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Comparative Assessment of Human Exposure to Phthalate Esters from House Dust in China and the United States

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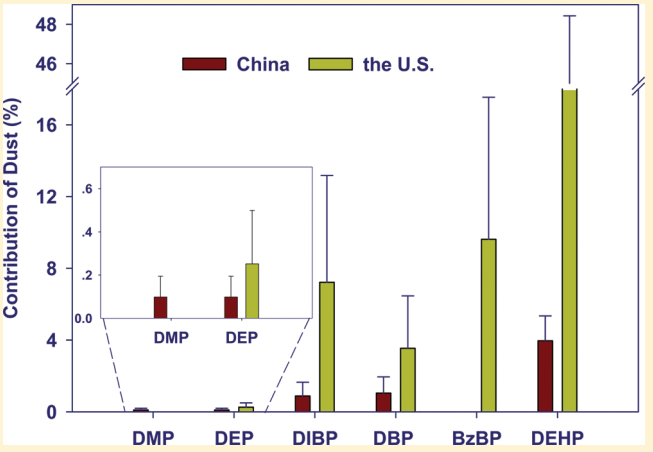
Supporting Information

ABSTRACT: Because of volatilization and leaching from their application in consumer and personal care products, phthalate esters are ubiquitous contaminants in the indoor environment. In this study, we measured concentrations and profiles of 9 phthalate esters in indoor dust samples collected from six cities in China (n = 75). For comparison, we also analyzed samples collected from Albany, New York, USA (n = 33). The results indicated that concentrations, except for dicyclohexyl phthalate (DCHP) and bis(2-ethylhexyl) phthalate (DEHP), and profiles of phthalate esters varied significantly between the two countries. Concentrations of diethyl phthalate (DEP), di-n-hexyl phthalate (DNHP), and benzyl butyl phthalate (BzBP) were 5 to 10 times higher in dust samples collected from Albany than those from the Chinese cities. In contrast, concentrations of di-iso-butyl phthalate (DIBP) in dust samples from Albany were 5 times lower than those from the Chinese cities. We estimated the daily intake (DI) of phthalate esters through the routes of dust ingestion and dermal dust absorption. The extent of contribution of indoor dust to human exposures varied, depending on the type of phthalate esters. The contribution of dust to DEHP exposure was 2−5% and 10−58% of the estimated total DIs in China and the USA, respectively. On the basis of the estimates of total DIs of phthalates, extrapolated from urinary metabolite concentrations, the contributions of inhalation, dermal absorption, and dietary intake to total DIs were estimated. The results indicated that dietary intake is the main source of exposure to DEHP (especially in China), whereas dermal exposure was a major source for DEP. This is the first study to elucidate sources of human exposure to phthalates among the general population in China.



INTRODUCTION

Phthalates are used as plasticizers in numerous consumer products and building materials. Several million tons of phthalates are produced worldwide every year for the production of soft polyvinyl chloride (PVC) and other plastics. As a consequence, phthalates are present in the indoor environment and in food. Studies have shown that humans are exposed to phthalates on a daily basis.[1−4] Exposure of humans to phthalates occurs via inhalation, dermal absorption, and dietary intake. A scenario-based exposure model used for the assessment of sources of phthalate exposure among Europeans showed that dermal application of consumer products dominated the sources of exposure to dimethyl phthalate (DMP), diethyl phthalate (DEP), and benzyl butyl phthalate (BzBP), whereas dietary intake was the major source of exposure to di-n-butyl phthalate (DBP), di-iso-butyl phthalate (DIBP), and bis(2-ethylhexyl) phthalate (DEHP).[5] In a study of the general population in Japan, dietary intake and inhalation accounted for less than 50% of the total daily exposure to DMP, DEP, and DBP, whereas dietary intake was the dominant source of exposure to BzBP and DEHP.[6] Application of exposure models for the evaluation of sources of phthalate exposures yielded variable results.[7] In general, earlier studies [5−7] indicated that the sources of human exposure to phthalates vary, depending on the geographic area and the type of phthalate ester.

