



Research Article

Di-(2-Ethylhexyl) Phthalate (DEHP) in House Dust in Canadian Homes: Behaviors and Associations with Housing Characteristics and Consumer Products

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Background. Di-(2-ethylhexyl) phthalate (DEHP), which is ubiquitous in indoor environments, was the predominant phthalate measured in house dust in the Canadian CHILD Cohort and was found to be associated with a large increased risk of childhood asthma. **Objective.** To inform interventions by identifying sources of DEHP in dust and assessing behaviors related to DEHP concentrations in house dust. **Methods.** DEHP levels were measured in 726 dust samples collected at ~3 months of age in CHILD as well as in ~50 homes at two time points (June and November) in the CHILD pilot study. DEHP metabolites were measured in urine for a subset of the ~3-month-old infants. Housing characteristics were assessed at the time of dust and urine collection. Numerous factors from these surveys were investigated as potential sources of DEHP using univariate analyses and multivariable regressions. Correlations between DEHP in dust and urinary metabolites and between repeat dust samples were examined to study the relationship between dust measurement and DEHP exposure. **Results.** Overall, DEHP dust concentrations were higher for lower-income families. Homes with vinyl flooring in the kitchen and bathroom showed higher levels of DEHP than those without vinyl flooring. The quantity of vinyl furniture and the presence of mold were associated with higher DEHP concentrations, while the use of mattress covers reduced concentration. No other significant associations were found. DEHP concentrations in dust were consistent over 6 months, although the correlation between dust and DEHP metabolites in urine was low. **Conclusion.** DEHP in house dust persisted over multiple months, contributed to infant internal exposure, and was associated with specific housing characteristics. These findings may inform the public on their choice of building materials and products, as well as future policies, aimed at reducing the health risk associated with exposures in the indoor environment especially for children.

1. Introduction

Phthalates are a group of widely used semivolatile organic compounds and are ubiquitous in the indoor environment [1–3]. Low molecular weight phthalates, such as diethyl

phthalate and dibutyl phthalate, can be commonly found in personal care products, cosmetics, and adhesives [4], while high molecular weight (HMW) phthalates, such as di-(2-ethylhexyl) phthalate (DEHP), are widely used as plasticizers and can be found in vinyl-containing materials and

products (e.g., food packaging, vinyl furniture and flooring, and polyvinyl chloride (PVC) panels) [4–6]. Since phthalates are not chemically bound to those materials, they can be released into the surrounding environment and be present as gas, particle, and dust phases [7, 8].

Among all phthalates commonly existing in the indoor environment, DEHP has been frequently detected in house dust at high concentrations [5, 9–15]. Due to its high molecular weight and low vapor pressure, gaseous DEHP, which may present a brief inhalation exposure, also adheres to surfaces, can be resuspended in air, and settles as dust particles in the home [16]; thus, house dust acts as a reservoir and can play an important role in nondietary exposure pathways to DEHP. The transfer from DEHP-containing products to the dust phase at room temperature occurs slowly; thus, the emission of DEHP from those products to indoor air can be chronic, persisting for several months and even years with ongoing exposures once incorporated into dust [17]. Compared with urine measurements of DEHP exposure, which characterize the total exposure from all routes, but only reflect acute exposure levels due to its short half-life (<48 h) [18–20], a one-time dust measurement can better represent chronic exposure to this chemical associated with the indoor environment [19, 21, 22]. Once phthalates are partitioned into dust, they can enter human bodies through multiple routes, including inhalation, dermal absorption, and ingestion [23]. Since children have frequent hand-to-mouth behavior and spend time closer to the floor (i.e., sitting, playing, crawling), they are more likely to be exposed to higher levels of pollutants through dust [2].

Phthalates, as one class of synthetic endocrine-disrupting chemicals, are suspected to modify hormone levels and alter the functionality of immune systems [24, 25] and may contribute to inflammation [13, 26]. Exposure to phthalates has been linked to many adverse health outcomes in children and adults, including allergic diseases and asthma [1, 14, 27–29], neurological disorders such as attention-deficit/hyperactivity disorder, and decreased neurodevelopmental performance [30–32]. Young children are more vulnerable to phthalate exposure due to their developing immune system and disproportionately higher exposure level; and the first year of life is thought to be the critical time window for environmental exposures, meaning that exposure during this time period may lead to long-term impacts on health [33, 34]. A previous epidemiological study using a large Canadian birth cohort, CHILD, found that DEHP is the most prevalent phthalate in dust in Canadian homes, and exposure to DEHP during the first year of life was significantly and strongly associated with asthma diagnosis at age 5 and recurrent wheeze between 2 and 5 years [14].

Although DEHP has been banned in cosmetics and been regulated in the soft vinyl used in toys and childcare articles (e.g., products for feeding and teething) to 1,000 mg/kg, or <0.1% by weight (*w/w*) in Canada since June 2011 [35], it has not been restricted in other household consumer products which may contain DEHP, thus leading to higher concentrations in the home environment and contributing to children's overall exposure. Although possible common

sources of DEHP have been widely reported, it remains unclear what characteristics of the home environment explain the large variability of DEHP concentrations in dust in Canadian homes. It is important to determine those factors to inform exposure reduction measures and identify children at high risk for exposure. Some housing factors that may not be the direct source of DEHP but can enhance the release of DEHP from sources or reduce DEHP accumulation in dust are also important to investigate for relevant interventions.

Further, social factors such as socioeconomic status (SES) have also been suggested to have a significant impact on phthalate exposure [36, 37]. For example, lower-income families are likely to be exposed to higher phthalate levels as measured by urine [19]. However, few studies used dust measures when examining the impact of SES on phthalate exposure, and the possible underlying factors explaining the relationship are not well understood. Our study primarily aims at identifying factors influencing DEHP levels in house dust in Canadian homes by examining associations with housing characteristics in the CHILD Cohort Study. Furthermore, we examined whether SES affects DEHP exposure and people's choice of building materials and consumer products. CHILD undertook extensive home environment assessments at multiple time points [38] providing a unique opportunity to explore factors related to potential indoor DEHP sources and behaviors that may influence the level of DEHP in dust. Given the reliance on house dust DEHP concentration in CHILD as an indicator of exposure, in this paper, we also aimed to gain more insight into the representativeness of this single-time, pooled (i.e., from two rooms) dust DEHP measure by examining its relationship with total but more acute exposures, as indicated by urine measurements [19], and also within the home and seasonal variability of DEHP concentrations in the dust.

