Research Article

Potential Health Risks of Ingesting Microplastics Measured in Crops and Recommendations for Reducing Them

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Abstract

Plastics are an integral part of modern life and are essential to agriculture and food packaging. “Plastics” refers to an immense number of materials that are created from different polymers and an estimated 13,000 chemical additives. While the presence of plastics in the environment has been a concern for decades, their breakdown products, micro- and nanoplastics (MNPs), are an emerging human health and environmental concern. Recent studies on microplastic contamination in our air, water, land, food, and bodies provide the data needed to begin evaluating the resultant health effects and creating exposure reduction strategies. In this work, we utilize a mixtures hazard index (HI) method to identify harmful additives found within plastics identified in agricultural products. We illustrate the relative potency of a few toxic substances within microplastics such as DEHP, which is possibly 2,000 times more toxic than available potential substitutes. Additionally, we demonstrate that a few toxic additives can result in an elevated hazard index for the amount of microplastics detected in crops. Thus, the hazard index method can be applied to aid in the development of a new generation of plastics with lower toxic potential.

Introduction

Plastics have made life safer in many ways. In the agricultural sector alone, they help preserve foods, ensure seed integrity, prevent glass-related injuries, reduce the transmission of diseases, and protect products from decay [1]. Nearly all commercial plastics are complex mixtures of polymers, which are made up of joined repeating units known as monomers, and many additional chemicals not bound to the polymer backbone. When monomers are not fully incorporated into the polymer, they can be released as the plastic breaks down and cause unanticipated harm to the environment and health. When the monomers are completely polymerized, or cured, the toxicity is greatly reduced. Over the decades plastics have been engineered to increase their utility through a combination of monomers and a variety of additives. As usage demands changed, many plastics have been re-engineered [2]. However significant health risks from the chemicals commonly used in plastics is still a pressing concern that requires further consideration and re-engineering.

The discovery of cancers caused by uncured monomers, led to more efficient curing coupled with burning off the residuals [3,4]. The understanding that di(2-ethylhexyl)phthalate (DEHP) causes developmental and endocrine (hormonal) effects has led to laws in California and North Carolina preventing its use in some medical devices and toys [5,6]. Similarly, the FDA recommends that DEHP not be used in medical devices and toys [7]. Internationally, the European Union (EU) has taken appropriate steps towards its elimination [8]. Currently, the EPA is seeking to limit DEHP use in the USA for the protection of workers and the environment [9].

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Abbreviations
mg/kg/day: milligrams per kilogram (body weight) per day; DEHP: di(2-ethylhexyl) phthalate; LOAEL: lowest observed adverse effect; DBP: di-n-butyl phthalate; DEP: diethyl phthalate; MNPs: micro- and nanoplastics; MRL = minimum risk level; ppm = parts per million

As a result, DEHP's use has been eliminated in some products. However, DEHP continues to be widely utilized, despite being possibly 2000× more toxic than available substitutes that provide the same physical characteristics, such as flexibility, to the plastics [10,11]. For example, the lowest observed adverse effect (LOAEL) for DEHP after 20 days of oral exposure is 0.04 mg/kg/day [12]; the LOAEL for 13 weeks of oral exposure to DBP (di-n-butyl phthalate) is 80 mg/kg/day [13]; and the LOAEL for DEP (diethyl phthalate) for 3 weeks exposure is 1,753 mg/kg/day [14]. The differences between these LOAELs are striking; the LOAEL of DEHP is (80/0.04 =) 2000 times lower and for a period that is almost (91/20=) 5 times shorter than DBP. The simplified comparison of the LOAELS is one step.

Since the lowest published LOAEL does not necessarily reflect the severity of the health effects for each substance, one can review the severity of the collective effect levels within each chemical's toxicological profile [12-14]. When available, we selected government summaries of the peer-reviewed LOAEL literature (rather than the individual studies) as the agencies undergo additional validation of the studies for the development of policy. This allows us to conclude that DEHP is much more toxic. One should also consider the duration of exposures when comparing relative toxicity. In this case, the DEHP LOAEL duration is 20 days and comparable to the DBP LOAEL duration of 3 weeks but is much shorter than the 13-week duration for the DEP LOAEL. While the DBP and DEP profiles are dated, there are no lower LOAELs identified in the most recent US government assessments [15,16]. Other substitutes are even less toxic [17-22].

