Environmental Science & Technology Letters

pubs.acs.org/journal/estlcu

Letter

Occurrence of Polyethylene Terephthalate and Polycarbonate Microplastics in Infant and Adult Feces

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Cite This: *Environ. Sci. Technol. Lett.* 2021, 8, 989−994

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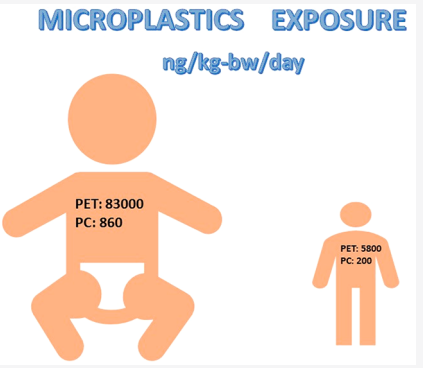
ABSTRACT: Although human exposure to microplastics (MPs) and the health effects thereof are a global concern, little is known about the magnitude of exposure. In this study, we quantitatively determined the concentrations of polyethylene terephthalate (PET) and polycarbonate (PC) MPs in three meconium and six infant and 10 adult feces samples collected from New York State. PET and PC MPs were found in some meconium samples (at concentration ranges from below the limit of quantification [<LOQ] to 12,000 and <LOQ−110 ng/g dry weight, respectively) and all infant stool specimens (PET: 5700−82,000 ng/g, median, 36,000 ng/g; PC: 49−2100 ng/g, median, 78 ng/g). They were also found in most (PET) or all (PC) adult stool samples but at concentrations an order of magnitude lower than in infants for PET MPs (<LOQ−16,000 ng/g, median, 2600 ng/g). The estimated mean daily exposures from the diet of infants to PET and PC MPs were 83,000 and 860 ng/kg body weight per day, respectively, which were significantly higher than those of adults (PET: 5800 ng/kg-bw/day; PC: 200 ng/kg-bw/d). Our study suggests that infants are exposed to higher levels of MPs than adults.



INTRODUCTION

Pollution by microplastics (MPs), plastic particles <5 mm in size, is a global concern due to their potential risk to humans and environmental health.[1,2] Although studies of MPs over the past decade have focused primarily on their effects on the oceanic environment, there has also been growing concern regarding their human health effects.[3,4] A few studies have reported human exposure doses to MPs through ingestion of indoor dust[5] and food items such as salt,[6] beverages,[7] and drinking water.[6] An average weekly ingestion dose of MPs in the range of 0.10−5 g has been estimated.[8] Thus, although it is known that humans are exposed to MPs, the magnitude and variability of exposure doses and intake rates globally remain unknown.

Plastics were long thought to be inert, and following ingestion, they pass through the gastrointestinal (GI) tract and excrete through the biliary pathway.[9] However, recent studies suggest that MPs < 10 μm in size can cross cell membranes and reach the circulatory system.[10] Studies have reported the presence of MPs in human and pet animal stool specimens, providing evidence that these particles can pass through the GI tract.[11,12] In one study, stools collected from eight human volunteers (three men and five women, 33−65 years old) from several countries were reported to contain 20 pieces of MPs of sizes 50−500 μm per 10 g of stool, with polypropylene (PP: 62.8%) and polyethylene terephthalate (PET: 17%) MPs predominating; the authors estimated an average excretion rate of 25 MP particles per 100 g of stool.[11] Another study reported an excretion rate of 0.03−677 mg/week based on the analysis of cat and dog stools collected from New York State, U.S.A.[12]

Little is known about the possible toxic effects of MPs in humans, but a few exposure studies *in vitro* and laboratory animals have reported adverse health effects.[13−17] Exposure to polystyrene (PS) nanoplastics caused reduction of human lung cell viability, cell cycle arrest, activation of inflammatory genes, and promotion of cell apoptosis.[17] Exposure to PS MPs in mice during gestation caused metabolic disorders in offspring,[14] indicative of transgenerational effects. The occurrence of MPs in human placenta further highlighted the potential transgenerational effects of these chemicals in humans,[18] given that they could affect the developing fetus.