2. Methods

2.1. Study Population. This study is based on data from the CHILD Cohort Study. CHILD is a multicenter longitudinal birth cohort study that recruited more than 3500 pregnant women from four sites across Canada: Toronto, Vancouver, Edmonton, and Manitoba (Winnipeg, Morden, and Winkler) from 2008 to 2012 and followed the children of those participants into childhood and adolescence to collect information on exposures and health outcomes [39]. The study was designed to understand the impacts of genetic and environmental factors during pregnancy and early life, on the development of allergy, asthma, and other noncommunicable diseases later in life.

A case-cohort study nested within CHILD was subsequently designed to investigate the association between asthma and recurrent wheeze and phthalate exposure measured from settled dust; thus, a subgroup ($N = 726$) of house dust samples was selected from the whole cohort for assessing phthalate exposure. Details of this case-cohort study design including inclusion and exclusion criteria have been described in our previous study [14]. A pilot study, referred to as mini-CHILD, was conducted in Vancouver before the

main CHILD study to evaluate exposure assessment protocols and refine those methods before implementation in the main cohort. The mini-CHILD study was based on 50 homes that were randomly selected and visited for dust collection and home environment investigation at two time points approximately six months apart.

Ethics approval for study protocols was obtained from each of the study center's respective research ethics boards, and approval to conduct these analyses was received from the University of Toronto Health Sciences Research Ethics Board (protocol no. 36169).

2.2. Sample Collection and Analyses. House dust samples were collected by trained research assistants (RAs) during a comprehensive home environment assessment when the infants were approximately 3-4 months old, between 2010 and 2012. For the pilot study ("mini-CHILD") conducted in Vancouver, two home visits for dust sampling were completed in June and November separately in 2010. During each visit, dust was sampled in both the bedroom and the most used living room.

A consumer-model vacuum cleaner attached to a dehydrogenated aluminium nozzle accommodating two nylon thimble filters was used to maximize dust collection [38]. Sampling in the most used living room was conducted on a 2 m² area of carpet or the whole room if there was no carpet in the room. Sampling in the bedroom, determined to be the child's primary sleeping area, involved vacuuming a combination of the surface of mattresses or mattress covers, if used, and the adjacent floor space. The two samples were pooled for analysis in CHILD, while they were analyzed separately in mini-CHILD, for which a total of 35 homes during the first visit and 38 homes during the second visit had DEHP data for both rooms, which were further used to assess the variability of DEHP in dust concentrations within each home.

To ensure quality assurance and quality control (QA/QC), a control dust sample was taken by using 100 mg of the NIST vacuum dust standard (SRM 2585) for every 20th home sampled. All dust samples in the thimbles, including the control sample, were stored in sterile glass bottles and transported to the lab within 2 weeks of collection. In the processing lab, large observable particles and materials were removed from the sample, and the remaining samples were sieved through a 150 μ m screen and were weighed and stored in aliquots at -80°C for biological and chemical analyses. For samples analyzed as part of the case-cohort study, equal amounts of dust from the child's sleeping area and the most used living area were combined, consisting of ~50 mg of dust in each room (~100 mg total), and then a 20-25 mg aliquot was analyzed by gas chromatography mass spectrometry (Agilent GC-MSD, Agilent Technologies Inc., Palo Alto, CA, USA). Details of the analytical procedure can be found in our previous work [14].

Urine samples were collected at the same time as dust collection when the child was at around 3 months of age. Details of the sampling and processing of urine samples have been detailed previously [38]. Briefly, urine samples were collected by research assistants during the home visit from

babies' diapers which were fresh at the start of the visit and included a plastic barrier and separate absorbent cotton pads which were subsequently squeezed at the end of the visit for urine collection. The lab analysis includes an enzymatic deconjugation, followed by high-performance liquid chromatography with tandem mass spectrometry, and all urine metabolite concentrations were adjusted by specific gravity, which was determined at the time of urine collection. A subset of these samples was selected for phthalate metabolite analysis, as reported by Navaranjan et al. [19]. In this paper, we focused on the sum of three DEHP metabolites ((Σ DEHP), mono-2-ethylhexyl phthalate (MEHP), mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP), and mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)) from the urine obtained at the same time as dust collection. There were a total of 351 urine measurements corresponding to children with available DEHP in dust data.

2.3. Home Environment Assessment. Housing characteristics were assessed by trained RAs at the time of dust collection ("home assessment"), and a home environment-related questionnaire was completed by the family. Details of these two complementary exposure assessment tools were described by Takaro et al. [38]. Briefly, various physical environmental exposures across 15 domains have been characterized in CHILD homes, mainly including general home characteristics (e.g., period of home construction and dwelling types), indoor pollution exposure sources (the covering of floors, walls, and furniture; cleaning and chemical products; personal and childcare products; mold indicators; and pets), and traffic-related air pollution. For our study, we identified potential predictors for DEHP concentration in dust based on previous literature and practical knowledge of the possible ingredients used in those materials and products. Thus, we examined multiple housing factors related to general home characteristics such as home construction period and dwelling type; building materials such as flooring and wall covering; and behavior-related factors such as the use of plastic/vinyl furniture and toys, polish products, cleaning habits, and factors related to home ventilation, such as ambient temperature on the dust/urine sampling day for each participant, which was obtained for each study center from the long-term climate station operated by Environment and Climate Change Canada.

2.4. Statistical Analysis

2.4.1. Primary Analysis. The concentrations of DEHP on the original scale were right-skewed; therefore, natural log-transformed DEHP concentrations were used in statistical analyses. First, univariate analyses were conducted to compare the distribution of DEHP by housing characteristics using the *t*-test and ANOVA for normally distributed log-transformed data and the Mann-Whitney *U*-test and Kruskal-Wallis test for nonnormally distributed data. Box-plots were used to present the differences in DEHP concentrations by these housing characteristics, and the results of univariate analyses were shown in the plots.

Then, for those variables reaching significant levels in the univariate analyses, multivariable linear regression models were performed to assess the association between these housing factors and the continuous DEHP concentrations in the dust to identify the strongest possible sources in the CHILD homes. Conclusions of the possible sources and related factors were made based on the multivariable regression model. Three covariates were adjusted for in the model: household income (<60,000, 60,000-100,000, 100,000-150,000, and >150,000), outdoor temperature on the sampling day, and study center. Household income was found to be related to phthalate metabolite concentrations in the CHILD Cohort [19]. The average outdoor temperature on the sampling day was considered to be a better indicator of the season of sampling given the diversity of climate among the centers and may also indicate other relevant factors such as indoor-outdoor exchange rate or ventilation (i.e., more frequent window opening during warmer outdoor temperatures). The study center was included as a covariate considering that homes from four cities might have different home conditions (e.g., consumer product choices and other unmeasurable factors) or possible differences in research assistant approaches for the home assessment.