In the US, the current policy is to recommend limitation of DEHP but not to ban it. This is concerning with the current plastic waste practices as DEHP remains in the plastic after the product is no longer in use and breaks down into micro- and nanoplastics (MNPs) in our environment [3,23,24]. Microplastics are defined as plastic particles <5 mm in size; nanoplastics are between 1 and 1,000 nanometers. Due to their small sizes, MNPs are impossible to remove completely once they are present [3,4].

The fate and transport of plastics, and the resulting MNPs, in the agriculture industry is troublesome. MNPs are in direct contact with seeds, found in the compost applied to plants, present in grey water sometimes used for watering, and have been found within each part of the hydrologic cycle [23-26]. There is new information to suggest that MNPs even reduce a plant's ability to photosynthesize [27]. The use of plastics will result in human exposure warranting minimization of the toxicity of the plastics. While the true toxic effects of MNPs exposures remains uncertain, we know that some components in certain MNPs are harmful. [3,4,28,29]. The simultaneous or sequential exposure to many of these components can become a mixtures exposure problem with several of the chemicals impacting common health endpoints. Preventing all unnecessarily toxic additives in plastics is one important step in making our one world safer for people and the environment.

This manuscript aims to provide a method to identify and prioritize harmful components found in many plastics and microplastics and to utilize recent data on microplastics found in crops to calculate potential health risks. We do this by applying two related strategies to identify and quantify the harmful components within plastics: 1) observed adverse effect levels and 2) hazard quotients/hazard index. We then apply the latter strategy to calculate if exposures to MNPs in crops are at or below levels that are theoretically safe. The results and strategy can be directly applied to assess potential risks from microplastics measured in other foods with the recent reporting of microplastic mass content rather than particle count. This method, in addition to providing a framework for determining the theoretically safe exposure levels, identifies the critical data gap of the actual mass content of the components within the microplastics.

The objectives of this manuscript are: a) to utilize recent data on microplastics found in crops to calculate potential health risks, b) to provide a hazard index framework to identify and prioritize harmful components found in many plastics and microplastics, and c) to demonstrate how the hazard index approach can be used to evaluate plastic component alternatives to reduce the risk to humans. The hazard index framework is used to calculate if exposures to MNPs in crops are at or below theoretically safe levels. While eliminating plastics in agriculture is not feasible due to their numerous benefits, substituting particularly harmful components with safer alternatives is a feasible goal which will be aided by the techniques presented here. The results and strategy can further be applied to assess potential risks from microplastics measured in other foods if microplastic mass content data is available. However, as this work highlights MNPs, mass data remains a critical data gap. Throughout this manuscript, we use MNP and MP when addressing data from other studies because most studies only measured the MP portion. While the MPs make up the majority of the mass for dose calculations, the fact that we needed to rely on both data sets, highlights the nano plastic data gap.

Discussion

Quantifying health impact of MNP in food crops

In 2020, the National Academies of Sciences, Engineering and Medicine (NASEM) hosted a microplastics workshop to identify early actions to protect public health [30]. At the workshop, an example was provided of the relative risks of antimony (Sb), a heavy metal commonly used in plastics, within microplastics found in air and water using a hazard quotient/hazard index approach [31].

When estimating a mixture's health risk, one must consider all the components of the mixture and their duration of exposures. The most commonly used method to estimate the toxicity of a mixture is the hazard index approach [32,33]. This method evaluates a mixture's components individually using each substance's known hazardous properties and concentrations to calculate a hazard quotient (HQ) for each component. The HQ is defined as the ratio of its exposure and allowable level. Then, a summation of the HQs of all components of a mixture gives the hazard index (HI), which indicates the safety of exposure to the mixture overall. A HI is a ratio of the exposure to a theoretically safe level, defined as 1. Thus, a HI<1 means that the exposure is theoretically safe. While a HQ or HI value >1 does not always mean that the exposure is unsafe, it is cause for concern, and it supports exposure reduction or the need for further analysis. It is especially prudent to limit exposures if only one component in a mixture has a HQ>1. The HIs of various mixtures can then be used to compare and thus prioritize the mixtures with the highest toxicity.