Studies have shown that phthalates elicit reproductive and developmental toxicities in laboratory animals.[8−11] For example, exposure of rats to DBP reduced fertility and altered ovarian function during pregnancy in females and/or sperm counts in

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Table 1. Phthalate Ester Concentrations and Frequency of Occurrence in Indoor Dust (μg/g, dry weight) Collected from Albany (USA) and Several Cities in China

			DMP	DEP	DIBP	DBP	DNHP	BzBP	DCHP	DEHP	DNOP	total
China	Beijing (n = 11)	median	0.7	0.4	12.6	18.9	nd	0.6	nd	156	nd	255
		range	nd −1.6	0.1−0.6	7.2−83.2	7.0−31.5	nd	0.1−1.1	nd	47.6−883	nd −0.5	63−930
	Guangzhou (n = 11)	median	0.3	0.2	11.1	11.6	nd	0.2	nd	146	0.4	173
		range	0.2−0.9	0.2−0.8	4.5−63.9	9.2−58.7	nd −0.1	0.1−12.0	nd	56.6−949	nd −2.7	75.6−1080
	Jinan (n = 13)	median	0.06	0.1	10.4	9.3	nd	0.1	nd	98.2	nd	151
		range	nd −0.7	nd −0.3	2.6−19.7	2.3−128	nd −0.7	nd −0.1	nd	9.9−252	nd −1.3	24−303.
	Qiqihaer (n = 12)	median	0.1	1.5	26.0	21.9	nd	0.2	nd	348	0.3	428
		range	0.1−0.3	0.8−6.1	13.2−299	10.9−147	nd −0.1	0.2−0.6	nd −0.3	149−939	nd −1.1	180−1040
	Shanghai (n = 21)	median	0.2	0.4	33.6	26.9	nd	0.2	nd	319	0.3	401
		range	0.1−0.8	nd −45.5	7.0−85.9	1.5−96.2	nd-0.2	nd −7.4	nd −0.1	117−1380	nd −0.6	204−1540
	Urumchi (n = 7)	median	0.5	0.8	32.8	170	nd	0.4	nd	563	0.6	765
		range	0.3−8.2	0.3−1.0	6.5−87.9	77.9−1160	nd −3.7	0.2−1.2	nd	204−8400	0.2−45.7	450−8590
	Total (n = 75)	median	0.2	0.4	17.2	20.1	nd	0.2	nd	228	0.2	295
		range	nd −8.2	nd −45.5	2.6−299	1.5−1160	nd-3.7	nd −12.0	nd −0.3	9.9−8400	nd −45.7	24.4−8590
		frequency	99%	100%	100%	100%	23%	99%	15%	100%	64%	
USA	Albany (n = 33)	median	0.08	2.0	3.8	13.1	0.6	21.1	nd	304	0.4	396
		range	nd −3.3	0.7−11.8	0.7−34.4	4.5−94.5	0.1−11.4	3.6−393	nd −0.3	37.2−9650	nd −14.1	87.1−9670
		frequency	94%	100%	100%	100%	100%	100%	18%	100%	82%	

males. Studies [12,13] have shown a negative relationship between environmental phthalate exposure and intelligence in children or childhood behavior. Concerns have been raised over the potential effect of chronic phthalate exposure on human health.[14]

Phthalate esters are not chemically bound to products [15] and can be easily released into the indoor environment. House dust is a reservoir of many semi- and nonvolatile substances, including phthalate esters. Indoor dust has been reported to contain high concentrations (on the order of tens to hundreds of μg/g) of several phthalate esters.[16−20] In China, phthalate esters account for 90% of the plasticizer usage in PVC production (i.e., over one million tons per year).[21] Despite the high production and consumption of plastic products in China, only two studies have reported the concentrations of phthalates in Chinese populations,[22,23] and few studies have reported the occurrence of phthalates in Chinese soil.[24−26] Nevertheless, occurrence of phthalate esters in indoor dust and the magnitude of human exposure in China are not known.