To validate associations found for DEHP dust levels, DEHP concentrations were also dichotomized based on the median value. Then, logistic regression models were performed to examine the association between the binary DEHP (high/low exposure) and those characteristics. Log-transformed DEHP concentrations were added to the model as the dependent variable, and each factor was added as an independent variable. DEHP levels were further categorized into quartiles, and a chi-square test was used to investigate the relationship between DEHP quartiles and each housing characteristic.

To determine whether to stratify the analysis of possible sources by specific factors, we first assessed whether the flooring type (carpeting vs. noncarpeting) that was vacuumed for dust sampling by RAs had an impact on DEHP levels. Since no significant association was found, we did not stratify analyses by the vacuumed floor type.

2.4.2. Secondary Analysis. To assess the relationship between urinary DEHP metabolites and DEHP concentration in dust, those participants with both urine and dust samples ($N = 354$) were included in this subanalysis, and the Spearman correlation coefficient was calculated. The molar sum of DEHP metabolites was calculated (formula: $(\sum \text{DEHP}(\text{nmol/ml}) = (\text{MEHP} * (1/278.34)) + (\text{MEHHP} * (1/294.34)) + (\text{MEOHP} * (1/292.33)))$) to represent DEHP metabolites in urine as a total.

Using the pilot study data ("mini-CHILD"), the correlations of phthalate dust concentrations between two home visits were calculated to examine the consistency of measurements over time. DEHP concentrations measured in mini-CHILD were further dichotomized into high and low concentrations based on the median level and examined for consistency of levels between the two rooms where dust was collected: the child's bedroom and the most-used living room.

To assess the effect of SES (household income) on DEHP exposure, ANOVA was used to determine the difference of

DEHP dust concentrations across four categories of household income. Then, multivariable linear regression was performed to further examine the association between household income and DEHP, adjusting for study center and house volume. The associations between SES and housing factors related to DEHP concentrations were examined using the chi-square test. This analysis is aimed at exploring the underlying factors which may explain the relationship between SES and DEHP exposure. All analyses were conducted in RStudio.

3. Results

3.1. Participant Demographics and Building Characteristics.

A total of 726 families with DEHP measured in dust in CHILD were included in the analyses. Table 1 describes the characteristics of those households. Participants were almost equally distributed across four geographic locations across Canada, with a higher percentage from Manitoba and Toronto (~30% each) and a relatively lower percentage from Vancouver and Edmonton (~20% each). Nearly half (47%) of the participating homes had an average annual household income over CA\$100,000. Half of the families lived in a single-family detached house (51%), followed by apartments (20%) and townhouses or semidetached houses (17%). Only 6% of the families lived in a multifamily building. Forty percent of the homes were built between 1940 and 1990; 16% were built before the 1940s; and 30% were built after 1990. Over half of the dust samples were collected when the outdoor temperature was between 0 and 16°C, and 21% and 25% of samples were collected when it was below 0°C and above 16°C (representing relatively colder and warmer seasons, respectively). The distribution of all housing characteristics related to building materials and human-controlled behaviors that were investigated in our study is summarized in Table S1 (building materials from the RA assessment) and Table S2 (consumer products and behaviors potentially related to DEHP exposure). Over 50 housing characteristics that were derived from the home environment questionnaire and RA home assessment were examined for their potential impacts on DEHP levels in the dust. However, several building characteristics were rarely observed (i.e., >98% did not use the material) in this cohort, such as vinyl flooring in bedrooms and living rooms and wallpaper and PVC panel as the wall covering; thus, they were excluded from being further analyzed. Only characteristics with adequate variation (frequency >2% in each category of the factor investigated, e.g., more than 2% answered 'yes' to using a product) were analyzed for their associations with the DEHP concentration.

3.2. DEHP Concentrations in Dust and Urine. Table 2 summarizes the concentrations of DEHP in dust and its metabolites (MEHP, MEOHP, and MEHHP) measured in urine when children were 3 months of age. Over 99% of all 726 dust samples analyzed in our study had detectable levels of DEHP. The median concentration of DEHP in dust in this study was 232 µg/g, with a broad range from 9.6 µg/g to 2675 µg/g and a right-skewed distribution. The median

TABLE 1: Characteristics of the 726 homes in CHILd with phthalate measured in dust.

Characteristic	Total (N = 726)
Study center	
Edmonton	140 (19.3%)
Toronto	200 (27.5%)
Vancouver	173 (23.8%)
Manitoba	213 (29.3%)
Household income (CA\$)	
<60000	116 (16.0%)
60000-100000	186 (25.6%)
100000-150000	190 (26.2%)
>150000	153 (21.1%)
Missing	81 (11.2%)
House volume (excluding basement) (m ³)	
0-200	187 (25.8%)
>200-250	150 (20.7%)
>250-350	190 (26.2%)
>350	178 (24.5%)
Home built period	
1939 or earlier	115 (15.8%)
1940-1969	142 (19.6%)
1970-1989	143 (19.7%)
1990 or later	221 (30.4%)
Missing	105 (14.5%)
Dwelling type	
Single-family detached house	371 (51.1%)
Townhouse/semi-detached	120 (16.5%)
Apartment	146 (20.1%)
Multifamily home	43 (5.9%)
Other	13 (1.8%)
Mean ambient temperature on the sampling day (°C)*	
<0	151 (20.8%)
0-≤10	225 (31.0%)
>10-16	163 (22.5%)
>16	182 (25.1%)
Missing	5 (0.7%)

*Outdoor temperature was used as a proxy for the season.

urinary DEHP concentration was 0.6 ng/mL, 1.47 ng/mL, and 1.54 ng/mL for MEHP, MEOHP, and MEHHP, respectively, which were consistent with results from the larger set reported by Navaranjan et al. [19].

3.3. DEHP Levels in Dust by Home Conditions. DEHP concentrations in dust were significantly different by multiple housing characteristics. Figure 1 presents the significantly different distribution of natural log-transformed DEHP concentration ($\mu\text{g/g}$) by those factors. No more than 2% of the homes had a recent renovation involving the addition of vinyl flooring since the child was born, and thus this effect could not be examined.

