatic species. However, due to the routes of human exposure and the variability in toxicity observed between taxonomic groups, the amount of information that can be used to estimate the effects of exposure to microplastics in humans is extremely limited. In order to gain results that are more relevant to human health outcomes, proper model systems must be used. A couple systems that would be more appropriate are primary human cell lines and laboratory mice. The results observed in the literature thus far may not be applicable to human health but may provide endpoints to investigate in relevant systems. Further studies are needed in order to best understand the effects of microplastic exposure in humans.

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