There are many factors in applying the mixtures method, such as how the duration of exposure is addressed and the uncertainty of the studies from which a dose-response is developed. As with the NASEM presentation, here we use exposures of less than one year, termed intermediate exposure, and protective community-based uncertainty as explained in the Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles (explained in appendix C of any of the referenced toxicological profiles) [34]. Our calculations use an intermediate minimum risk level (MRL) for each substance from the toxicological profiles.

NASEM Workshop Calculation

The example at NASEM identified that if microplastics (MPs) found in drinking water were made entirely of antimony (Sb), they would have an HQ >1 given an exposure of:

Concentration of MPs in drinking water = 9 MPs/L, as found by Kosuth et al. [34].

Daily particle exposure for people from drinking water = 9 MP/L x 3 L of water/day = 27 MPs/day

While no mass was provided, Kosuth et al., did report MP sizes. These were used to calculate 4 size categories, which results in a mass estimate of 1.1 mg for the 27 MPs/day [30].

Dose of MPs in water for an 85 kg (body weight) person = 1.1 mg/day/85 kg = 0.013 mg/kg/day.

Intermediate MRL for Sb = 0.0006 mg/kg/day.

$$For\ 1.1\ mg\ Sb\ /\ kg\ /\ day,$$

$$HQ = \frac{Dose\ of\ MPs\ in\ water\ for\ 85\ kg\ person}{Intermediate\ MRL\ for\ Sb} = \frac{0.013\ mg\ /\ kg\ /\ day}{0.0006\ mg\ /\ kg\ /\ day} = 22$$

A HQ of 22 is well over the theoretical safe value of 1. However, plastics contain many more components than just Sb, and the mass calculation included many assumptions in that case [30]. Therefore, this example could underestimate risk and is not an accurate evaluation of the safety of MPs in drinking water. The same NASEM presentation also identified the numerous data gaps that needed to be addressed in order to accurately assess if MNP mixtures are safe, especially mass measurements.

Quantifying individual components in agricultural food crops and evaluating health effects

Several studies since 2020 have filled some of those data gaps, including measurements in water, air, cabbage, carrots, broccoli, spinach, and apples, which allows us to assess several components within the microplastic mixture [34-41]. However, it is important to note that most studies focus exclusively on microplastics and neglect nanoplastics due to difficulties in isolating and measuring them. We will discuss assessment methods that address these issues. The Mixtures Method framework is not only a proven way to quantify the toxicity of plastic products but can be directly applied to agricultural products in which microplastic contamination has already been measured.

In doing so, common agricultural products that have absorbed microplastics via air, water, or soil can be evaluated for potential human risks they pose. As an example of applying the HI method, we looked at the exposure of substances within MPs found in and on cabbages. The highest mass concentrations of MPs currently reported are: 19.84 µg/g in cabbage grown on small farms [38].

Given that a bowl of cabbage is about 50-100 g, we calculate the following mass of microplastics as:

50-100 g (Cabbage wt.) x 19.4 µg/g = 0.970-1.9 mg of MPs.

DEHP, cadmium, and lead are common components of plastics known to cause reproductive and developmental effects. Thus, a plastics mixtures assessment involving all three substances can be appropriate. Exposure calculations and summaries of the toxicity for each is listed below.

DEHP exposure calculation

Applying the common phthalate (DEHP in particular plastics) percent ranges in plastics of 20-80% [12,42,43], we can calculate phthalate exposures. This gives:

A phthalate range of 0.194-1.6 mg/bowl of cabbage.

For a 30-90 kg individual, the dose range becomes: 0.0022- 0.053 mg/kg/day.