Multiple combinations of questions about vinyl floor in the kitchen and bathroom were used to examine the effects of using vinyl floor in the home. Significantly higher concentrations of DEHP were observed in homes using vinyl floor tiles in both the kitchen and the bathroom compared with homes without vinyl tiles in any of these rooms (Figure 1(a)). Similarly, higher concentrations were observed in homes with vinyl floor tiles in any of these two rooms compared to other flooring types. Older (>3 years) and newer vinyl flooring (≤ 3 years) in these two rooms were compared and did not impact on DEHP concentrations. Significantly higher DEHP levels were also observed among homes with more pieces of plastic or vinyl furniture (Figure 1(b)). There was evidence that homes with mold had significantly higher DEHP concentrations (Figure 1(d)). Additionally, significantly lower DEHP concentrations were observed among families using an allergy control mattress covering on the child's own sleeping bed in the child's bedroom (Figure 1(c)). This inverse relationship still remained after adjusting for parental history of asthma and household income in regression models. No significant difference of DEHP concentration was observed for other building characteristics and consumer products (e.g., dwelling type, plastic toys and playmats, plastic window blinds, home construction period, polish products, and painting in the bedroom and living room) that were investigated. Those insignificant results are presented in Figure S1.

Results using dichotomized DEHP concentrations as the outcome are consistent with those found with the continuous DEHP concentrations (Table S3). Homes with the highest DEHP quartiles were more likely to use vinyl flooring and plastic/vinyl furniture and less likely to use mattress coverings compared to homes with lower DEHP quartiles (Table S4).

3.4. Associations between DEHP Concentrations and Housing Characteristics. Table 3 presents the results of the associations between DEHP mass concentrations and home conditions based on multivariable linear regression. The quantity of plastic/vinyl furniture, vinyl flooring in the kitchen and bathroom, and mold was included in the model to identify their relative influence on DEHP in dust. All of these variables remained significantly associated with continuous DEHP levels in the model after adjusting for study center, household income, and ambient temperature on the sampling day. The use of vinyl flooring in the kitchen and bathroom was found to have the largest magnitude of association with DEHP concentration in dust.

3.5. Household Income and DEHP Concentrations and Related Housing Factors. Figure S2 suggests that families with higher household income tend to have lower levels of DEHP concentrations. To further examine the association of income with DEHP levels, multivariable linear regression models were performed for income and log-transformed DEHP concentration adjusting for study site and house volume. The study site was adjusted in the model due to differences in housing characteristics across four cities. House volume, which was measured by RAs during home

TABLE 2: Concentrations of DEHP measured in house dust ($\mu\text{g/g}$) and corresponding DEHP metabolite concentration (ng/mL) measured in urine, both sampled at 3 months of age.

Phthalate and metabolites	N	Min	5th percentile	Median	95th percentile	Max	Geomean
DEHP in dust ($\mu\text{g/g}$)	726	9.6	65.3	231.9	858.5	2675.3	232.9
MEHP (ng/mL)	354	0.0007	0.06	0.6	5.7	59.3	0.6
MEOHP (ng/mL)	354	0.05	0.3	1.5	5.9	30.7	1.3
MEHHP (ng/mL)	351	0.07	0.3	1.5	8.6	43.6	1.5
Σ DEHP in urine (nmol/mL)	—	0.0005	0.0032	0.01	0.074	0.3	0.02

Notes: MEHP is the primary DEHP metabolite, and MEOHP and MEHHP are the secondary metabolites measured in urine samples. Σ DEHP in urine indicates the molar sum of these three DEHP metabolites in urine and was calculated using the formula (Σ DEHP(nmol/mL) = (MEHP * (1/278.34)) + (MEHHP * (1/294.34)) + (MEOHP * (1/292.33))).

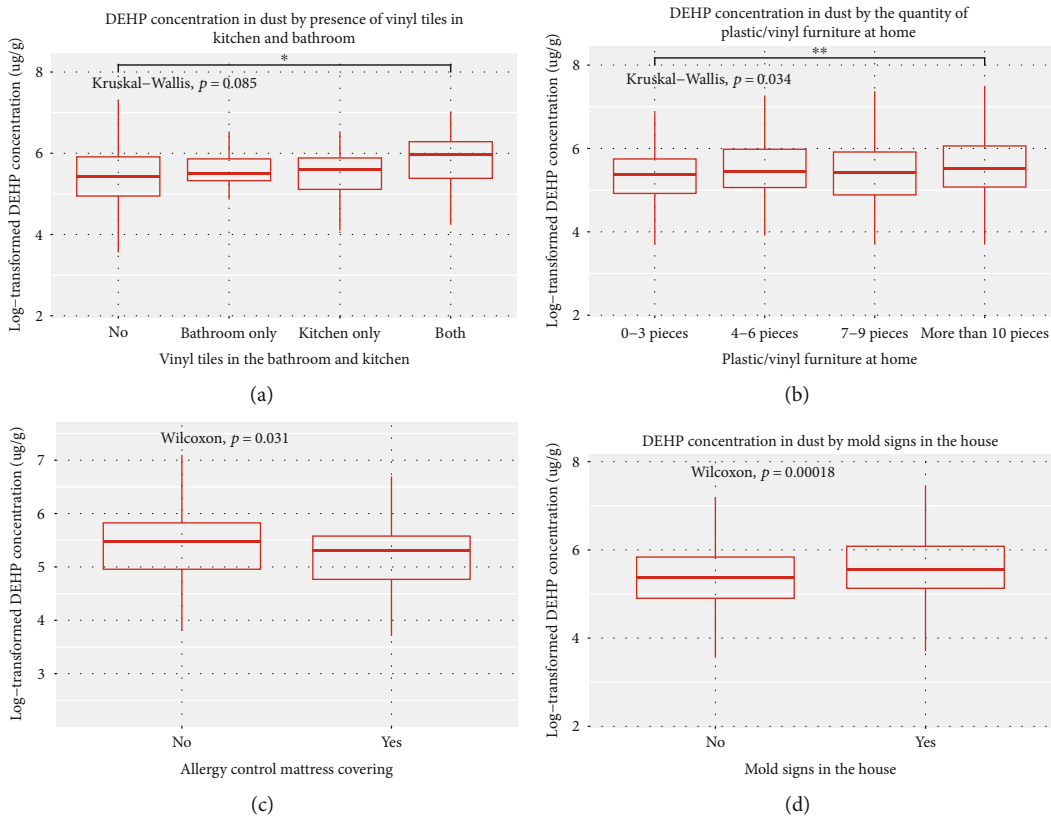


FIGURE 1: Differences of DEHP concentrations by housing characteristics: (a) 962 vinyl floor tiles in the kitchen and bathroom; (b) the quantity of plastic/vinyl 963 furniture; (c) mold signs at home; (d) allergy control mattress covering used on 964 the baby’s sleeping bed. Notes: the star marks in (a) and (b) represent the significance level of the difference between two specific groups (i.e., compared “both” with “no” in (a) and compared “0-3 pieces” with “more than 10 pieces” in (b)). “*” indicates p value < 0.05 . “**” indicates p value < 0.01 . The Kruskal-Wallis p value indicates whether there were significant differences across the four categories overall.