While it is known that humans are exposed to MPs, the magnitude of exposure is poorly understood. Available data suggest a wide range of doses (e.g., tens to billions of MPs daily or 0.1−5 g weekly) calculated through various empirical and probabilistic exposure models.[8,11,12,16] These uncertainties about exposure doses have resulted in controversies regarding the potential risks that MPs may pose to human health. A few studies have reported human intake of MPs through select

Received: July 15, 2021
Published: September 22, 2021

sources and pathways,[2,19] but research describing human body burdens of MPs remains lacking. A major limitation in exposure studies is the lack of sensitive analytical methods to determine MPs in human/biological matrices. Although biomonitoring studies could provide an integrated assessment of exposure, such studies are still in their infancy. Because plastic particles >150 μm in size are reported to be excreted in feces, however, determination of MPs in stools would provide some information regarding the magnitude of exposure. Furthermore, MPs have been reported to occur in human placenta,[18] but no reports of the occurrence of MPs in meconium (feces of newborn) and infant stools have been available to date. Therefore, in this study, we determined PET and polycarbonate (PC) MPs, which are mainly used in the production of textile fibers, water bottles, and mobile phone cases,[20,21] in a convenience sample of meconium and infant and adult feces collected from New York State, U.S.A., using a depolymerization method followed by liquid chromatography−tandem mass spectrometry (LC-MS/MS) analysis. Human exposure doses to PET and PC MPs were assessed from the concentrations measured in feces.

MATERIALS AND METHODS

Chemicals. PET (3−5 mm) and PC (3 mm) granules were purchased from Goodfellow Cambridge, Ltd. (Huntingdon, England). 1-Pentanol was purchased from Fisher Scientific (Pittsburgh, PA, U.S.A.). Terephthalic acid (TPA: >99% purity) was purchased from Toronto Research Chemicals, Inc. (North York, ON, Canada). Deuterated terephthalic acid (D_4-TPA: 99%) and bisphenol A (BPA: >99%) were purchased from Sigma-Aldrich (St. Louis, MO, U.S.A.). $^{13}C_{12}$-BPA (99%) was purchased from Cambridge Isotope Laboratories (Andover, MA, U.S.A.). High-performance liquid chromatography (HPLC)-grade methanol and water were supplied by J.T. Baker (Phillipsburg, NJ, U.S.A.). Solid-phase extraction (SPE) cartridges (HLB, 200 mg/6 cc) were purchased from Waters (Milford, MA, U.S.A.).

Sample Collection. Feces from six one-year-old infants and meconium specimens from three newborns from New York City were collected as part of the New York University (NYU) Children's Health and Environment Study (CHES) in 2019. CHES is an ongoing, clinically enrolled, longitudinal cohort study of pregnant women and their children that is part of the Environmental Influences on Child Health Outcomes (ECHO) program.[22] Samples from infants and newborns were from diapers. For meconium specimens, the newborn's first diaper with stool was collected, and a spatula was used to transfer meconium into cryovials for storage at −80 °C. Similarly, stool from soiled diapers of one-year-old infants was transferred into cryovials and stored at −80 °C. Efforts were made to exclude meconium and stools that came in direct contact with diapers during the transfer of samples from diapers. Adult feces samples (n = 10) were collected from 10 volunteer donors 30−55 years old from Albany, New York, U.S.A., during August 2019. Adult volunteers were instructed to deposit their stool on a clean porcelain surface, and the top portion was scooped into a 50 mL polypropylene (PP) container. Samples were transferred to the laboratory within 5 h and stored at −20 °C until analysis.

Sample Extraction. Stool samples were lyophilized, ground using a stainless steel pestle and mortar, and sieved through a 2 mm mesh-sized stainless steel sieve. An aliquot of each sample was depolymerized and extracted through an SPE method similar to one described previously.[23] Details of the extraction method are presented in the Supporting Information (Text S1). The method for the extraction of freely available forms of TPA and BPA in feces, prior to depolymerization, is described in Text S1.