Applying the DEHP intermediate MRL of 0.0001 mg/kg/day, the maximum HQ range becomes:

$$\frac{Dose\ of\ DEHP\ per\ bowl\ cabbage}{Intermediate\ MRL} = \frac{0.0022\ to\ 0.053\ mg/kg/day}{0.0001\ mg/kg/day} = 22\ to\ 530$$

Using the maximum values suggests that one can get an MRL dose by eating 0.036 - 4.8 mg of the referenced cabbage.

Table 1 lists the intermediate MRLs and similar EPA comparison values for phthalates. This can be used to evaluate varying HIs for different microplastic component mixtures. By using DEP as a DEHP substitute, the HQ would become (0.0022 to 0.053 mg/kg/day)/6 mg/kg/day = 0.00036 to 0.0088. This example highlights the beneficial applications of using a substitute component to minimize toxicity. Other substitutes, such as DINCH, result in a HQ close to that of DEP. However, mammalian studies are limited for this substance [21,22].

Table 1: Acute, Intermediate, and Chronic Ingestion Minimal Risk Levels (MRLs) and Related Comparison Doses for Various Phthalates Used in Plastics Production.

Phthalate	Acute Ingestion MRL (mg/kg/day)	Intermediate Ingestion MRL (mg/kg/day)	Chronic Ingestion MRL (mg/kg/day)	References
DEHP [di(2-ethylhexyl) phthalate]	0.003	0.0001	n/a	ATSDR 2022 (12)
DBP (di-n-butyl phthalate)	0.5	n/a	n/a	ATSDR 2001 (13)
DEP (diethyl phthalate)	7	6	n/a	ATSDR 1995 (14)
DnOP (di-n-octyl phthalate)	3	0.4	n/a	ATSDR 1997 (17)
BBP (benzyl butyl phthalate)	n/a	n/a	0.2 RfD*	US EPA 2014 (18) US EPA 2026 (19)
DINCH (dilso nonyl cyclohexane-1,2-dicarboxylate)	n/a	(0.7-to-3 MRL equivalent) **	n/a	David 2015 (21) Bhat et al., 2014 (22)

n/a = not available
RfD = Reference Dose
*The 2014 RfD was based on a point of departure that is currently being considered.
**MRL equivalents are derived by assuming a modifying factor of 100 due to variability and animal to human extrapolation from the NOAEL (300 mg/kg/day) or use of a reference dose derived from a benchmark dose of 21 mg/kg/day.

Summary of toxic effects of DEHP

High levels of DEHP were seen in prematurely born children receiving medical care in hospitals over twenty years ago [44,45]. This was especially concerning given what was known about DEHP's toxicity then [45], and we know even more now [11,12].

DEHP acts as an endocrine disruptor. The developing fetus and the male reproductive system are sensitive targets of toxicity. DEHP exposure has been linked to decreased testosterone and sperm motility in men, though human fertility issues are not definitively established. Animal studies show high DEHP levels can damage testes, reduce fertility, and cause liver/kidney damage. Exposure during pregnancy may negatively impact child development, potentially leading to preterm birth, altered puberty, and male newborn malformations [12].

High or chronic exposure is associated with significant adverse health effects, particularly in animal studies, and it is classified by several federal agencies as a probable human carcinogen. Studies in rats and mice have shown that high, long-term DEHP doses also can lead to liver, pancreatic, and testicular cancers. While the link to human cancer is not certain, the DHHS considers it "reasonably anticipated to be a human carcinogen," and the US EPA calls it a "probable human carcinogen". Additionally, high DEHP levels may cause liver and kidney damage and could potentially increase allergies and asthma [12].

Cadmium exposure calculation

Another hazardous component frequently found in plastics and microplastics is cadmium (Cd). It is a component of yellow or red pigments used in plastic mixtures, and concentrations can be as high as 25,000 mg Cd/kg plastic [46]. This value is used to calculate the potential amount Cd in cabbage as a result of MPs as was done with DEHP.

Utilizing the above estimate for a 30-90 kg individual, where the MP dose from a bowl of cabbage was: 0.0022- 0.2 mg/kg/day.