assessment, was found to be related to DEHP levels in univariate analyses. Dwelling type was considered but included in the model due to its high correlation with house volume. We found that the association between DEHP concentrations in dust and income remains statistically significant ($\text{Pr}(>F) = 0.04$) from the multivariable regression. Therefore, higher-income families in CHLD are likely exposed to lower DEHP concentrations in dust.

The association between household income and the factors related to DEHP in our study was investigated to examine whether the relationship between income and DEHP could be reflecting income-related differences in home conditions.

Table S5 shows that households with higher income levels were less likely to use vinyl flooring in the kitchen or bathroom ($p < 0.05$) and less likely to exhibit signs of mold (p value at the borderline of 0.05). Higher-income families also tended to report using mattress covers on the baby’s own sleeping bed. No association was found between household income and the number of pieces of plastic/vinyl furniture.

3.6. *Representativeness of a Single House Dust Sample.* To understand the extent that DEHP in house dust influenced children’s actual total exposure to DEHP, we examined the correlation between DEHP in dust and its metabolites in

TABLE 3: Adjusted associations between DEHP concentration in the dust ($\mu\text{g/g}$) and housing characteristics from the multivariable linear regression model.

Home characteristics	Effect estimates on log scale (95% CI)	% change of DEHP concentrations compared to the reference group (95% CI)	Pr > t	p value (ANOVA)
Quantity of plastic/vinyl furniture				0.03*
0-3 pieces ^a	0	—	—	
4-6 pieces	0.25 (0.08-0.43)	28% (8%-54%)	0.004**	
7-9 pieces	0.09 (-0.10, 0.28)	9% (-10%-34%)	0.37	
More than 10 pieces	0.20 (0.01-0.39)	22% (1%-48%)	0.04*	
Vinyl flooring in the kitchen or bathroom				0.05 .
No ^a	0	—	—	
Bathroom only	0.05 (-0.32-0.42)	5% (-28%-52%)	0.78	
Kitchen only	0.07 (-0.31-0.44)	7% (-27%-55%)	0.72	
Both kitchen and bathroom	0.56 (0.15-0.97)	75% (16%-164%)	0.007**	
Mold signs in the home				0.01**
No ^a	0	—	—	
Yes	0.17 (0.04-0.3)	19% (4%-35%)	0.02*	

Models adjusted for study center, ambient temperature on the sampling day, and household income. “% change of DEHP concentrations compared to the reference group” is presented to suggest the effect of each factor on DEHP in dust concentrations in a more intuitive way. This is calculated using the formula $\% \text{change} = (e^{\beta} - 1) * 100$. ^aThe reference group of each variable. Stars represent statistically significant: *: p value < 0.05; **: p value < 0.01; .: p value < 0.1. Pr > |t| indicates the significance level of each group of the variable compared to the reference group of the variable.

urine samples, both collected at 3-4 months of age. Additionally, we assessed how representative one single house dust sample, as done for all of the CHILD, is for chronic exposure to DEHP by examining the within-home and temporal variability of DEHP concentrations in dust in the subsample with two measurements.

3.7. Correlation between DEHP in Dust and Its Metabolites in Urine. Correlations between oxidative metabolites (i.e., MEOHP and MEHHP) measured at the same time point were high ($r > 0.8$, $p < 0.05$), as shown in Figure 2. The Spearman correlation between DEHP in dust and the molar sum of DEHP metabolites in urine that were both measured at 3 months of age was low but significant ($r = 0.21$, $p < 0.05$).

3.8. Within-Home and Temporal Variability of DEHP Concentrations in Dust. Correlations between DEHP concentrations measured at two time points, six months apart, were moderate to high, as presented in Table S6. Those correlations were calculated separately for bedrooms (Spearman’s correlation coef. = 0.62, $p < 0.01$) and living rooms (Spearman’s correlation coef. = 0.70, $p < 0.01$). To gain insight into the variability of DEHP within homes (i.e., to what extent does DEHP spread among rooms), the mini-CHILD data from the two separate rooms were compared. The geometric mean concentrations of DEHP were slightly higher in the bedroom (shown in Table S6), and correlations between the two rooms during each visit were moderate; the Spearman correlations were 0.45 ($p < 0.01$) and 0.44 ($p < 0.01$) during the first and second visits, respectively. When DEHP levels in each room were further dichotomized (high/low) based on the median level, we found that in the 1st visit, 75% of homes with a low level of DEHP

concentrations in the bedroom also remained low in the most used living room, while 64% of homes with a high level of DEHP concentrations in the bedroom remained high in the living room. Similar patterns were observed for the 2nd home visit (shown in Table S7).

4. Discussion

To our knowledge, this is the largest study to date investigating factors in the home environment influencing DEHP concentration in house dust within a multicenter birth cohort. We were uniquely able to undertake a comprehensive assessment of possible sources of DEHP in typical Canadian homes given the unprecedented data on home environments, including detailed home assessments by trained research assistants, collected as part of the CHILD Cohort Study. We further explored the impact of socioeconomic status (household income) on DEHP levels with the inclusion of several explanatory factors.