In our previous study,[27] we reported that urinary concentrations and compositions of phthalate metabolites varied among three cities in China (Guangzhou, Qiqihaer, and Shanghai), suggesting that phthalate exposure within the Chinese population varies by location. In this study, we investigated the occurrence of 9 phthalates [DMP, DEP, DBP, DIBP, BzBP, DEHP, di-*n*-hexyl phthalate (DNHP), dicyclohexyl phthalate (DCHP), and di-*n*-octyl phthalate (DNOP)] in 75 indoor dust samples collected from six cities in China. For the purpose of comparing phthalate concentrations and profiles, we analyzed 33 indoor dust samples collected from Albany, New York, USA. Further, on the basis of a one-compartment pharmacokinetic model, the urinary phthalate metabolite concentrations reported for China and the USA [28] were used for the estimation of total daily intakes (DIs). From the estimated values of total DIs, the contributions of dust and other pathways (inhalation, dermal, and dietary) to phthalate exposures were calculated.

The objectives of this study were (a) to determine the concentrations and profiles of phthalate esters in indoor dust collected from several locations in China and in Albany, New York, USA; (b) to examine the relationship between urinary phthalate metabolite concentrations and phthalate ester concentrations in indoor dust from China and the USA; (c) to estimate the DI rates of phthalates through dust; and (d) to evaluate the contribution of various exposure routes to the total DIs of phthalates in China and the USA.

METHODS AND MATERIALS

Standards. Phthalate ester standards, DMP, DEP, DBP, DIBP, BzBP, DEHP, DNHP, DCHP, and DNOP, and their corresponding d_4 (deuterated) internal standards (except for BzBP) were purchased from AccuStandard Inc. (New Haven, CT), with a purity of >99%.

Sample Collection and Preparation. Dust samples were collected from six cities in China, Beijing (n = 11), Shanghai (n = 21), Guangzhou (n = 11), Urumchi (n = 7), Jinan (n = 13), and Qiqihaer (n = 12), from May to July 2010 (Figure S1) ("S" designates figures and tables in the Supporting Information). Dust samples from China were collected by sweeping the floor and wiping the top of furniture. Dust samples were also collected from Albany, New York, USA, between December 2007 and January 2008, as well as during May 2010 from vacuum cleaner bags of several homes. Samples from the USA contained more particles from carpet flooring than those from China. All samples were sieved through a 2 mm sieve, packed in clean aluminum foil, and stored at −4 °C until analysis.

Dust samples were extracted (0.05 to 0.1 g) in a 12 mL glass centrifuge tube. Samples were spiked with 250 ng of internal standards and allowed to equilibrate for 3 h at room temperature. Samples were extracted three times by shaking in an orbital shaker for 30 min each time, with 4 mL of hexane:acetone (4:1, v:v) followed by centrifugation at 4000 rpm for 5 min. The combined extracts were concentrated under a gentle stream of nitrogen to 1 mL for instrumental analysis.

Instrumental Analysis. Determination of concentrations of phthalate esters was carried out with gas chromatography (Agilent Technologies 6890 N) coupled with mass spectrometry (Agilent Technologies 5973) in the selective ion-monitoring (SIM) mode. The details of the method are given in the Supporting Information. The limits of quantification (LOQ) were calculated from the lowest concentration of the calibration curve and a nominal sample weight of 1.0 g. The LOQ of DNOP and 8 other phthalate esters analyzed in dust samples was 10 ng/g and 2.0 ng/g, respectively.

Quality Assurance and Quality Control. The extraction efficiency of the analytical method was tested by performing a fourth extraction of the samples, followed by the first three extractions, as described above, for selected (n = 12) samples. The results indicated that only trace levels of DBP (2.0 ng/g), DIBP (<2.0 ng/g), and DEHP (3.6 ng/g) were found in the fourth extraction, which were <1% of the concentrations measured in the first three extractions combined. Only glass centrifuge tubes and glass pipets were used in extraction and cleanup steps and the glassware were cleaned by ultrasonication in acetone for 30 min prior to use. For each batch of 10 samples, a method blank (0.1 mL hexane), a spiked blank, and a pair of matrix-spiked samples (1 μg of individual phthalate esters)/duplicates were processed. Low concentrations of DBP, DIBP, and DEHP were detected in method blanks, with average respective concentrations of 4.2, 5.3, and 12.0 ng/g; these concentrations were considerably lower (<0.1%) than those found in samples and, therefore, were not subtracted from sample values. The average recoveries of target compounds in spiked matrices and blanks were 71−106% (except for DEHP, which was 166%) and 73%-110%, respectively. The average recoveries of internal standards in spiked matrices, blanks, method blanks and in dust samples were 70−132%, 59−108%, 67−106%, and 76−134%, respectively. Concentrations below the LOQ were assigned a value of zero for statistical analysis. Data analysis was conducted using SPSS Statistics, V. 17.0. Comparisons among different sample types were conducted using nonparametric tests. Statistical significance was set at $p < 0.05$.