Instrumental Analysis. Details of the instrumental analysis are presented in Tables S1 and S2. The concentrations of PET- and PC-based MPs were calculated as [$C_{\text{depolymerization}}$ (concentration of TPA or BPA after depolymerization) − C_{free} (concentration of TPA or BPA before depolymerization)] of TPA and BPA, divided by 0.90 and 0.86, respectively.[23]

Quality Assurance/Quality Control. Potential contamination of PET and PC arising from diaper samples was a concern, and efforts were made to remove the outer surface of feces that had come in contact with diapers. It is worth noting that most diapers are made of PP on the side that comes in contact with infant's skin,[24] and therefore, PET and PC contamination was expected to be insignificant. To ensure the accuracy of the analytical method, we tested the depolymerization efficiency of alkaline digestion by fortifying PET and PC granules in feces samples. The recoveries of PET and PC fortified into feces samples were >95%. The round-bottomed flasks were muffled at 450 °C for 12 h prior to use. PET was detected in procedural blanks at an average concentration of 1600 (±87) ng/g; no PC was found in any of the procedural blanks. The elevated background concentration of PET, in reagent blank, was from reagents and chemicals such as KOH used in extraction. Background subtraction was therefore performed for PET during the quantification of the concentrations in feces samples. Quantification of TPA and BPA was performed by an isotope dilution method with D_4-TPA and $^{13}C_{12}$-BPA, respectively, as internal standards. TPA and BPA were spiked along with D_4-TPA (100 ng) and $^{13}C_{12}$-BPA (50 ng) into select feces samples and passed through the entire analytical procedure (n = 3). Recoveries of TPA and BPA in spiked feces samples were 80%−87% and 71%−75%, respectively (Table S3). A midpoint calibration standard and methanol were injected after every 10 samples as a check for drift in instrumental sensitivity and carryover of target compounds between samples, respectively. For samples with concentrations above the calibration range, dilutions were performed with water/MeOH (8:2 v/v) prior to reanalysis. PET and PC MPs concentrations in feces are reported on a dry weight (dw) basis unless specified otherwise. Samples were anonymized prior to analysis, and the analysis was approved by the Institutional Review Board of the New York State Department of Health.

Data Analysis. Instrumental data were acquired using the Analyst 1.6.2 software package (Sciex Inc., Framingham, MA, U.S.A.). Statistical analyses were performed with GraphPad Prism 8 (GraphPad Software, San Diego, CA, U.S.A.) and SPSS 22.0 (IBM, Armonk, NY, U.S.A.). Concentrations below the limit of quantification (LOQ, Table S3) were assigned a value equal to the LOQ divided by the square root of 2 in the calculation of mean. Differences between groups were compared using a Mann−Whitney test. A value of $p \leq 0.05$ was considered statistically significant.

MPs present in feces represent the unabsorbed fraction of ingested dietary sources. Therefore, the measured concentrations of MPs in feces were thought to be from diet. Due to the small sample size, meconium (also derived maternally from amniotic fluid) was not included in intake calculations. To estimate daily intake (EDI) of MPs, feces concentrations were

https://doi.org/10.1021/acs.estlett.1c00559
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extrapolated as dietary exposure doses. The following equation was used for the calculation of EDI

$$\text{EDI} = \frac{\text{feces concentration}\left(\frac{\text{ng}}{\text{g}}\right) \times \text{feces excretion rate}\left(\frac{\text{g}}{\text{day}}\right)}{\text{average body weight (Kg)}}$$

where EDI is the estimated daily intake (from diet); mean values of feces concentrations were used. The average weights of infants and adults were estimated at 7 and 64 kg, respectively.[25] The mean dry weights of feces excreted by one-year-old infants and adults daily were reported to be 20[26,27] and 87 g,[27,28] respectively.

RESULTS AND DISCUSSION

Microplastics in Feces. We detected PET and PC MPs in the feces of six infants, at concentrations in the ranges of 5700−82,000 ng/g (median: 36,000 ng/g dw) and 49−2100 ng/g (median: 78 ng/g dw), respectively. Among the three meconium samples analyzed, we detected PET MPs (12,000 and 3200 ng/g dw) in two samples and PC MPs (110 ng/g dw) in one sample (Table S4). Among the 10 adult stool samples analyzed, eight contained PET MPs at concentrations in the range of 2200−16,000 ng/g (median: 2600 ng/g dw), and all samples contained PC MPs in the range 37−620 ng/g (median: 110 ng/g dw). We previously reported the concentrations of these two types of MPs in feces of pet cats and dogs from New York State. For PET MPs, the reported concentrations in cat and dog feces were <LOQ−340,000 ng/g (median: 61,000 ng/g dw) and 7700−190,000 ng/g (median: 30,000 ng/g dw), respectively,[12] similar to those that we found in infant feces. However, the concentrations of PET MPs we measured here in adult feces were an order of magnitude lower than those found in pet or infant feces. For PC MPs, the reported concentrations in cat and dog feces were <LOQ−13,000 ng/g (median: 230 ng/g dw) and <LOQ−26,000 ng/g (median: 160 ng/g dw), respectively.[12] The concentrations of PC MPs in feces of dogs and cats were similar to those found in both infants and adults. There is one past report of PET and PP MPs in adult feces.[11] That study, however, used flotation spectroscopy and presented results as number and size of MPs rather than mass, precluding direct comparison with our results.