We found that household income was significantly associated with DEHP concentrations in house dust after adjusting for confounding factors. Consistent with our previous observation based on urine samples [19], lower-income families tend to have a higher level of DEHP concentration and that, at least partially, housing characteristics contribute to this pattern. To evaluate the utility of dust samples as an exposure indicator of DEHP, we assessed the stability of DEHP concentration across time, its variation within the home, and its correlation with total exposure indicated by urine metabolites. We found that the correlations between DEHP concentrations measured during two home visits (~6 months apart) were moderate to strong, indicating that the level of DEHP in dust was consistent over a relatively

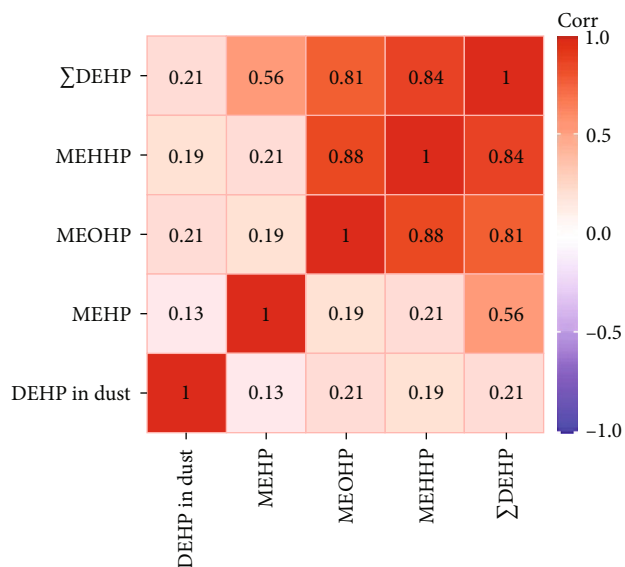


FIGURE 2: The Spearman correlations between DEHP in dust ($\mu\text{g/g}$) and urinary DEHP metabolites (ng/mL) collected in children at 3 months. Notes: (1) blank pixels represent insignificant Spearman correlations ($p > 0.05$). (2) Pixels with color represent significant correlation, and the darker the color is, the higher the correlation is, which is also reflected by the values.

long period of time. From the dust and urine correlation analysis, we found that DEHP in dust significantly contributed to infant total exposure in the CHILD subjects. These findings altogether suggest that dust is a good surrogate for chronic DEHP exposure in children and thus is rationale supporting the use of dust concentrations as an exposure metric in epidemiological studies assessing the health effect of DEHP exposure. We also found considerable variability within the home, but when DEHP levels were high or low in the most-used room relative to other homes, they also tended to be relatively high or low in the child's bedroom.

4.1. Measured DEHP Concentrations. We compared the median DEHP concentrations observed in our study with previous studies that measured DEHP in house dust across the world. Results are presented in Table S8. The median DEHP concentration measured in our study ($232 \mu\text{g/g}$) was roughly equivalent to the measurements reported in previous studies (e.g., the Kingston Allergy Birth Cohort) conducted in Canada [3, 40]. It is much lower compared with another Canadian House Dust Study, however, which had a median DEHP concentration of $426 \mu\text{g/g}$ [41]. Given the sampling years of those Canadian studies, reduced levels of DEHP in our study might be explained by the decade-old regulations on phthalates in Canada [35]. Specifically, six commonly used phthalates, including DEHP, were limited to soft vinyl toys and childcare articles, which came into force in June 2011. As these items were thought to be one of the major sources of DEHP at home, it was expected that restricting DEHP in those products would reduce exposure levels, as observed in the studies conducted after 2011. The median concentrations of DEHP found in European countries

mostly had higher levels than the concentrations in our study, up to a five-fold increase as found in Bulgaria [5, 42–44]. This might also be partly dependent on whether the sampling year was before or after 2005 when the European Union regulated phthalates in toys and childcare products [45].

Recent studies conducted in the US and China [1, 12, 15, 46] found lower median concentrations of DEHP compared with our results. The differences of DEHP concentrations observed across countries may reflect different lifestyles determined by cultural and social factors, such as building materials that are frequently used in the country and household ventilation. Further, sampling strategies can also influence the measurements, e.g., dust sampled from different microenvironments in the same home can vary [1, 47, 48]. Additionally, sampling season, which is related to air exchange and temperature, is also suspected to affect the measured concentrations in dust. For example, a study conducted in the US found that DEHP concentrations in settled dust in the winter were almost three times higher than the measured concentration in the summer [1].

In our mini-CHILD pilot-study analysis, however, we found that DEHP concentrations measured during two home visits separated by 6 months were correlated, suggesting that a one-time measurement of DEHP concentration in dust is reliable to represent DEHP exposure over a long period of time (i.e., ~ 1 year) in epidemiological studies. This finding is also supported by a previous study that tested the variability of semivolatile carpet-dust chemicals across repeated samplings [49].

On average, DEHP concentrations in dust were slightly higher in the bedroom compared to the most used living room; however, the correlation of continuous DEHP concentrations between these two rooms was moderate but significant, and dichotomized DEHP concentrations (high/low) were highly consistent between these two rooms. Considering that the correlations between the two rooms were not strong in our mini-CHILD pilot study, it appears that DEHP in homes does vary considerably by room, possibly as a result of the regular introduction of room-specific sources or room differences in ventilation and cleaning; thus, a detailed home environment assessment or questionnaire about characteristics in each room would be necessary to fully investigate sources. Our main study collected dust from both rooms but combined them into one dust sample for each home during the chemical analysis. Future studies could consider analyzing dust samples from different microenvironments within each home separately to investigate possible sources of phthalates more deeply.

4.2. Associations between DEHP and Housing Characteristics

4.2.1. Vinyl Flooring in the Kitchen and Bathroom. We observed strong associations between the vinyl floor in the kitchen and bathroom and DEHP concentrations in the dust. Since our dust samples were not collected from the floor surface in the kitchen or bathroom, this suggests that DEHP could be transported from those microenvironments to other rooms. It remains undetermined whether DEHP is

transported in the form of dust or air. Given that off-gassing of HMW phthalate from the vinyl floor generally occurs during the first few weeks to months after installation, depending on other factors such as temperature and ventilation [50], information on when the vinyl floor is introduced is useful to understand the mechanism. Further, we speculate that due to the transfer of DEHP from the floor material to dust [16, 51, 52], the DEHP concentration in dust in the kitchen and bathroom of houses with vinyl floors may have been greater, and hence infants spending more time in those rooms could have experienced even higher exposures. Broadly, these observations indicate that avoiding vinyl floors in selected rooms (e.g., bedrooms) may not be sufficient in reducing DEHP exposure at home. Bornehag et al. and Bi et al. also reported that DEHP concentrations in dust were associated with PVC and vinyl flooring in the house [1, 5]. A study in Japan concluded that the number of areas with PVC interior material, including the floor, wall, and ceiling of the living room, was related to higher DEHP median concentrations [9]. Those authors stated that most Japanese dwellings used PVC wall and ceiling coverings, whereas in the context of Canada, PVC is rarely used as a ceiling covering and thus was not assessed by our RAs. Vinyl flooring in bedrooms and living rooms was not investigated due to infrequent use, but laminate floors in those rooms were examined but were not found to be associated with DEHP levels.