RESULTS AND DISCUSSION

Phthalates in Indoor Dust. The median and range of concentrations of phthalate esters found in dust samples are shown in Table 1. DMP, DEP, DIBP, DBP, BzBP, and DEHP were found in >94% of the dust samples analyzed; DNOP was found in ~60% of the samples analyzed and DCHP was seldom detected (<18%). DNHP was found in all of the dust samples from Albany (USA), but it was found in only 23% of the samples from the Chinese cities (Table 1). In dust samples collected from China, DEHP, DBP, and DIBP were the predominant compounds; concentrations of DEHP (median: 228 μg/g) were 10 times higher than the concentrations of DBP (20.1 μg/g) and DIBP (17.2 μg/g), and the concentrations of these three compounds were 100−1000 times higher than the concentrations of the other six phthalate esters analyzed. In dust samples collected from Albany, DEHP, BzBP, and DBP were the predominant compounds found; concentrations of DEHP (304 μg/g) were 10 times higher than the concentrations of BzBP (21.1 μg/g) and DBP (13.1 μg/g), 100 times higher than the concentrations of DEP (2.0 μg/g) and DIBP (3.8 μg/g), and 1000 times higher than the concentrations of the other six phthalate esters analyzed.

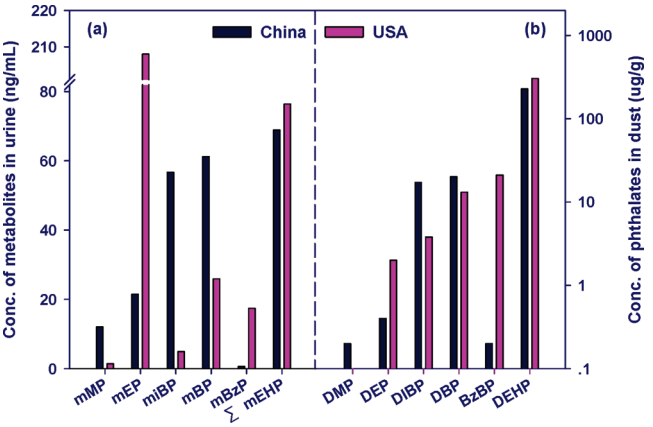

Figure 1. Concentrations of phthalate metabolites (ng/mL) in human urine and concentrations of phthalate esters (μg/g) in dust samples from China and the USA. (Concentrations of urinary phthalate metabolites for China are from ref 27 and for USA are from ref 35; mMP (monomethyl phthalate), mEP (monoethyl phthalate), miBP (mono(2-isobutyl phthalate); mBP (mono-*n*-butyl phthalate); Σ mEHP (sum of mono(2-ethyl-5-carboxypentyl) phthalate (mECPP), mono-[(2-carboxymethyl)hexyl] phthalate (mCMHP), mono(2-ethyl-5-oxohexyl) phthalate (mEOHP), mono(2-ethyl-5-hydroxyhexyl) phthalate (mEHHP), and mono (2-ethylhexyl) phthalate (mEHP) in China, and the sum of mECPP, mEHHP, mEHP, and mEOHP in the USA).