We found significant differences in the patterns of two types of MPs between infant and adult feces. PET concentrations were significantly higher in infant feces than in adult feces (Mann−Whitney, $p = 0.01$), whereas concentrations of PC MPs were not significantly different between the two age groups (Mann−Whitney, $p = 0.664$) (Figure 1). The MPs measured in infant and adult feces were thought to be primarily derived from dietary sources. High concentrations of MPs in the feces of one-year-old infants can be attributed to extensive use of plastic products/articles such as baby feeding bottles, sippy cups, utensils (spoons, bowls), plastic teethers, and toys, among others, during that growth stage. One-year-old infants are known to frequently mouth plastic products and clothing. In addition, studies have shown that infant formula prepared in PP bottles can release millions of MPs,[29] and many processed baby foods are packaged in plastic containers that constitute another source of exposure in one-year-old infants. Furthermore, textiles are a source of PET MPs. Infants often chew and suck cloths, and therefore, exposure of this age group to MPs present in textiles is a greater concern. Carpets made of PET and PP can be another source of MP exposure, as infants

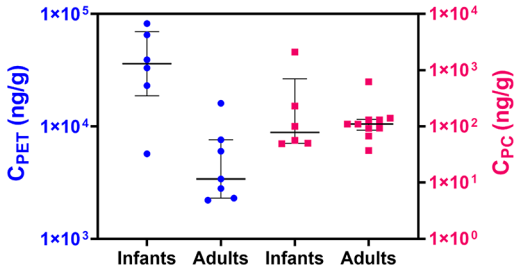

Figure 1. Concentrations (dry weight) of polyethylene terephthalate (PET) and polycarbonate (PC) microplastics in infant ($n = 6$) and adult feces ($n = 10$). Dots represent individual samples. Upper and lower lines represent interquartile ranges. Middle lines represents median values.

crawl on the carpeted surfaces frequently. The greater exposure of MPs in infants than in adults is further supported by the concentrations of plastic additives, namely, phthalates, especially di(2-ethylhexyl) phthalate (DEHP), which are significantly higher in infant urine than in that of adults.[30]

TPA and BPA in Feces. We detected free TPA in all stool samples. The concentrations of TPA in infant feces were in the range of 390−1600 ng/g (median: 1200 ng/g dw), whereas those in meconium were 110−1600 ng/g (median: 500 ng/g dw) and those in adult feces were 180−3000 ng/g (median: 410 ng/g dw). Free BPA was less prevalent than TPA, being detected in only one (out of six) infant's stool samples (16 ng/g), two out of three meconium samples (73 and 136 ng/g), and none of the 10 adult stools (Table S4). Being low-molecular-weight (MW) compounds, TPA (MW: 166) and BPA (MW: 228) are expected to be excreted primarily via urine. The reported concentrations of TPA and BPA in adult urine from the U.S.A. are in the ranges of 8.8−1250[31] and <LOQ−20.9 ng/mL,[32] respectively. Nevertheless, the concentrations we measured in feces were similar to those in urine. It is possible that PET and PC MPs are degraded by gut microbiota to yield TPA and BPA, respectively.

The daily fecal excretion rate for adults is reported to be 87 g[27,28] on a dry weight basis, whereas urine excretion volume is 1700 mL.[33] We found no correlation between the concentrations of PET and TPA either in infant feces ($r = 0.611$, $p = 0.108$) or in adult feces ($r = 0.571$, $p = 0.180$). This could be because PET MPs are excreted predominantly in feces, whereas TPA is excreted in urine. Other sources of TPA and BPA may also contribute to the observed lack of correlation between MPs and these monomers.