4.2.2. Vinyl Furniture and Toys. We found a statistically significant positive association between DEHP concentrations in dust and the number of pieces of vinyl furniture in each room and the whole house. To our knowledge, no previous study has examined the impact of using plastic or vinyl furniture on DEHP exposure in the indoor environment. Additional characteristics of the furniture (e.g., size and solid plastic versus soft vinyl) in each house were not recorded, so we are not able to gain further insight regarding types of furniture to avoid. However, we have high confidence in the consistency of our strategy for counting furniture with significant vinyl content given the rigorous training of our RAs.

Vinyl toys in CHILD families were not found to be associated with increased levels of DEHP concentration in dust. This could be due to regulations on DEHP in soft vinyl toys [35] that came into force in 2011 in Canada since our dust sampling was conducted between 2010 and 2012. A recent study in Europe also suggested that the banned phthalates were no longer a major concern in PVC toys and childcare articles, rather, phthalate alternatives such as bis(2-ethylhexyl) terephthalate (DEHT) were most frequently detected in toys [53]. In CHILD, we have not yet looked for evidence of plasticizer alternatives in the dust. Given the likelihood that some DEHP-containing toys were still in use, other explanations for the lack of an association could be our dust collection from the floor and mattresses, if vinyl toys were predominantly kept off the floor and bed. However, DEHP levels were also not significantly higher in homes using plastic/foam playmats in the child's bedroom or the living room compared with homes not using them, though there was a

tendency of increased DEHP concentration when there were 1-4 pieces of playmats in the child's bedroom compared with none. Thus, we cannot exclude the possibility that some plastic/foam playmats in use might have been sources of phthalates at home.

4.2.3. Mattress Covering. One unanticipated finding is that DEHP concentrations in dust were lower in those families with an allergy control mattress cover on the baby's sleeping bed in the child's own bedroom (thus likely a crib). The statistically significant inverse association was shown in both the univariable analysis and the multivariable model adjusting for parental history of asthma and household income. Parental history of asthma was adjusted since we assume that parents with asthma might modify their behaviors (e.g., cleaning more frequently) and choices of products (e.g., allergy mattress covers), which could be related to lower DEHP levels. A previous experimental study in the US found that almost all crib mattress covers that they analyzed contained at least one plasticizer, including DEHP or its alternative, bis(2-ethylhexyl) isophthalate (isoDEHP) [54]. In our study, the material of mattress covers was not recorded and was not available to distinguish vinyl mattress covers from other more "natural" materials. We speculate that the inverse association between the covering and DEHP concentrations might indicate that some aspect of the mattress is also a DEHP source, such as from its construction materials or dust previously accumulated in the mattress that is resuspended with use. The covering on the mattress thus then reduces the release of such phthalates into the air and adjacent surfaces where the dust was collected, leading to the lower concentration we observed in some samples associated with homes where mattress covers were used. A study conducted in Swedish preschools found that the presence of foam mattresses was associated with elevated diisononyl phthalate (DiNP) concentrations in dust collected from elevated painted wood surfaces such as shelves but not associated with DEHP levels [13].

4.2.4. Mold. Evidence of mold was found to be associated with increased DEHP concentrations in the dust in the CHILD homes. Mold is not a source of phthalate, but it can be used as a proxy for relative humidity/dampness and indoor temperature [55]. Hsu et al. also found a higher DEHP level in relation to dampness or visible mold at home [27]. It has been suggested that phthalates could be released into the indoor environment from the degradation of phthalate-containing materials via two stages: the material phase (i.e., diffusion within the vinyl products) and the gas phase (from surfaces to the dust and air) [56]. Moisture is thought to accelerate the diffusion of DEHP inside the material, which affects the first stage of phthalate release. Based on experimental studies, higher moisture in PVC material, such as wallpaper and vinyl floor, led to greater emission of DEHP [5, 57]. Thus, mold signs in the home, indicating dampness, could be related to higher moisture in the phthalate-containing material and associated with higher DEHP concentrations in settled dust. Another possible explanation is that dust particles tend to adhere to one

another at higher humidity levels at home, becoming heavier and more likely to settle on surfaces, such as floors and beds [58]. DEHP in the settled dust could thus be affected by the humidity level at home. In our study, dust samples were collected on the floor and mattress surfaces, and DEHP concentrations measured in those dust samples could be higher among damp homes (i.e., homes with mold).

Therefore, there are several possible explanations for the observed association between DEHP concentrations and mold in the home. Controlling humidity or moisture build-up in the home (e.g., by increasing ventilation) might help slow the release of DEHP from its sources.

4.2.5. Polish Products. We did not find any association between the frequency of using polish products (e.g., floor polish and dust spray/polish) and DEHP concentrations in dust. Kolarik et al. found that Bulgarian families using any type of polishing agent weekly had a significantly higher DEHP concentration in dust compared with homes which never used such products [42], although a chamber study did not find an effect of floor wax polish on DEHP but on dibutyl phthalate [59]. Leather polish was found to be associated with higher DEHP levels in Chinese homes, but the association might be due to artificial leather containing phthalates [15].

Additionally, since carpets act as a reservoir for dust, we investigated the flooring type (carpeting vs. noncarpeting) that was vacuumed for dust sampling by RAs but found no significant difference between carpets and other floor types.

Possible protective factors (e.g., the use of air cleaners and vacuums and increased ventilation) that could reduce the buildup of DEHP in dust were explored, but no significant effects were found. For instance, the frequency of opening windows in the summer and winter was not found to be associated with DEHP concentrations. This was also suggested by previous studies which explored the effect of home ventilation rate, and some phthalates, such as di-n-butyl phthalate (DnBP), had a negative association with ventilation rate but not DEHP [5, 42]. It is unclear why DEHP concentration in dust was not found to be influenced by ventilation, but it might be related to its lower volatility compared with DnBP.

4.2.6. Correlation between DEHP in Dust and Urine. The correlation between DEHP in dust and the molar sum of DEHP metabolites in urine is low but significant ($r = 0.21$, $p < 0.05$). After adjusting for study center, child sex, and season in the regression model, DEHP in dust still remains significantly associated with the DEHP metabolite sum in urine (data not shown). Therefore, dust accumulated in the CHILd homes is believed to represent a non-dietary pathway for DEHP exposure in infants, though whether ingestion, inhalation, or dermal contact contributes more was not explored in our study.