Concentrations of individual phthalate esters in dust samples were significantly different ($p < 0.05$) between China and the USA, except for DCHP and DEHP ($p > 0.30$). Concentrations of DEP, DNHP, and BzBP were 5 to 10 times higher in dust samples from the USA than those from China, whereas concentrations of DIBP were 5 times lower. No significant difference existed ($p > 0.10$) in the concentrations of total phthalates (sum of the nine phthalates) in dust samples from China and the USA. The median concentrations for total phthalates in dust samples from China and Albany (USA) were 295 and 396 μg/g, respectively.

Similar to what was reported in several earlier studies,[18,29−31] DEHP was the most abundant phthalate ester in indoor dust samples from China and the USA. The highest reported concentration of DEHP in house dust was 3210 μg/g, from Denmark.[29] In the present study, concentrations of DBP in dust samples from Albany (USA) were approximately 4 times higher than those of DIBP, much similar to the pattern reported from Norway,[30] Massachusetts, USA,[16] and Sweden.[18] Concentrations of DBP in dust samples from China were similar to the concentrations of DIBP, as reported from Germany [31−33] and Denmark.[19] Concentrations of DEP (on the order of 1.0 μg/g) in dust samples from Albany were lower than the values reported for Germany [31,33] (6 μg/g), Norway [30] (10 μg/g), and Bulgaria (340 μg/g),[34] whereas concentrations of DEP (on the order of 0.1 μg/g) in dust samples from China were the lowest reported to date.

Comparison of Phthalate Ester Concentrations in Dust with Urinary Phthalate Metabolite Concentrations. The median concentrations of urinary phthalate metabolites reported by the National Health and Nutrition Examination Survey (NHANES) of the USA in 2003−2004,[35] and the concentrations determined in urine samples collected from China in 2009 by our group, [27] are shown in part a of Figure 1. The median concentrations of phthalate esters in indoor dust samples from Albany (USA) and China, analyzed in the present study, are shown in part b of Figure 1. The patterns of phthalate esters in indoor dust

Table 2. Equations Used in the Calculation of Daily Intakes of Phthalate Esters by Dust Ingestion, Dermal Absorption, Inhalation, and Dietary Intake

routes	no.	daily intakes (DI, μg/kg-bw/day)	parameters
dust ingestion	1	$DI = \frac{C_{dust} f_1 f_2}{M_1}$	C_{dust} is phthalate concentration of dust (μg/g), f_1 is the indoor exposure fraction (hours spent over a day), f_2 is the soil ingestion rate (g/day), and M_1 is the body weight (kg);
dermal uptake	2	$DI = \frac{C_{soil/dust} A M_2 f_1 f_3}{M_1}$	$C_{soil/dust}$ is phthalate concentration of dust or soil (μg/g), A is the body surface area (cm^2/day), M_2 is the soil adhered to skin (mg/cm^2), and f_3 is the fraction of phthalate absorbed in the skin;
	3	$DI = \frac{CPCP_s M_3 f_4 f_5 f_6}{M_1}$	C_{PCPs} is phthalate concentration of personal care productions (PCPs) (μg/g), f_4 is the retention factor, f_5 is frequency of use of PCPs per day, f_6 is the dermal uptake fraction of phthalate, M_3 is the daily amount of PCPs;
inhalation	4	$DI = \frac{C_{air} f_1 f_7}{M_1}$	C_{air} is phthalate concentration of air (ng/m^3), and f_7 is the inhalation rate (m^3/day);
dietary intake	5	$DI_{dietary} = DI_{estimated} - DI_{inhalation} - DI_{dermal}$	$DI_{estimated}$ is the total phthalate intakes estimated from human urine phthalate metabolites and DI_{dermal} is the sum of dermal uptake of soil and dust and PCPs.

