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Should we worry about the accumulation of microplastics in human organs?

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Since the second half of the 20th century, we have entered the "plastic age", a new era where plastics represent the majority of man-made materials. In a few decades, 8,300 million metric tons of plastics have been produced, and the subsequent plastic wastes have been accumulating in marine, freshwater and terrestrial habitats, following their own biogeochemical cycle. At the current rate of production, it is predicted that more than 10,000 million metric tons of mismanaged plastic wastes will be dispersed in the natural environment by 2050.[1]

Large plastic wastes can have direct negative effects on wildlife through different mechanisms, including the ingestion of plastic debris. At least 1,565 wildlife species, living in different environments, have been documented to ingest plastic remains.[2] What about us? Is plastic also in the daily menu of humans? Large plastic wastes released in the natural environment do not entirely degrade but break down in smaller particles, becoming micro (<5 mm) and nanoplastics (<1 µm). Evidence is now rapidly accumulating showing that we do ingest plastic specks on a daily basis, and recent work even suggests that this can represent thousands of particles per day.[3]

The finding that we are exposed to microplastics in our diet has been corroborated by the detection of plastic particles in human stools.[4] But should we be worried about it? After all, we could see plastic debris as inert particles that merely transit through our body with no effect. This view has been challenged by evidence showing (among others) that i) microplastics are not inert particles (e.g., they can release additives, plasticizers and other toxic compounds) and can carry pathogenic micro-organisms with antimicrobial resistance genes; ii) microplastics disrupt the integrity of the intestinal barrier and can be uptaken into the bloodstream.[5,6] Therefore, ingested and inhaled microplastics can potentially be trapped and accumulate in different tissues and organs, such as lungs or placenta.[7] Whether the amount of microplastics found in human organs is enough to produce health damaging effects is, however, still unclear, although the smallest particles (nanoplastics) might actually enter cells, trigger the inflammatory response and interfere with the normal cellular activity.

The findings reported by Horvatits et al.[8] in this issue of eBioMedicine add to the previous results of microplastic accumulation in human organs and tissues. Although this work should be taken with caution due to the small sample size, it takes a step forward in our understanding of the potential negative effect of microplastic accumulation. One of the challenges we have to face when assessing microplastic contamination of human tissues is to make sure that any particle found in the samples does not come from background contamination. Plastic objects are so pervasive in our daily life that background contamination is a critical and serious issue when assessing microplastic pollution in biological samples. Horvatits et al. assessed microplastic contamination in liver, spleen and kidney samples. The number of plastic particles ranged from 0 to 2.2 per gram of healthy tissue, but this was indistinguishable from background contamination of blank samples, which also ranged from 0.2 to 2.2 particles. However, liver samples from patients suffering from liver disease (cirrhosis) had a 8-fold increase in plastic contamination compared to blank and to liver samples from healthy individuals [median number of particles per gram of tissue = 8.4 vs 0.6 (blank), and 8.4 vs 0.7 (healthy liver)].

Why do patients with liver disease accumulate more plastic particles in the liver compared to healthy individuals? The sample size and sampling scheme do not allow establishing a causal relationship between microplastics and liver pathology. It could well be that the higher number of plastic particles recovered from cirrhotic livers is a side-product of the pathology and not the actual triggering factor. Nevertheless, even though microplastics alone might not trigger liver disease, it cannot be excluded that they might contribute to accentuate and exacerbate liver fibrosis.[9]

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Several questions are still pending and will require larger case series to properly address the relation between cirrhosis and microplastic accumulation. Is there a dose-dependent effect? The number of particles per gram of tissue was quite variable within the limited samples of diseased livers (ranging from 4.6 to 11.9) and we might reasonably wonder to what extent this is a relevant parameter to take into account. A related question involves the sources of variability in microplastic contamination. Does this come from differences in exposure among individuals? Diet and lifestyle in general are key factors triggering chronic liver disease, and the amount of ingested microplastics also depends on our feeding habits (e.g., highly processed food, plastic packaging).[10] Therefore, teasing apart the contribution of different geographic and socio-economic factors, and establishing the role (if any) of microplastic pollution as a possible cause of human pathologies will be one of the challenges we will face in the upcoming years.

Contributors

Both authors contributed to the writing of the manuscript.

Declaration of interests

The authors have no conflicts of interest to disclose.

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