The dose range for the higher Cd estimate becomes: 0.2 mg/kg/day x 25,000 ppm = 0.005 mg/kg/day
The intermediate MRL for Cd is 0.0005 mg/kg/day [47]. Thus, the upper estimate of the HQ =10.

As shown above, both DEHP and Cd could present a theoretical HQ>1 individually. Because the effects of combined exposures to many chemicals are considered additive in the absence of known interactions, these two values can be added to get a total hazard index of the mixture HQ >>2 [17,31,32,47-49]. Here we would add the Cd (HQ = 10) and the DEHP (HQ =530) resulting in a HI =540.

Summary of toxic effects of cadmium

Cadmium is a highly toxic metal that accumulates in the body, particularly the kidneys and liver, with a biological half-life exceeding 25 years. It is classified as a known human carcinogen.

The long-term effects of cadmium exposure include kidney damage, bone disease, and respiratory damage. The kidneys are the most critical, most sensitive, and primary target organ for chronic cadmium exposure (both oral and inhalation). It causes renal tubular dysfunction (proteinuria), which can progress to chronic kidney disease. Long-term Cd exposure leads to reduced bone mineral density, fragile bones (osteoporosis), and osteomalacia (softening of the bones), which increases the risk of fractures. Chronic inhalation causes chronic bronchitis, emphysema, and decreased lung function [47].

The scientists of Our1World have independently found associations between cadmium exposure and hearing loss, ALS, Autism, Parkinson's, and a shortening of telomeres, which protect our DNA from damage [50-54]. The genotoxic (DNA damaging) effects could permit other co-pollutant exposures to exert their own further DNA damage [55]. In the case of ALS, cadmium is associated with dozens of different mutations to the SOD1 gene [51]. While the mechanisms of action are not fully understood, it is clear that lower Cd exposures reduce the relative risks of association with these serious health outcomes. Furthermore, Cd is found at higher levels in pregnant woman and preferentially passes through to the developing fetus [56].
The contribution of Cd from red or yellow dyes in MNPs on the total body burden of Cd is not currently understood. However, the added value of color should not be worth the toxic potential. Especially since read and yellow microplastics have been found in the liver, the most sensitive endpoint for Cd [57].

Lead exposure calculation

Additionally, some plastics are known to contain lead chromate, lead sulphate, and/or lead molybdate at concentrations that can be double of that of cadmium to produce different red pigments [46]. Since there are no safe levels of lead (Pb), in any of its forms, there is a potential of a HQ>1 for the ingestion of any amount of lead. Because Pb has common health endpoints with both Cd and DEHP, the total HI increases to levels that are comparable to the uncertainty factors associated with the derivation of the MRLs [12].

Summary of toxic effects of lead

Ingesting lead causes widespread, often irreversible damage, primarily to the nervous system, kidneys, and cardiovascular system [58]. It is highly genotoxic to people and animals [55]. The neurological system is the most sensitive target. In children, low levels cause reduced IQ, learning disabilities, short attention span,

and behavioral problems. High levels can cause encephalopathy, convulsions, coma, and death. There are already high baseline exposures to children in some communities as it is still found in some older house paints [59].

Lead exposure in adults can result in anemia, hypertension, and reproductive harm in adults. It increases blood pressure, particularly in middle-aged and older adults. Because lead interferes with heme synthesis, exposure can reduce hemoglobin production. Long term exposures can cause chronic nephropathy and kidney damage. It is found to reduce fertility in men and women and has been associated with miscarriages and premature births. Lead acts by mimicking or replacing calcium, disrupting vital cellular processes throughout the body [58].

Limitations of Current MNP Data in Agricultural Products and in the Proposed Health Assessment Method

For demonstration purposes, we selected a single MP measurement for cabbage from a study that identified higher levels than found elsewhere, despite not including the much smaller nanoplastics. Measurements recently made on carrots, apples, and spinach are lower [35-38]. However, the science of MNP identification in various media has been a significant data gap (particularly the lack of mass measurements), but recent data collection in some media, environmental and biologic, has led to identification of previously unaccounted MNPs [41,60-63]. Thus, even higher levels are possible as more data is gathered.