Exposure Assessment. The estimated average daily exposure doses via dietary sources to PET and PC MPs in one-year-old infants were 83,000 and 860 ng/kg-bw/d, respectively, and those in adults were 5800 and 200 ng/kg-bw/d, respectively (Table 1). The few available studies have reported a wide range of MP exposure doses in humans.[8,11,12,16,34] One study reported MP exposure in the range of tens to billions of particles daily depending on the sources and pathways.[16] Another found exposure doses of 0.1−5 g weekly through multiple pathways.[8] PET exposure at a rate of 0.16 particles/kg-bw/d and PC exposure at a rate of 0.0063 particles/kg-bw/d have also been reported.[11] A probabilistic lifetime exposure model predicted an intake rate of 184 ng/capita/d for children and 583 ng/capita/d for adults, from nine different sources.[34] Our values are 2−3 orders of magnitude higher than those predicted through the probabilistic model, a

https://doi.org/10.1021/acs.estlett.1c00559
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Table 1. Comparison of Daily Intakes of Polyethylene Terephthalate (PET) and Polycarbonate (PC) Microplastics Calculated from Mean Concentrations in Feces and Indoor Dust

	Infants		Adults	
	PET (ng/kg-bw/d)	PC (ng/kg-bw/d)	PET (ng/kg-bw/d)	PC (ng/kg-bw/d)
Feces (dietary sources)	83,000	860	5800	200
Indoor dust[a]	120,000	750	6600	41

[a]Data are from Zhang et al.[37] Concentrations in feces are thought to be primarily from diet.

difference that can be attributed to the limited exposure sources and pathways included in that model. The dietary intake of MPs in the U.K. has been reported to be 40 mg/capita/d.[35] Thus, the available data suggest a wide range of exposure doses to MPs depending on the approach used in estimating exposures.[36] Our estimate falls within the values reported in earlier empirical analyses that were based on the concentrations of MPs reported in various exposure sources. Since most of the plastic products for infants are PP based, the concentrations of PP MPs in feces may be larger than those of PET and PC. We believe that the estimated ingestion doses of MPs, based on the concentrations measured in feces, were derived primarily from dietary sources. It is likely that human exposure to MPs vary widely between individuals.

A recent study reported human exposure to PET and PC MPs through indoor dust ingestion, finding respective exposure doses of PET and PC of 6600 and 41 ng/kg-bw/d in U.S.A. adults and of 120,000 and 750 ng/kg-bw/d in U.S.A. infants.[37] These results suggest that PET intake through dust ingestion is higher than that from diet. On the other hand, diet has also been identified as a major source of PC intake in adults[6] (Table 1). Another recent study reported that inhalation is the major pathway of human exposure to MPs.[2] The sources and pathways of human exposure to MPs remain a subject of considerable debate.[36]

Our study is the first, to our knowledge, to quantitatively determine PET and PC MPs in the stools of adults and infants from the U.S.A. We found that concentrations of PET in infant feces were significantly higher than those in adult feces. It should be noted that the sample size is limited, making it impossible to further extrapolate factors that could affect the exposures. Although the surface of baby diapers that come in contact with the skin is made of PP[24] which is not measured in this study, contamination with MPs from diapers in diaper-collected infant stools cannot be ruled out; in general, caution should be exercised to eliminate sources of contamination during feces collection. Although clutch collection is the preferred method of sampling for the analysis of MPs in feces, plastic-free diapers may also be used. In the absence of such products, background levels of MPs present in diapers should be examined prior to use in sampling. Data in the literature regarding fecal excretion rates in children and adults vary widely, and the values selected in this study for exposure dose calculation can affect the results. Nevertheless, our data provide baseline evidence for MP exposure doses in infants and adults and support the need for further studies with a larger sample size to corroborate and extend our findings.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/acs.estlett.1c00559.

Sample extraction method; HPLC mobile phase gradient; MS parameters for the analysis of TPA and BPA; method quantification limits and recoveries of TPA and BPA; and concentrations of PET MPs, PC MPs, TPA, and BPA in individual samples (PDF)

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Notes

The authors declare no competing financial interest.

■ ACKNOWLEDGMENTS

We thank the volunteers for providing feces samples. Analysis was performed at the Wadsworth Center, New York State Department of Health, Albany, New York, United States. The study was funded, in part, by the National Institute of Environmental Health Sciences of the U.S. National Institutes of Health under Award No. U2CES026542 (to K.K., analytical method development) and NIH Office of the Director (UG3/UH3OD023305) (to L.T., infant feces samples). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. This work was supported in part by the National Natural Science Foundation of China (41722304)

and the 111 Program of the Ministry of Education, China (T2017002), and by the Chinese Scholarship Council (CSC 201806200120).

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