Table 3. Daily Intakes of Several Phthalate Esters from Indoor Dust (ng/kg-bw/day) for Various Age Groups in China (Several Cities) and the USA (Albany, New York)

	Dust Ingestion							Dust Dermal Absorption						
	DMP	DEP	DIBP	DBP	BzBP	DEHP	total	DMP	DEP	DIBP	DBP	BzBP	DEHP	total
							China							
infants	0.7	1.3	60.6	70.9	0.8	804	938	0.003	0.01	0.3	0.4	0.002	0.3	1.1
toddlers	0.9	1.6	71.5	83.7	0.9	949	1110	0.002	0.007	0.2	0.3	0.002	0.2	0.8
children	0.3	0.5	23.4	27.4	0.3	311	363	0.002	0.006	0.2	0.3	0.001	0.2	0.6
teenagers	0.2	0.3	14.3	16.7	0.2	190	221	0.001	0.004	0.1	0.2	0.0009	0.1	0.5
adults	0.1	0.3	12.0	14.1	0.2	160	186	0.0006	0.002	0.06	0.1	0.0005	0.07	0.2
							USA							
infants	0.3	7.1	13.4	46.0	74.3	1070	1210	0.001	0.06	0.06	0.3	0.2	0.4	1.0
toddlers	0.4	10.0	18.8	64.5	104	1500	1700	0.0007	0.04	0.05	0.2	0.2	0.3	0.8
children	0.1	2.8	5.2	17.8	28.8	414	468	0.0006	0.03	0.04	0.2	0.1	0.3	0.6
teenagers	0.1	1.7	3.2	11.1	17.9	257	291	0.0004	0.02	0.03	0.1	0.09	0.2	0.5
adults	0.1	1.4	2.6	8.8	14.3	206	233	0.0002	0.01	0.01	0.06	0.04	0.1	0.2

and the corresponding metabolites of phthalate esters in human urine were consistent. Concentrations of mEP (monoethyl phthalate, a metabolite of DEP) and mBzP (monobenzyl phthalate, a metabolite of BzBP), in urine samples from the USA were 1 order of magnitude higher than the concentrations found in urine samples from China, which corresponded with the concentrations of DEP and BzBP in dust samples from these two countries, that is, DEP and BzBP concentrations in house dust from the USA were 5 to 10 times higher than those in dust samples from China. A similar pattern was also observed for mMP (monomethyl phthalate, a metabolite of DMP) concentrations in urine and DMP concentrations in dust; mMP concentrations in urine from China were 10 times higher than the concentrations found for urine samples from the USA, which corresponded with (3 times) higher concentrations of DMP in dust from China than those from the USA. The similarity in patterns of phthalates in dust and their corresponding metabolites in urine suggest that dust is an important source of human exposure to phthalates.

Human Exposure to Phthalates via Dust Ingestion and Dermal Absorption. Humans can be exposed to phthalates in dust via ingestion and dermal absorption.[5,36] The DIs (ng/kg-bw/day) of phthalates through dust ingestion and dermal absorption can be respectively estimated with eqs (1) and (2), as shown in Table 2. Because body weights and dust ingestion rates vary by age, we estimated the DIs of phthalates for five age groups: infants (<1 yr), toddlers (1−3 yr), children (4−10 yr), teenagers (11−18 yr), and adults (≥19 yr). The details of the calculation and data sources are shown in Table S1 of the Supporting Information.

The estimated DIs of phthalates via dust ingestion and dermal absorption varied, depending on the age group (Table 3). In China, the total DI of phthalate esters through dust ingestion ranged from 186 ng/kg-bw/day for adults to 1110 ng/kg-bw/day for toddlers. The total DI of phthalate esters through dust dermal absorption ranged from 0.2 ng/kg-bw/day for adults to 1.1 ng/kg-bw/day for infants and decreased with age. In the USA, the total DI of phthalates through dust ingestion ranged from 233 to 1700 ng/kg-bw/day. The DI estimate for toddlers was higher than those for other age groups, in both China and the USA. The total DI of phthalate esters through dust dermal absorption was 0.2−1.0 ng/kg-bw/day and decreased with age. Overall, the total DI of phthalate esters through dust ingestion was 100−1000 times higher than that through dermal absorption.