The above example has focused on only three of several possible pollutants associated with MNPs. However, the assessment of combined effects of co-pollutants in a mixture is not simple. Many interaction studies are conducted with a small number of co-pollutants of interest. In several of these cases, the dose-response effect was a little less than additive as is the case of phthalates mixed with dioxin, but not so with other mixtures [64]. Combined effects of certain mixtures have been documented to be much greater than the sum of the components, such as with cigarette smoke and radon, silica dust, or asbestos [65,66]. In addition to the chemical toxicity, the particulate nature of MNPs presents its own toxicity [30]. Data gaps in the particulate-toxicity preclude its current including in this mixtures assessment. Overall, microplastic mixtures toxicity is an emerging area that needs to be further studied to fully understand its impact on the environment and human health.

Conclusion

While plastic products offer many advantages, including health benefits, micro- and nanoplastics do not. Here we identified two related assessment methods that can be applied to old, new, and hypothesized plastics to help identify the least harmful mixtures. It can be also used directly on assessment data. This HI mixture method can be utilized for all components of current plastics found in the environment and thus contribute to community impact assessments. Given specific measurements of MNPs in the environment, a dose and total risk can be calculated for the known toxicities. The same process can be used to assess new plastic blends prior to production to determine the lowest combined hazard index achievable with minimum impact to the performance. As shown above, replacing DEHP with most alternative plasticizers results in a lower theoretical risk. In some cases, it is possible to eliminate high, medium, and even low toxicity phthalates [67].

Using a simple comparison of LOAELS, we showed that DEHP was possibly >2000 times more toxic than other phthalates. Using a hazard index approach (HI) with MRLs, that a bowl of cabbage could contain enough microplastics to create a HI>>1 for a mixture of DEHP, cadmium, and lead. The HI method can also be applied to other produce and foods found to contain micro and nanoplastics should that data offer a means to calculate mass of plastics. The limitation of this approach is that the percentages of all the potential chemical components of the plastic are not readily available as is their toxicity data.

There are substitutes for DEHP that are less toxic and the bright colors added to the plastics also add to the overall toxicity of plastics and microplastics. Additionally, if buyers were aware that a less colorful or white plastic product had a lower overall hazard index due to reduced heavy metals such Cd and Pb, they might opt for the color change. Outside of the private sector, this method can also help governments with high volume plastic purchases opt for less toxic specifications.

While the colored pigments might not be an issue for plastics designed for agricultural uses, litter and deposition of plastics from the hydrologic cycle onto crops do include pigmented plastics.

Recommendation

Removing the most harmful additives in all plastics is prudent. The prioritization process explained here can be an approach towards reducing harmful exposures associated with microplastics. By using this process, we explain removing or substituting three plastic components viz., DEHP, cadmium, and lead can reduce the overall toxicity of plastics and their breakdown products.

This method can be further utilized to determine the risk posed by microplastics in other food products. However, ongoing research on the alternatives to DEHP and other harmful plastic components is a pivotal next step to better quantifying the toxicity of micro- and nanoplastics.

The US EPA is currently undertaking the assessment of phthalates with a primary goal of protecting workers and the environment. Phthalate-containing MNPs are already in the environment. Thus, lifetime exposures to phthalates along with plastic additives are already occurring. Hence, inclusion of lifetime exposures should be an essential part of such an assessment.

Further research on the phthalate substitutes is necessary to ensure that promising substitutes, such as DINCH, have adequate data to estimate safe values.

A cradle to grave approach for plastic products, which includes limiting single use plastics, recycling, and reusing, is also essential [68]. The collection and removal of larger plastics already in our environment before they are broken down into MNPs is the key but nearly impossible.

Our1World is involved with each of these steps, including clean ups and studying constructed wetlands for the capture and reduction of pollutants like plastics and microplastics. We also support the development of policy that re-examines the full lifecycle of products and their impact before people and the environment are affected, such as the "true disclosure" bill before the US Congress [69].

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