To our knowledge, few studies simultaneously sampled dust and urine to analyze phthalate concentrations and their correlations. One German study on 3–14-year-old children did not observe correlations between the levels of DEHP in dust and its metabolites in urine [21]. Similar findings were

shown in a study on Danish children at 3–6 years of age [43]. Compared to these studies, the CHILd subjects were infants. At this age, they spend a greater proportion of time at home and potentially have closer contact with dust given time on the floor, possibly crawling [21, 23, 34]. Furthermore, as 3-month-old infants, their diet was less varied than that of children at age 3 and above, thus reducing dietary exposure to DEHP and enabling the dust-related pathway to be more clearly assessed. Specifically, in CHILd, only a small proportion (<10%) of children were introduced to solid foods by 3 months of age (data not shown), although there was variation in the source of milk, with around one-third of children being bottle-fed with formula at 3 months of age.

The contributions of inhalation, dust ingestion, and dermal absorption of dust to children's total intake of phthalates in preschool children were examined by Bekö et al. [23]. They found that daily ingestion of indoor dust from their preschool and home contributed up to 8% of the total intake of DEHP. The finding was supported by another study recently conducted in South China, which suggested that indoor dust, following indoor air, contributed up to 5.2% of the total daily intake of phthalates [60]. In their stratified analysis, toddlers were found to have the highest daily intake of phthalates through dust. Given that dietary intake is considered to be the main exposure route for high molecular weight phthalates, this proportion of nondietary exposure routes from dust was expected and not negligible [22].

Further, a randomized controlled trial on nearly 300 children from ages 1–3 found that reducing house dust could significantly lower the concentration of the sum of DEHP metabolites in urine [61], indicating that dust control plays an important role in reducing phthalate exposure.

4.2.7. SES and DEHP Levels. Lower household income families tend to be exposed to higher levels of DEHP in the dust. This result is consistent with findings from our previous study in CHILd, which found an inverse association between household income and levels of urinary DEHP metabolites [19]. Another similar study on women of reproductive age in the USA also found that lower SES was associated with higher concentrations of urinary metabolites of DEHP in pregnant women [37]. In contrast, increased DEHP concentrations were observed among families with higher household income in China [15]. The heterogeneous relationships observed in these studies might be due to different lifestyles in China and North America. To explore the underlying mechanisms of the observed association between SES and DEHP concentration in dust in our study, we investigated whether vinyl flooring, furniture, mattress covers, and mold, which were associated with increased DEHP concentrations, were also related to lower household income. We found that higher-income households are less likely to use vinyl flooring and be impacted by mold, but are more likely to use mattress covers. Given the positive association between vinyl floor, mold, and DEHP concentration and the negative association between the mattress cover and DEHP concentration, these factors might partly explain the observed negative association between SES and DEHP exposure in CHILd.

One of the strengths of our study is the large sample size of dust data from a cohort study compared with previous studies, as shown in Table S8. Another main strength of the study is that numerous home conditions that could be associated with exposure levels were obtained in the home environment questionnaire and RA home assessment. However, despite CHILD's detailed home environment data, much of the observed variability in DEHP levels in dust among homes could not be explained. This might be due to the limitations of relying on questionnaires or a standard RA survey for data collection. One limitation of this study is that it was not feasible, in a large cohort, to collect sufficient data on all products brought into the home, including plastic packaging materials, or on all infant or family behaviors that may influence DEHP levels. Another limitation of this study is that the CHILD Cohort represents higher-SES families. Therefore, our findings are more relevant to high-income families and may not be generalized to lower-income families.

5. Conclusions

This study indicates that vinyl flooring in kitchens and bathrooms, vinyl furniture, and mold in the home significantly increase the concentration of DEHP in house dust in Canadian households. Additionally, we observed that lower SES families are exposed to higher DEHP levels and are more likely to use vinyl floor and be impacted by mold in the home. Therefore, the observed association between SES and DEHP exposure could be partly mediated by the choice of materials and products in their homes. Further, a single-point sampling of DEHP in the dust is a reasonable representative of chronic exposure in children. Specifically, DEHP concentration in dust was found to be significantly associated with DEHP metabolite levels in urine, showing that dust is an important contributor to the total intake of DEHP in Canadian infants; DEHP concentrations in dust measured at two time points and the concentrations in different rooms within a home are significantly correlated.

Although regulations on phthalates in soft vinyl toys and childcare products have been implemented in Canada over a decade ago, we found that some DEHP-containing building materials and products are still related to higher DEHP exposure in Canadian homes. Therefore, more stringent policies may be needed to further mitigate indoor pollutants and protect children's health. Thorough premarket testing is needed to protect young children from chemicals in household products, including those that have replaced banned plasticizers such as DEHP. Moreover, the choice of flooring and other materials inside the home can make a difference in DEHP in dust levels. Other home conditions which could enhance the release of DEHP from sources can also modify the DEHP levels and thus should be paid attention to. Research designed to test the effectiveness of interventions that reduce these exposures in homes is needed.

Abbreviations

CHILD: Canadian Healthy Infant Longitudinal Development Study

DEHP: Di-(2-ethylhexyl) phthalate
 MEHP: Mono-(2-ethylhexyl) phthalate
 MEHHP: Mono-(2-ethyl-5-hydroxyhexyl) phthalate
 MEOHP: Mono-(2-ethyl-5-oxohexyl) phthalate
 DBP: Dibutyl phthalate
 HMW: High molecular weight.

Data Availability

The CHILD Cohort data used to support the findings of this study, including questionnaires and chemical analysis data, are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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Supplementary Materials

Table S1: distribution of building materials from the RA home assessment. Table S2: distribution of consumer products and behaviors related to DEHP exposure from the questionnaire and home assessment. Table S3: crude odds ratio (95% CI) of housing characteristics for dichotomized DEHP concentration in logistic regression models. Table S4: distribution of housing characteristics by DEHP quartiles. Figure S1: distribution of DEHP concentrations by other characteristics (insignificant results). Figure S2: DEHP in dust concentrations by household income groups. Table S5: distribution of DEHP-related housing characteristics by household income. Table S6: correlation between DEHP concentrations across two home visits. Table S7: distribution of dichotomized DEHP levels in bedrooms and most used family rooms. Table S8: comparison of median phthalate concentrations ($\mu\text{g/g}$) in dust as reported in other studies. (*Supplementary Materials*)

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