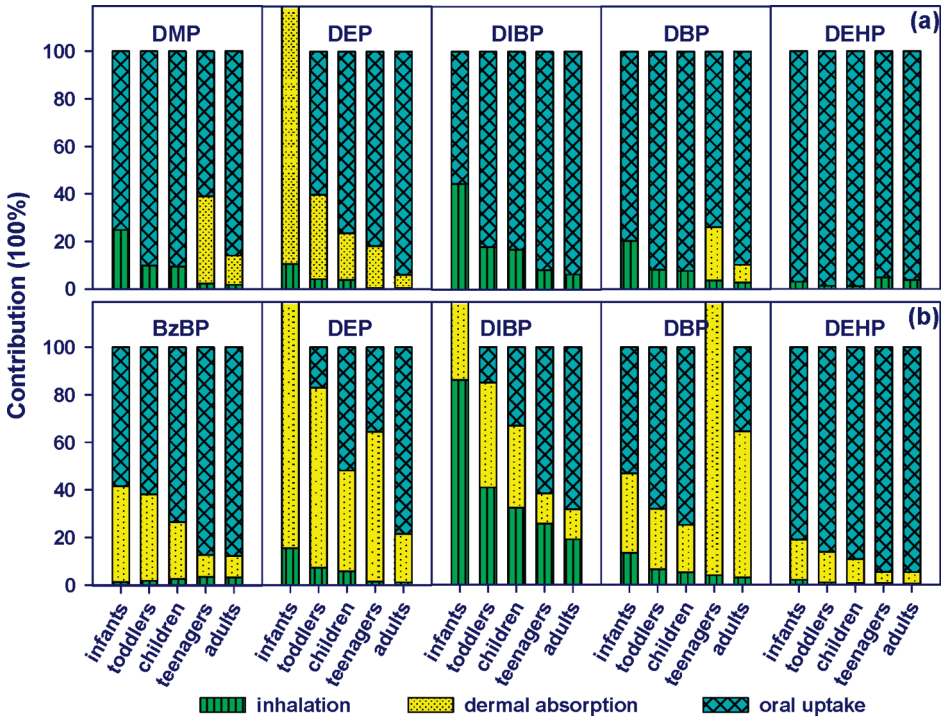

Figure 2. Contribution of different routes to total exposure to phthalates in (a) China and (b) the USA; the contributions of dermal absorption of DEP for infants were 113% and 237% in China and the USA respectively, and dermal absorption of DBP for teenagers and DIBP for infants in the USA were 178% and 58%, respectively.

Urinary phthalate metabolites have been used as biomarkers of exposure to phthalates. In our previous study,[27] we estimated the DIs of DMP, DEP, DBP, and DEHP on the basis of concentrations of phthalate metabolites measured in urine samples from China. Similarly, a few earlier studies have reported the total DIs of phthalates from urinary metabolite concentrations in the USA.[28,37] On the basis of the reports of total phthalate intake estimated from urinary concentrations, we calculated the contribution of dust (via dermal absorption and ingestion) to the total DI of phthalates. Our results indicated that indoor dust accounted for less than 0.2% of the total intakes reported for DMP and DEP, less than 2.2% of the intakes for DIBP and DBP, and 2−5% of the intakes for DEHP in China. In the USA, indoor dust accounted for less than 1% of the total intakes reported for DEP, 1−16% of the intakes for DIBP and DBP, 3−21% of the intakes for BzBP, and 10−58% of the intakes for DEHP (Table S2 of the Supporting Information). The results indicated that indoor dust is an important source of human exposure to some phthalate compounds, especially for the high-molecular-weight phthalate esters.

The DIs of phthalate esters via dust ingestion have been reported in previous studies. In Berlin, Germany,[33] respective DI values for DEHP, DBP, DEP, and BzBP were 110, 8, 6, and 10 ng/kg-bw/day for adults; the estimated DIs of phthalate esters for children in Germany were 50 times higher than those estimated for adults. The estimated intake of DEHP via dust ingestion by children in Denmark was ∼1000 ng/kg-bw/day [19] and was 5000−54100 ng/kg-bw/day (age <11) in the USA.[38] The estimated intakes of DEHP by the US children in our study were lower (400−1500 ng/kg-bw/day) than those reported earlier.[38]

Contribution of Various Routes to Phthalate Exposures. On the basis of the total DI values estimated from urinary concentrations in China[27] and the USA,[28,37] we estimated the contribution of dust and other sources/pathways to phthalate exposures for various age groups. Because data were not available for all compartments and for all phthalate esters, the exposure assessment required several assumptions and approximations. However, such assessment will help in understanding the major pathways of human exposure and has been performed for several European countries and the USA.[5,7,38] To our knowledge, this is the first study to examine the sources and pathways of human exposure to phthalates in China.

For the exposure assessment, we estimated the DIs through inhalation (via indoor and outdoor air) and dermal absorption (via dust, soil, and personal care products) pathways. On the basis of these estimates, we calculated dietary intakes as total DIs minus DI values estimated for dermal and inhalation pathways (eqs 2−5, Table 2). The reported concentrations of phthalates in indoor and outdoor air, soil, personal care products, and the measured concentrations of phthalate esters in dust in this study were used for the estimation of DIs via dust ingestion, dermal absorption, and inhalation.[5,24,28,31,37,39−44] Details regarding all parameters and values used are given in Tables S1 to S5 (Supporting Information).

The contributions of various routes to total DIs of phthalates by the Chinese and US populations are shown in Figure 2. Because of the low concentrations of mMP in urine samples from the USA[1,35,45] and mBzP in urine samples from China,[27] we did not estimate DIs for these two phthalate esters.

In China, as shown in part a of Figure 2, dietary intake was the main source of exposure to all phthalate esters, especially for DEHP (>95%) for all age groups. For DMP, inhalation

accounted for 2−25% of the total intakes, and decreased with age. For DEP, because of its high concentrations in personal care products [5,43] and its high dermal absorption coefficient,[5] dermal absorption was the dominant source for infants (114%), and accounted for 5−36% for other age groups. For DIBP and DBP, inhalation accounted for 6−44% of the total intake.

In the USA, dietary intake was the main source of BzBP exposure for all age groups (>58%). Dermal absorption accounted for ∼40% of the BzBP intake for infants and toddlers. Dietary intake was the main source for DEP exposure among US adults and children (52−78%), whereas dermal absorption was dominant for infants, toddlers, and teenagers (>63%). Inhalation was the dominant pathway of exposure for DIBP among US infants (>86%), whereas dietary intake predominated for adults and teenagers (>60%). Dermal absorption was the dominant source of DBP for U.S. teenagers and adults (62−179%), whereas dietary intake was the main source for other groups (53−75%). For DEHP, dietary intake was the main contributor to exposure for all age groups (>86%) in the USA, and its contribution increased with age.

Overall, for all phthalate esters in the two countries, dietary intake is the major source of exposure to phthalates in adults; contributions of dietary sources were lowest for infants and increased with age; contributions of dermal absorption and inhalation were highest for infants and decreased with age. Intakes of DEP, DIBP, DBP, and DEHP through dermal absorption and of DEP and DIBP through inhalation were higher for the populations in the USA than for those in China. The contributions from dietary intakes of DEP, DIBP, DBP, and DEHP in China were higher than those in the USA.

The sum of contributions of all the three exposure routes examined in this study, to the total DIs estimated from urinary phthalate metabolite concentrations, totaled 100%, with few exceptions. Therefore, the approach used in the present study for the estimation of contribution from inhalation, dermal absorption, and dietary intake is valid. The contribution of dermal absorption to DEP intake in infants from China and the USA, and to DBP intake in teenagers in the USA, was greater than 100%; this is because the DIs estimated from dermal absorption exceeded the total DIs estimated based on urinary metabolite concentrations. This suggests that the estimates of DIs based on urinary metabolite concentrations of phthalates are potentially underestimated or that the dermal absorption coefficients used may be overestimated.

Our results suggest that the contribution of dietary sources to the DI of phthalates increases with age, as has been reported for EU countries and Japan.[5,6] Dietary intake was the main source of exposure to DEHP, whereas dermal dust ingestion/absorption was the major source of exposure to DEP.

■ ASSOCIATED CONTENT

Ⓢ Supporting Information. Map of dust sampling locations in China, instrumental analysis method, parameters and sources used for the calculation of DIs of phthalates through various routes, and estimated daily intake results. This material is available free of charge via the Internet at http://pubs.acs.org.

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